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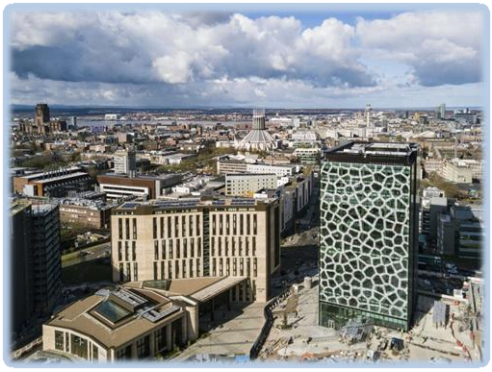
Annual Meeting

26th February 2026

The Spine

LIVERPOOL, UK



Inflammatory Comments

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Meeting Schedule

08.00-09.15	**Neuro-ophthalmology Allied Professionals Breakfast Meeting**	
09.30-09.40	**Welcome: Ailbhe Burke** **Chair: Jonny Virgo**	
09.40-10.10	**The epidemiology of optic neuritis**	Tasanee Braithwaite Consultant Ophthalmologist, St Thomas' Hospital, London
10.10-10.40	**Update on multiple sclerosis**	Carolyn Young Consultant Neurologist, The Walton Centre, Liverpool
10.40-11.20	**Update on MOGAD and NMO**	Saif Huda Consultant Neurologist, The Walton Centre, Liverpool
11.20-11.50	**Coffee**	
	Chair: Luke Bennetto	
11.50-12.30	**Paediatric CNS inflammatory disease**	Siobhan West Consultant Paediatric Neurologist, (R0A) Manchester University NHS FT
12.30-1.00	**When uveitis is a neuro-ophthalmic problem**	Nima Ghadiri Consultant Medical Ophthalmologist, St Paul's Eye Unit, Liverpool
13.00-14.00	**Lunch**	

	Chair: Ailbhe Burke	
14.00-15.00	**UKNOS Annual Lecture: Genome-wide analysis of aquaporin-4-positive neuromyelitis optica spectrum**	Lars Fugger Professor of Neuroimmunology, University of Oxford
15.00-15.30	**Service update: Non-medical professionals in neuro-ophthalmology: A curriculum and training routes for advanced clinical practice**	Mags Dayan Consultant Ophthalmologist, Royal Victoria Infirmary, Newcastle upon Tyne
15.30-16.15	**Poster viewing and tea**	
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	Early OCT Indicators are associated with degree of vision loss in acute NAION	Brian Woods

	False alarm or red flag? Predicting true papilloedema in paediatric optic disc swelling	Shie Wei Chan
	Trends in Neuro-Ophthalmology diagnoses and hospital admissions across England: A 10-year service evaluation	Surina Mittal
17.15-17.20	**Introducing the IIH Advance Trial**	Jessie Gew Clinical Research Fellow, University of Birmingham
17.20	**Prize giving**	
17.30	**Close of meeting**	

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List of Presented Posters

Speaker Biographies

Tasanee Braithwaite

Dr Tasanee Braithwaite is a Consultant Ophthalmologist (specialising in neuro-ophthalmology and uveitis) to the Medical Eye Unit at Guy's and St Thomas' NHS Foundation Trust in London. She also works as a Senior Lecturer at King's College London, holding a dual appointment between the School of Lifecourse and Population Sciences and the School of Immunology and Microbial Sciences, thanks to award of a Research Excellence Fellowship by the King's Health Partners Centre for Translational Medicine. She aims to advance delivery of predictive, preventive, personalised and participatory medicine for patients affected by inflammatory/infectious eye and brain conditions. Working with her KCL research team and a network of global collaborators, her current research programme focuses on:

- understanding the UK epidemiology of eye diseases and associated risk factors
- exploring polygenic risk of autoimmune disease in patients with inflammatory eye conditions
- developing tools to better quantify the impacts of inflammatory eye diseases on quality of life for use in clinical trials of new treatments
- advancing estimates on the global burden of eye diseases associated with potentially avoidable vision loss.

She has published widely, attracting over forty thousand citations. She currently serves as co-Chair of the Association for Research in Vision and Ophthalmology (ARVO) Clinical, Population Health and Data Science Section Annual Meeting Program Committee.

Carolyn Young

Professor Young has been a consultant neurologist at the Walton Centre NHS Trust since 1992 and set up the MS service there. She has been professor of neurology at the University of Liverpool since 2006 and principal investigator for over 160 trials. Prof Young has written over 200 papers; been a Member of Editorial Board of the Cochrane Multiple Sclerosis and Rare Diseases of the Central Nervous System Review Group from 2013; a member of NIHR CRN Neurosciences Group from 2014-2025; and is now a workstream lead in the new National Institute for MND Research.

She is Chief Investigator of the Trajectories of Outcome in Neurological Conditions (TONiC; https://tonic.thewaltoncentre.nhs.uk/) study and the complementary Factors Influencing Neurological Disease Risk and Severity (FINDeRS;

https://www.finders-study.org/) study. Prof Young's research focuses on improving care for people with MND and Multiple Sclerosis through examining their illness trajectories, assessed by patient reported measures including digital health, and investigating why these trajectories vary.

Saif Huda

Saif Huda completed his medical degree in Liverpool and neurology specialist training at The Walton Centre. He went on to complete a DPhil in neuroimmunology at the University of Oxford, followed by a post CCT clinical fellowship in neuroimmunology.

He is an NIHR clinician scientist and divides his time between his roles as a consultant neurologist at The Walton Centre and senior clinical lecturer and associate professor at the University of Liverpool. He is the clinical lead for the UK NMOSD highly specialised service, leads autoimmune neurology services at The Walton Centre, and serves as Director of the Liverpool Neuroscience Research Facility. His team delivers a national multidisciplinary clinical service for patients with autoimmune neurological diseases including NMOSD, myelin oligodendrocyte glycoprotein associated disease and multiple sclerosis.

Saif Huda's laboratory focuses on immune tolerance mechanisms and biomarker discovery in antibody mediated disease, with particular emphasis on clinical and serological markers that help predict relapse risk and long-term outcomes. He is a principal investigator for multiple Phase Three clinical trials in NMOSD, MOGAD and autoimmune encephalitis.

Siobhan West

Siobhan West is a Consultant Paediatric Neurologist at Royal Manchester Children's Hospital. After basic paediatric training in the North West, she decided to specialise in paediatric neurology and has subsequently developed an interest in neuroinflammatory conditions. This area has expanded over recent years with greater understanding and recognition of these conditions. She is the clinical lead of NOR-CNID, the highly specialised commissioned service that manages children in the north of England with demyelinating conditions including multiple sclerosis. She regularly contributes to the national UK-CNID meetings.

As well as her general neurology workload, she is part of the North of England specialist Neurofibromatosis type 1 service.

Nima Ghadiri

Nima Ghadiri is a Consultant Medical Ophthalmologist at Liverpool University Hospitals NHS Foundation Trust and an Honorary Clinical Associate Professor at

the University of Liverpool. His clinical practice focuses on ocular inflammation, neuro-ophthalmology and the links between eye disease and systemic conditions. He is actively involved in translational research and clinical trials in uveitis and orbital inflammation.

Lars Fugger

Lars Fugger is a Consultant Clinical Immunologist at the John Radcliffe Hospital at Oxford University Hospitals and professor of Neuroimmunology at the University of Oxford.

Scientifically, he has focused on translational studies in multiple sclerosis research and has been investigating how genes interplay with environmental factors to confer risk of the disease. This has been achieved by using a multidisciplinary approach with a large network of collaborators.

Lars Fugger trained at the University of Copenhagen in Denmark and at Stanford University in USA. He got his first clinical chair in Clinical Immunology in Denmark in 1996 and moved to Oxford in 2002 to work at the John Radcliffe Hospital. He became Professor of Clinical Immunology at the University of Oxford in 2004 and was appointed to the vacant chair of Neuroimmunology in 2007. He has won major European Prizes for his multiple sclerosis research and was elected to The Academy of Medical Sciences in 2010. He was the chairman of the Danish MRC from 2007-2010. Her Majesty the Queen of Denmark knighted him in 2011 for services to medicine.

Platform presentations

Calcitonin gene-related peptide induces headache attacks in people with idiopathic intracranial hypertension

Andreas Yiangou,[1,2] Thien Phu Do,[3] Susan P. Mollan,[1,4] Mark Thaller,[1,2] James L. Mitchell,[1,2,5] Caroline W. Mugo,[1,6] Georgios Tsermoulas,[1,2] Lisa J. Hill,[6] Samuel J. E. Lucas,[7,8] Messoud Ashina,[3,9] Alexandra J. Sinclair[1,2]

1. Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, B15 2TT, UK
2. University Hospitals Birmingham NHS Foundation Trust, Birmingham, B15 2GW, UK.
3. Department of Neurology, Centre for Discoveries in Migraine, Danish Headache Centre, Copenhagen University Hospital-Rigshospitalet, Copenhagen, 2600, Denmark.
4. Ophthalmology Department, Queen's University, Kingston, K7L 4V7, Ontario, Canada.
5. Academic Department of Military Rehabilitation, Defence Medical Rehabilitation Centre, Stanford Hall, LE12 5QW, UK.
6. School of Biomedical Sciences, Institute of Clinical Sciences, University of Birmingham, Birmingham, B15 2TT, UK.
7. School of Sport, Exercise and Rehabilitation Sciences, University of Birmingham, Birmingham, B15 2TT, UK.
8. Centre for Human Brain Health, University of Birmingham, Birmingham, B15 2TT, UK.
9. Department of Clinical Medicine, University of Copenhagen, Copenhagen, 2200, Denmark.

Introduction

Calcitonin gene-related peptide (CGRP) is a key mediator in migraine pathophysiology. Idiopathic intracranial hypertension (IIH) headache phenotype is now understood to be typically migraine-like, but it is unclear whether CGRP directly provokes IIH headaches or alters intracranial pressure (ICP) dynamics.

Methods

We conducted a randomised, double-blind, placebo-controlled, two-way crossover trial to address this. Twenty women with IIH and no prior migraine were randomly assigned to receive a 20-min continuous intravenous infusion of CGRP (1.5 µg/min) or placebo (isotonic saline). The primary outcome was the difference in the proportion of participants who developed a provoked headache attack between CGRP and placebo during the 12 h observation after infusion. Secondary outcomes included the area under the curve (AUC) for headache intensity from -10 min to 12 h, the timing and duration of headache features, and baseline-adjusted changes for vital signs, cerebrovascular haemodynamics and ICP.

Results

Seventeen participants with mean (SD) age 26.7 (6.4) years completed both visits. Twelve (71%) participants developed a typical IIH headache attack with migraine-like features after CGRP compared with three (18%) after placebo (risk difference 53%; 95% CI, 26–79; P = 0.004). The AUC-10min-12h for headache intensity was higher after CGRP than after placebo (P = 0.016). The mean ICP remained unchanged, whereas ICP amplitude increased significantly after CGRP (P = 0.005). Vital signs and cerebrovascular haemodynamics AUC-10min-90min were significantly altered after CGRP (increased: heart rate (P < 0.001), tissue oxygenation index (P = 0.041), oxygenated haemoglobin (P < 0.001) and decreased: mean arterial pressure (P = 0.010), middle cerebral artery blood velocity (P = 0.006)).

Discussion

CGRP reliably provoked typical IIH headache attacks (which have migraine-like features) and increased ICP pulse amplitude, as measure of intracranial compliance, without altering mean pressure. These findings provide mechanistic support for CGRP involvement in headache attributed to IIH and justify prospective evaluation of CGRP pathway blockade in this population.

Early OCT Indicators are associated with degree of vision loss in acute NAION

Brian Woods, Physics Department, School of Natural Sciences, University of Galway, Ireland
David Szanto, Department of Ophthalmology, Icahn School of Medicine at Mount Sinai and New York Eye and Ear Infirmary, New York NY
Jui-Kai Wang, Department of Ophthalmology, University of Texas Southwestern Medical Center, Dallas, Texas
Aaron Golden, Physics Department, School of Natural Sciences, University of Galway, Ireland
Mona K. Garvin, Department of Ophthalmology and Visual Sciences, University of Iowa Hospitals and Clinics, Iowa City, IA
Randy H. Kardon, Department of Ophthalmology and Visual Sciences, University of Iowa Hospitals and Clinics, Iowa City, IA
Mark J. Kupersmith, Department of Ophthalmology, Icahn School of Medicine at Mount Sinai and New York Eye and Ear Infirmary, New York NY

Purpose

We aim to characterize longitudinal structural changes in non-arteritic anterior ischemic optic neuropathy (NAION) using optical coherence tomography (OCT) and evaluate their relationship with visual outcomes in a large clinical trial cohort.

Methods

We used OCT data including peripapillary retinal nerve fiber layer thickness (pRNFLT), peripapillary total retinal thickness (pTRT), optic nerve head volume (pONHV), and segmentation of the macular ganglion cell-inner plexiform layer (GCIPL) thicknesses from 715 participants in the QRK207 trial (NCT02341560) imaged within 14 days of NAION onset (mean: 8.0 ±SD: 3.2). Imaging was performed at Screening, Enrollment, Month 2, Month 6, and Month 12. We analyzed relative differences in study and fellow eye ONH, sectoral correlations, and rates of early OCT metric increase. Associations with best-corrected visual acuity (BCVA) and visual field (VF) outcomes were assessed with regression analyses.

Results
Study eyes had ONH swelling at Screening (mean pRNFLT 241.0 ±74.8 µm; pONHV 6.70 ±1.27 mm^3), followed by marked thinning (pRNFLT 56.7 ±14.9 µm; pONHV 2.74 ±0.41 mm^3 at Month 6). For study eyes without prior NAION in the fellow eye, increased differences in baseline inter-eye ONH OCT metrics were associated with worse VF sensitivity and BCVA (e.g., inferior pRNFLT vs superior VF: rho = -0.35, p<0.001). Sub-analysis of participants with measurements between Screening and Enrollment (n=182; mean: 3.5 ±2.0 days) showed that GCIPL thinning (-3.0 µm, p<0.001) was detectable at an early stage. An initial rate of pONHV thickening of 0.15 mm3/day conferred a 4.0-fold higher odds of VF decline by Month 2 (CI 2.8-8.5, p < 0.001, n =222).
Conclusions
Macular GCIPL thinning is evident at an early stage following NAION onset. Increased baseline inter-eye pRNFLT asymmetry and early ONH volumetric increases in the first days of NAION onset are associated with worse BCVA and VF outcomes.

False alarm or red flag? Predicting true papilloedema in paediatric optic disc swelling
Shie Wei Chan, Hadi Masoodi, Nader Hassan, Bhamy Hariprasad Shenoy
Manchester Royal Eye Hospital, UK
Introduction
Papilloedema - true optic disc swelling secondary to raised intracranial pressure can progress to optic atrophy and permanent vision loss. Distinguishing true papilloedema from psuedopapilloedema can be challenging because both can present with disc elevation and blurred margins. Referrals for suspected papillloedema in children are rising, prolonging waits and diluting true positives.
Purpose
To evaluate referral accuracy, demographic and symptom profiles, and to develop a triage tool to distinguish true papilloedema from pseudopapilloedema within a tertiary paediatric pathway
Methods
We retrospectively reviewed 504 paediatric referrals (≤18 years) to the Manchester Royal Eye Hospital Optic Disc Swelling pathway from 2021-2025. Patients with a known prior diagnosis were excluded from triage derivation. Multivariable logistic regression analysis identified predictors of true papilloedema. We calculated sensitivity, specificity and negative predictive value (NPV) to develop triage rules.
Results
70 (14 %) had true papilloedema, and 434 (86 %) had pseudopapilloedema. Median age was 12 years; 58% were female. Predictors of true disease included vomiting (p = 0.004, OR 3.48), headache (p = 0.010, OR 1,94) and any visual symptom (p = 0.012, OR 2.83). Emergency department referral predicted true papilloedema (p < 0.001). The common causes were intracranial tumour (n=20), idiopathic intracranial hypertension (n=19), and venous sinus thrombosis (n=5). Common pseudopapilloedema phenotypes were optic disc drusen and titled disc. A high-risk triage rule: ED referral, vomiting, or headache plus a

visual symptom achieved sensitivity 73 %, specificity 54 %, and NPV 92 %. Age, sex, ethnicity and refractive status were not significant predictors.

Discussion

Only one in seven referrals (14%) represented true papilloedema, highlighting over-referral and the difficulty of disc assessment. Reliance on disc appearance alone risks delayed diagnosis of life-threatening pathology and may trigger unnecessary investigations such as neuroimaging and lumbar puncture. Early risk stratification using symptom burden (vomiting, headache, visual symptoms) and referral source (ED vs optometrist) may improve prioritisation. The proposed triage rule had a high NPV (92%), supporting its use as a rule-out tool to de-prioritise low-risk referrals while accelerating assessment for higher-risk children. The 14% diagnostic yield is lower than some published referral cohorts, likely reflecting referral thresholds. Improving optometrist and ED clinician education on pseudopapilloedema phenotypes and standardising referral information may reduce low-yield urgent referrals and protect capacity for true positives. This triage tool is intended to complement established neuro-ophthalmic pathways. Future work could prospectively validate this tool in multicentre cohorts.

Trends in Neuro-Ophthalmology diagnoses and hospital admissions across England: A 10-year service evaluation

Dr Surina Mittal, Ophthalmology, Oxford Eye Hospital, UK

Introduction

Neuro-ophthalmic conditions such as optic neuropathies, idiopathic intracranial hypertension (IIH) with papilloedema, and cranial nerve palsies require urgent ophthalmology review, imaging and often subsequent hospital admission. Incidences of such presentations are considered to be rising, though a formal long-term analysis of trends across England has not been conducted. Comprehensive understanding of trends in hospital episodes is critical for workforce and service planning, particularly in neuro-ophthalmology, where delayed patient care may have devastating, irreversible consequences.

Methods

A retrospective analysis of the Hospital Episodes Statistics (HES) data published by NHS England was conducted between April 2015 and March 2025. Three categories of primary neuro-ophthalmology diagnoses were created using ICD-10 codes; Optic Neuropathies (H46.X, H47.0, H47.2, H47.3), IIH/Papilloedema (H47.1, G93.2) and Cranial Nerve Palsies (H49.0-4, H49.8-9). Annual consultant episodes and hospital admissions for each diagnostic category were recorded and analysed with descriptive statistics. Trends were evaluated using simple linear regression, using slope and R2 to compare groups, with $p<0.05$ deemed statistically significant.

Results

Across the 10-year period, a total of 127,106 consultant episodes occurred with a primary diagnosis within neuro-ophthalmology, 102,932 (80.98%) of which resulted in hospital admission. IIH consistently accounted for the greatest overall hospital workload, though a

primary diagnosis of optic neuropathy dominated hospital admissions in neuro-ophthalmology. Regression analysis demonstrated statistically significant year-on-year increases in the number of consultant episodes across all diagnostic categories; Optic neuropathy: +103.6 episodes/year (R^2=0.80, p=0.0005), IIH: +250.1/year (R^2=0.77, p=0.0008), Cranial palsy: +33.4/year (R^2=0.56, p=0.0129). Despite statistically significant differences in the absolute number of hospital admissions for optic neuropathies (p=0.0009) and IIH (p=0018), there was a significant annual reduction in the proportion of hospital admissions from consultant episodes across all three groups.

Discussion

This national study provides robust, 10-year evidence of the rising escalation in neuro-ophthalmic activity across NHS services in England. Consistent, significant rises in hospital episodes for optic neuropathies, IIH and cranial nerve palsies suggest potential for increasing strain on neuro-ophthalmic inpatient and outpatient pathways. Persistently rising admission counts demonstrate the increasing burden of neuro-ophthalmic conditions on NHS services, though the decreasing proportion of admissions across all groups indicates that neuro-ophthalmic workflows are adapting to allow for more outpatient management of clinical presentations. Overall, these findings support the need for ongoing development and investment into neuro-ophthalmology services across NHS England, including clinic capacity, referral and imaging pathways, and multidisciplinary services to support the rising demand across England, mitigate avoidable hospital admissions, and preserve vision.

Poster Abstracts

1) An unusual presentation of progressive bilateral 6th nerve palsy and papilloedema

Bethany Ellis, North West Anglia NHS Foundation Trust
Haleema Zaman, North West Anglia NHS Foundation Trust
Evangelos Minos, North West Anglia NHS Foundation Trust

Case report

A middle-aged gentleman presented to the Eye Clinic with progressively worsening horizontal diplopia due to bilateral 6th nerve palsy over the course of 2 months, without any reduction in visual acuity. He had been investigated for other systemic symptoms, including incidental normocytic anaemia, episodes of frank haematuria, and joint pains in his hands and feet. No definite cause of these signs and symptoms had been found previously. After noting slightly blurred optic disc margins at the first visit, we later saw a progression to grade 2 papilloedema. Retrospective analysis of blood tests for his systemic symptoms showed that, in addition to his normocytic anaemia, there was a rapidly deteriorating renal function and elevated C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Magnetic resonance imaging (MRI) of the head and orbits showed meningeal thickening but no space-occupying lesions, orbital inflammation, or other pathology. After admission into hospital and further tests, he was found to have an elevated central anti-neutrophil cytoplasmic antibody (c-ANCA) count, leading to a diagnosis of granulomatosis with polyangiitis (GPA) as the cause for his symptoms. He was immediately initiated on potent immunosuppressive therapy and after a few months his condition stabilised, with his ophthalmic signs and symptoms receding almost completely.

Discussion

GPA typically presents with ophthalmic manifestations at more advanced stages of the disease. In our case, the patient did not have any signs of ophthalmic inflammation or sinus disease related to GPA, and all visual symptoms occurred solely due to CNS infiltration and meningeal inflammation. As there were also no signs of orbital disease in the MRI scans, the presence of papilloedema with diplopia is likely to have arisen specifically due to hypertrophic pachymeningitis related to GPA. This case is an important reminder to consider systemic vasculitis, such as GPA, in our differential diagnosis in middle-aged patients presenting with binocular diplopia with bilateral papilloedema, especially in the presence of systemic symptoms or inconclusive investigations. Patients may sometimes have an atypical course of the disease and ophthalmic symptoms and signs should be related to GPA or any other vasculitis, as occurred in our case. Prompt testing for vasculitis and appropriate recognition of features of clinical deterioration and worsening blood biomarkers were instrumental in reaching the final diagnosis with a positive outcome after treatment.

2) Inflammatory clues from an atypical case of optic disc swelling

Shie Wei Chan, Nishtha Singh, Ali Yagan, Mandagere Viswanath
Manchester Royal Eye Hospital

Case report

Unspecified systemic symptoms, such as headache, visual abnormalities, and breathlessness, presenting in the elderly may seem like a benign inflammatory condition, such as giant cell arteritis or a lower respiratory tract infection. However, we present an atypical case of an 80-year-old female who was diagnosed with paraneoplastic optic neuropathy (PON) following multiple investigations and clinical reviews.

She presented with vague systemic symptoms: headache, dizziness, flashers and floaters in the eye, and weight loss. She was initially treated for postural hypotension, vestibular dysfunction, gastritis, and hospital-acquired pneumonia. Ocular examination revealed bilateral disc swelling with mild vitreous haemorrhage. Differentials included giant cell arteritis, ischaemic optic neuropathy, venous

sinus thrombosis, primary vitreous lymphoma, uveitis, and vasculitis. Early investigations, including temporal artery ultrasound, neuroimaging, fluorescein angiography, and lumbar puncture, were all negative. Persistent optic disc swelling led to repeated admissions under the neuro-ophthalmology team. Results for CSF cytology and haematological malignancy were negative. Paraneoplastic antibody screening was positive for anti-Hu and anti-CV2/CRMP-5, suggesting an underlying malignancy. The patient developed vitreous haemorrhage and was treated with intravitreal gas injection, anterior chamber washout, pars plana vitrectomy, and high-dose intravenous methylprednisolone. Vitreous haemorrhage raised suspicion for primary vitreous lymphoma, but the vitreous biopsy was negative. Systemic malignancy screening for breast cancer was also negative. After eight months from the first presentation, there was still no formal diagnosis. A thorough history was repeated- she reported significant back pain, hip pain, and weight loss over the last few months. This prompted a PET-CT scan, which revealed mediastinal lymphadenopathy, bony lesions, and a right infrahilar nodule. She was diagnosed with right hilar malignancy (N2M1c) and commenced on palliative care.

Discussion

PON is rare, affecting 1 in 300 patients with malignancy. Its ophthalmic manifestations are variable and often appear after cancer diagnosis. It results in inflammation of the eye and damage at sites distant from the tumour, including the neuro-ophthalmic system. While PON typically manifests after a cancer diagnosis, our patient's visual symptoms preceded it. This case demonstrates how subtle ocular inflammatory features : mild vitreous inflammation and choroidal folds without uveitis can be early "inflammatory comments" signalling significant systemic disease. PON is commonly linked to small cell lung carcinoma, with antibodies such as anti-Hu known to cross-react with neuronal and glial tissue. We propose that paraneoplastic screening should be performed at initial presentation along with other top differentials in optic disc swelling, such as ischaemic and inflammatory pathology.

3) An Unusual Case of Painful Ophthalmoplegia and Ptosis

Dr Nuala Pepper, Foundation Programme, United Lincolnshire Hospitals NHS Trust
Dr Ruth Batty, Neuroradiology, Sheffield Teaching Hospitals NHS Foundation Trust
Dr Simon J Hickman, Department of Neurology, Royal Hallamshire Hospital, Sheffield Teaching Hospitals NHS Foundation Trust
Dr Eu-Wing Toh, Histopathology, Royal Hallamshire Hospital, Sheffield Teaching Hospitals NHS Foundation Trust

Case report

We present a 61-year-old woman, with a recent diagnosis of type 2 diabetes mellitus, who presented with severe right-sided ocular and facial pain. Within 24 hours she had developed right eye ptosis and loss of vision. She denied jaw claudication, fatigue, night sweats or weight loss. She was apyrexial. She had no perception of light in her right eye but could see 6/6 with her left eye. There was non-axial 3mm proptosis of the right globe, complete right-sided ophthalmoplegia with complete ptosis. The orbit was not tense and there was no erythema or oedema of the conjunctiva. The right anterior chamber was quiet, and the intraocular pressure was 9mmHg. The pupil was fixed, dilated and non-responsive to light with a reverse afferent pupillary defect. Fundal examination detected pale swelling of the optic nerve head with cloudy swelling of the retina and a cherry red spot. The temporal arteries were pulsatile and non-tender. There was decreased sensation in a V1 distribution with reduced corneal sensation. Her left eye was normal. Her inflammatory markers were raised (ESR 89mm/hr, CRP 162mg/L). ANCA, ANA and rheumatoid factor were negative and IgG4 levels were normal. Imaging of the head and orbits showed right orbital apex inflammation with involvement of the fat, muscles, optic nerve, and likely involvement of the cavernous sinus. There were inflammatory changes in the right frontal and ethmoid sinuses with no bony erosion or orbital collection. Nasal endoscopy excluded infective sinusitis. A temporal artery biopsy detected arteritis but no giant cells or granuloma were

seen. Once commenced on steroids, the inflammatory markers improved and her pain subsided quickly. Due to difficult diabetic control tocilizumab was commenced. There was no improvement in her right eye ocular abnormalities, however the left eye remains healthy with good visual function.

Discussion

Giant cell arteritis (GCA) has a wide spectrum of presentations and has been previously reported to cause an orbital apex syndrome. The differential diagnosis in our case included idiopathic orbital inflammation, other inflammatory conditions, and sinus inflammation or infections. Although the biopsy did not detect giant cells or granuloma formation, the negative autoimmune screen and normal IgG4 levels excluded other forms of arteritis, pointing to the presumed diagnosis of GCA. Keeping an open mind with timely multi-specialty input was imperative we think in preventing the other eye becoming affected.

4) Papilloedema as an Unusual Initial Manifestation of a Multisystem Disorder

Dr Elaina Pasangha, Haematology Registrar, University Hospitals Sussex NHS Foundation Trust
Dr Sarah Cooper, Consultant Neurologist, University Hospitals Sussex NHS Foundation Trust

Case report

A 41-year-old slim Nigerian woman with no significant past medical history presented with recent episodes of visual blurring lasting about 20 minutes and progressively worsening occipital headaches for a year. Eye examination revealed bilateral papilloedema. MRI brain imaging and venography was normal; lumbar puncture demonstrated raised cerebrospinal fluid (CSF) protein (0.9 g/L) and high opening pressure (56 cm CSF). Acetazolamide 500 mg twice daily was commenced. Blood tests showed low MCV, normal haemoglobin, thrombocytosis, and elevated prolactin. Her symptoms progressed over 11 months to include sensory and motor neuropathy affecting both legs up to knees, ascites and worsening blurred vision prompting hospital admission. Investigations showed elevated IgG lambda and hypothyroidism; she was started on levothyroxine. CT scan revealed chronic liver disease, splenomegaly, large ascites, and inguinal lymphadenopathy. Ascitic fluid cytology and inguinal lymph node biopsy were negative for malignancy. There were nonspecific itchy, hyperpigmented warty plaques on limbs. Nerve conduction studies indicated length-dependent neuropathy and carpal tunnel syndrome. Repeat MRI brain and spine showed symmetrical enhancement of cranial and spinal nerves without nodules or leptomeningeal disease. Bone marrow biopsy revealed 3% plasma cells and otherwise normal trilineage haematopoiesis. VEGF level was elevated at 4135 pg/mL. She was diagnosed with POEMS syndrome and started on chemotherapy with daratumumab, bortezomib, and dexamethasone (DVTD regimen eliminating thalidomide due to neuropathy).

Discussion

POEMS syndrome is a rare paraneoplastic syndrome linked to an underlying plasma cell disorder, the acronym of which stands for Polyradiculoneuropathy, Organomegaly, Endocrinopathy, Monoclonal plasma cell disorder, and Skin changes. Diagnosis requires meeting three major criteria—polyradiculoneuropathy, clonal plasma cell disorder, sclerotic bone lesions, elevated VEGF, and Castleman disease —and at least one minor criterion – organomegaly, endocrinopathy, papilloedema, thrombocytosis, and extravascular volume overload. Treatment focuses on eliminating the plasma cell clone, with radiotherapy preferred for isolated bone lesions, and systemic therapy reserved for disseminated disease. Papilloedema in POEMS is thought to relate to increased vascular permeability due to circulating VEGF and is likely an ocular manifestation of systemic oedema. Unfortunately its' presence is a poor prognostic sign. This case serves as a reminder that papilloedema may have extracranial origins.

5) Under Pressure: Deciphering the Cause of Optic Disc Oedema

Wei Jia Liu, Jasvir Virdee
University Hospital Southampton NHS Foundation Trust, Southampton, United Kingdom.

Case report

A 20-year-old Caucasian woman with HLA-B27 positive juvenile idiopathic arthritis (JIA), bilateral anterior uveitis, secondary glaucoma, and dense amblyopia in the right eye, presented with progressive visual decline in her left eye and bilateral asymmetric optic disc swelling. At the neuro-ophthalmology clinic, her visual acuity was 3/60 (left) and counting fingers (right); intraocular pressures (IOPs) were 8 mmHg and 23 mmHg, respectively. Fundus examination revealed marked left optic disc swelling with venous tortuosity, and milder swelling in the right eye. Following left eye aqueous shunt surgery for glaucoma, she had been experiencing fluctuating IOP ranging from 52 mmHg to 8 mmHg. At IOP 10 mmHg and below, optic disc swelling appeared to worsen. Differential diagnoses included raised intracranial pressure, uveitic optic nerve inflammation, optic neuritis, and hypotony-related disc oedema. Neuroimaging showed features of raised intracranial pressure (ICP), and lumbar puncture (LP) showed opening pressure of 20cmH2O, with normal constituents, while on acetazolamide. The clinical picture and IOP fluctuations suggested that super-added hypotony was the primary driver of significant left optic disc oedema. Concurrent probable IIH likely contributed to bilateral disc swelling. The patient underwent left aqueous shunt revision with anterior chamber reformation using Healon. Postoperatively, IOP rose to 32 mmHg with visual improvement to 6/12 and significant resolution of optic disc oedema.

Discussion

Ocular hypotony is an uncommon but vision-threatening cause of optic disc oedema. The translaminar pressure gradient (TLPG) – the pressure differential across the lamina cribrosa – reflects the balance between IOP and cerebrospinal fluid pressure (CSFP). When CSFP exceeds IOP, anterior displacement of the lamina cribrosa occurs, causing optic disc oedema. In this patient, we propose a dual-pathology mechanism: underlying IIH with concurrent hypotony resulting in papilloedema and marked left optic disc swelling. We speculate that optic nerve oedema may occur at relatively higher IOPs in the setting of raised ICP due to the TLPG effect, explaining worsening disc swelling at an IOP of 10mmHg. A normal LP opening pressure may not exclude IIH, as CSFP can fluctuate and be influenced by timing, posture, procedural factors, and recent therapy e.g. acetazolamide. Inflammatory processes, such as JIA-associated uveitis, may further increase vascular permeability and tissue fragility, exacerbating hypotony-related disc changes. Overlapping risk factors created a challenging diagnostic landscape. The dramatic visual recovery following surgical revision emphasises the importance of timely recognition and correction of hypotony to prevent irreversible optic nerve damage.

6) Sinus Mucormycosis Presenting as Orbital Apex Syndrome in a Newly Diagnosed Diabetic Patient: A Therapeutic Dilemma

Wei Jia Liu, Jasvir Virdee
University Hospital Southampton NHS Foundation Trust, Southampton, United Kingdom.

Case report

A 49-year-old woman presented with diabetic ketoacidosis (DKA) and was newly diagnosed with type II diabetes, likely precipitated by pancreatitis. During her admission, she developed a left-sided headache, diplopia, and ptosis. Examination revealed left proptosis, restricted extraocular movements, and subsequently, reduced visual acuity with a relative afferent pupillary defect (RAPD) and optic disc swelling – features consistent with left orbital apex syndrome (OAS). MRI of the brain and orbits showed predominantly left-sided acute sinusitis with mucosal thickening and fluid levels within the paranasal sinuses with diffuse orbital changes, including extraocular muscle swelling and intraorbital fat oedema. Given the new diagnosis of diabetes and imaging findings, rhino-orbital mucormycosis

was suspected. Empirical intravenous amphotericin B and broad-spectrum antibiotics were commenced, followed by functional endoscopic sphenoid sinus surgery (FESS). Initial sinus cultures grew Staphylococcus aureus and Cutibacterium acnes and the infectious disease MDT initially felt that mucor was unlikely given no reports of necrosis on ENT washouts and lack of growth yet on samples. Antifungal therapy was discontinued and antibiotics were rationalised. Due to ongoing visual decline, intravenous methylprednisolone was initiated to treat optic nerve inflammation at the orbital apex. Further histopathology of sinus tissue demonstrated fungal hyphae consistent with Zygomycetes. The patient was immediately restarted on antifungal therapy with posaconazole, and a more rapid steroid taper was implemented. Subsequent fungal PCR confirmed Rhizopus arrhizus, consistent with invasive mucormycosis. Following treatment, there was marked improvement in ocular motility, visual acuity, and optic disc swelling. Follow-up MRI demonstrated reduced orbital inflammation.

Discussion

This case highlights a rare presentation of sinus mucormycosis manifesting as OAS. There are diagnostic challenges in confirming mucormycosis in the orbital apex as direct orbital tissue sampling carries significant visual risk, necessitating reliance on sinus biopsy results. The differential between sinus mucormycosis with inflammation at the orbital apex versus infiltrative disease is one we must consider carefully. Corticosteroids, while essential for reducing vision-threatening inflammation, can exacerbate fungal proliferation. Significant visual decline with development of RAPD and disc oedema whilst on initial antifungals suggested that further anti-inflammatory treatment was necessary as a sight-saving measure. The patient's clinical and radiological improvement supported OAS due to an inflammatory response to sinus mucormycosis rather than direct fungal invasion. We recommend an MDT approach to monitor carefully for resolution of symptoms and the minimum steroid dose required. The reversal of DKA in a timely fashion will also help control invasive fungal disease.

7) Diagnosing and managing functional visual disorder

Nisha Gurung, Martin Rhodes, Heidi Laviers, Arani Nitkunan
St George's University Hospitals NHS Foundation Trust

Introduction

Functional visual disorder (FVD) is a subtype of functional neurological disorder, characterised by visual disturbances inconsistent with organic pathology. ~3% of ophthalmology referrals are due to FVD, and 26-53% of patients have an underlying ophthalmic or neurological diagnosis. FVD can present either monocularly or binocularly with diplopia, field defects or vision ranging from blurred to no perception of light (NPL). We present a case of FVD in a patient with monophasic myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) and chronic pain syndrome.

Case report

A 57-year-old gentleman with a history of migraine and chronic back pain following 4 vertebral fusions, presented in 2017 in South Africa with a week of retro-orbital headache and bilateral blurred vision. He had NPL in the right eye (R) and minimal vision (not quantified) in the left eye (L). MRI confirmed bilateral optic neuritis. His vision recovered to baseline within 2 months following IV methylprednisolone, plasma exchange and azathioprine. In early 2018, he experienced a further episode of right retro-orbital headache and blurred vision. Visual acuities (VA) were 6/12 (R) and 6/5 (L), with impaired colour vision. MRI showed bilateral optic nerve enhancement, and MOG antibodies were positive, confirming MOGAD. VA improved to 6/6 bilaterally following a high dose of intravenous methylprednisolone and azathioprine. By mid-2018, treatment was discontinued upon seronegative MOG antibodies. In late 2018, he reported subjective visual deterioration. Objective assessment demonstrated stable VA (6/6-6/9), normal visual evoked potentials (VEPs), optic coherence tomography (OCT) showed stable retinal nerve fibre layer, and MOG antibodies remained negative. This episode was considered non-relapsing. Over subsequent years, he reported fluctuating "foggy"

vision, photophobia and periorbital discomfort. However, all objective assessments, including VA (6/6-6/9), MRI, MOG antibodies, OCT and VEP, repeatedly implied no new inflammatory activity. Comprehensive neuro-ophthalmic and orthoptic evaluation identified positive signs consistent with FVD. These included preserved binocular field despite subjective monocular visual loss. Neurological examination was unremarkable with positive Hoover's sign. Immunotherapy was discontinued and redirected with reassurance, psychological support and ongoing chronic pain management.

Discussion

This case highlights the diagnostic complexity and importance of differentiating FVD from monophasic MOGAD in a timely manner for optimised care. Many factors, including chronic pain, polypharmacy and stress, can contribute to the development of FVD and mimic organic pathological signs. Understanding the principles of functional vision tests and performing them seamlessly helps make a positive diagnosis, rather than one based solely on exclusion.

8) The Headache That Sent Us on an Unexpected Neurological Hunt

Dr Shuchi Kohli[1,2], Dr Simon Merrick[2, 3]. Mrs Anita Reynolds[2]
1 Mid Yorkshire NHS Hospitals Trust
2 Bradford Royal Infirmary NHS Teaching Hospitals
3 Leeds NHS Teaching Hospitals

Case report

A 37-year-old woman was referred from A&E with a five-day history of horizontal diplopia with complete right sided lateral rectus palsy. This followed a five-month history of persistent right-sided headache beginning after dental work, treated as presumed post-operative infection with multiple courses of oral antibiotics prescribed in primary and ENT care. She also reported dizziness, unintentional weight loss and reduced appetite, but denied fever, pulsatile tinnitus, or postural worsening of headache. Ocular examination was otherwise normal: pupils were equal and reactive, visual fields and colour vision were intact, and there was no relative afferent pupillary defect or proptosis. A recent private neurology review had resulted in a diagnosis of migraine-related pain with an MRI demonstrating sinusitis and a pineal cyst with no other abnormalities. Further to this, our ENT colleagues had requested a CT sinuses. Baseline bloods were unremarkable (CRP <1, normal WCC, platelets and neutrophils). The patient was followed up in the orthoptist clinic, who noted improvement of sixth nerve palsy, with no new symptoms.

Two months later she re-presented to eye casualty with worsening symptoms, including new right-sided partial ptosis with new horizontal diplopia. The sixth nerve palsy had resolved, but new deficits had developed involving cranial nerves III, VI and V, with subjective corneal hypoesthesia. Systemic symptoms had progressed, with worsening headache, nausea, dizziness, and flushing. Initial CTA/CTV reporting was unremarkable apart from right sphenoidal sinus thickening. Interestingly, a subsequent addendum to initial CTA report identified a soft-tissue mass in the lateral wall of the right cavernous sinus with stenosis of the adjacent cavernous internal carotid artery. Differential diagnoses ranged from inflammatory, infectious, neoplastic or vascular causes. After exclusion of the aforementioned pathologies, a diagnosis of Tolosa-Hunt Syndrome (THS) was made.

Discussion

THS is a rare, idiopathic inflammation of the cavernous sinus, with an estimated incidence of 1 per million annually. It typically presents with painful ophthalmoplegia involving one or more cranial nerves, often with fluctuating and progressive deficits which may lead to delayed diagnosis and treatment. This case highlights the diagnostic challenges of THS, particularly when initial imaging is inconclusive and symptoms mimic more common infective or sinus-related pathologies. Prompt diagnosis is crucial, as THS typically responds well to corticosteroids. It should be considered especially in patients with progressive cranial neuropathies.

9) Painful visual loss and optic perineuritis with systemic lymphadenopathy – a diagnostic challenge

Muhammad Sabeeh Ahmed, University College London Hospitals
Ashir Iqbal, King's College Hospital
Rogan Fraser, Moorfield Eye Hospital
Alibhe Burke, University College London Hospitals

Case report

A 55-year-old woman presented initially to Moorfields Eye Hospital with subacute, painful right-sided visual loss. Visual acuity was 6/36 with a mild relative afferent pupillary defect. Inflammatory markers were modestly raised (ESR 34 mm/h, CRP 5.2 mg/L). MRI demonstrated perineural enhancement of the right optic nerve consistent with OPN. She was treated with oral corticosteroids with partial improvement, but relapsed following taper. Subsequent admissions revealed evolving polyarthralgia, intermittent erythematous nodular rashes, and right optic disc swelling. Repeat MRI at the National Hospital for Neurology and Neurosurgery (NHNN) demonstrated concentric enhancement and thickening of the right optic nerve sheath. CSF analysis showed raised opening pressure (25 cm H_2O) and paired oligoclonal bands with normal protein and cell count. PET-CT identified metabolically active mediastinal and bilateral hilar lymph nodes with mild pulmonary nodularity. Ultrasound-guided lymph-node biopsy demonstrated non-necrotising granulomatous inflammation with Langhans-type giant cells. Infectious, autoimmune, and vasculitic screens were negative. In the context of recurrent optic perineuritis, systemic granulomatous lymphadenopathy, and supportive CSF findings, these results were in keeping with probable neurosarcoidosis.

Discussion

Optic perineuritis is an uncommon manifestation of neurosarcoidosis and may precede more widespread neurological involvement. Neurosarcoidosis can present with isolated cranial neuropathies, optic nerve sheath inflammation, raised intracranial pressure, or non-specific CSF abnormalities, and may mimic idiopathic or demyelinating optic neuritis. In this case, the combination of recurrent steroid-responsive optic perineuritis, FDG-avid mediastinal and hilar lymphadenopathy, non-necrotising granulomatous inflammation on lymph-node biopsy, and positive paired oligoclonal bands supported a diagnosis of probable neurosarcoidosis according to current diagnostic criteria. The absence of infectious, autoimmune, and vasculitic markers further strengthened this conclusion. This highlights the importance of considering neurosarcoidosis in patients with relapsing optic neuropathies, particularly when MRI demonstrates perineural enhancement or when systemic features (arthralgia, rash, lymphadenopathy) coexist. A structured multidisciplinary approach integrating neuro-ophthalmology, radiology, and systemic investigation is essential in early diagnosis and prevention of complications.

10) Is It Worse than GCA? Atypical GCA Mimic

Eh Chern Timothy Tan , Warrington Hospital
Haya Razzouk , Warrington Hospital
Ali Yagan , Manchester Royal Eye Hospital

Case report

A 55-year-old man presented to the Emergency Eye Department with several days of bilateral blurred vision and nonspecific headache. He was undergoing evaluation for leg claudication. Examination revealed reduced visual acuity bilaterally, normal anterior segments, and fundus findings of retinal hemorrhages with cotton wool spots. Blood tests showed elevated inflammatory markers, and a positive temporal artery Doppler suggested Giant Cell Arteritis (GCA). Carotid Doppler imaging revealed narrowing of the carotid arteries. The patient was admitted, initiated on high-dose oral

steroids, and referred to Rheumatology. However, two days later, he developed a gastric bleed, leading to further investigation and the diagnosis of gastric carcinoma.
Discussion
This case highlights the diagnostic challenges of GCA mimics and emphasizes considering occult malignancy in patients with atypical features or unexpectedly elevated inflammatory markers

11) The perils of the diagnostic shadow: Collapsin Response – Mediator Protein 5 (CRMP-5) Associated Optic Neuritis, Vitritis and Retinal Vasculitis

Priyal Taribagil[1], Lola Ogunbowale[1], Rogan Fraser[1,2], Ailbhe Burke[1,2]
1) Moorfields Eye Hospital, London
2) National Hospital for Neurology and Neurosurgery, Queens Square

Case report

A 67-year-old woman attended the emergency department with a two-week history of progressive right eye pain and frontal headaches, shortly followed by blurring of vision and transient visual obscurations. Her past medical history included chronic lymphocytic leukaemia (stable since January 2023), chronic obstructive pulmonary disease on a background of lifelong smoking, hypertension, and Jessner's lymphocytic infiltration. Initial evaluations did not detect the vitreous cells already present and she was diverted to her Haematology team for evaluation ?transformation of CLL causing optic neuropathy but also booked in for Neuro-Emergency evaluation at Moorfields Eye Hospital. This identified progressive deterioration of vision with bilateral asymmetric optic disc swelling, prominent vitreous inflammation & retinal vasculitic changes including sheathing. She was admitted acutely to the National Hospital for Neurology and Neurosurgery for acute diagnostic work up including contrast enhanced MRI (non-specific lymph nodes), lumbar puncture (raised cerebral fluid (CSF) protein, IgG, LDH and white cell count) & whole body FDG PET and acute biopsy of necrotic mediastinal nodes identified on the latter. High dose intravenous then oral corticosteroid therapy was started with some improvement in vision. Paraneoplastic antibodies revealed positivity for CRMP5, and diagnostic endobronchial ultrasound (EBUS) guided biopsy confirmed small cell lung cancer. The diagnosis of paraneoplastic CRMP5- associated optic neuritis, vitritis and retinal vasculitis was confirmed within four weeks of first presentation with visual symptoms and aggressive chemoradiotherapy was started without delay.

Discussion

This case highlights the risks of diagnostic overshadowing, noting Hickam's dictum that 'a patient can have as many diseases as they damn well please' – diverting this patient to their local Haematology service risked delaying diagnosis of a treatable cancer. The full examination including dilation despite the patient's reluctance was essential to understanding the clinical phenotype and guiding next steps in her timely care. CRMP-5 associated optic neuritis, vitritis and retinitinal vasculitis is a rare paraneoplastic ophthalmological entity. It is most associated with small cell lung cancer but can be seen in conjunction with several other cancers (thymoma, non-small cell, papillary, thyroid, breast and renal). This is a typical presentation. The underlying pathophysiology is thought to be associated with anti-CRMP5 (anti-CV2), an IgG neuronal intracellular antibody which activates cytotoxic T cells mediating neurological manifestations. Management includes steroids, intravenous immunoglobulin and treatment of the underlying malignancy.

12) The Plot Thickens: A Pachymeningeal Puzzle with Dramatic Enhancement

Dr Melissa Murphy, Dr Jonathan Virgo, Dr Tas Braithwaite, Dr Sunjay Parmar
The Medical Eye Unit, Guy's and St Thomas' Hospital, London

Introduction

Hypertrophic pachymeningitis presents a diagnostic dilemma with overlapping imaging features across inflammatory, infectious and neoplastic aetiologies. Neuro-ophthalmic complications may emerge months after initial presentation, with tissue diagnosis helping to solidify the diagnosis prior to immunosuppression initiation. This case demonstrates the progressive nature of inflammatory pachymeningitis and highlights key diagnostic pitfalls.

Case report

A 23-year-old female presented with new-onset persistent right hemicranial headache and general malaise. She had no prior ophthalmic history of note. Initial investigations revealed an elevated ESR at 90mm/hr and a positive Borrelia IgG and equivocal IgM; felt to be false positive following infectious diseases team review. Initial MRI brain showed right hyperintense dural thickening. CSF studies including opening pressure were unremarkable, and CT-PET scan was normal apart from a 1cm right lower lobe pulmonary nodule. Three-monthly serial MRIs noted no progressive dural changes. She was commenced on Amitriptyline for her headaches. Seven-months later, she developed progressive bilateral visual disturbances with diffuse dark spots and photopsias throughout most of the day. Visual acuity measured 6/6 in the right eye and 6/5-3 in the left eye. Colour vision was full with intact 24-2 static visual field perimetry. Bilateral disc swelling was confirmed (ppRNFL: right 167µm, left 276µm) with preserved macular ganglion cell layer (right 0.55mm, left 0.53mm). Repeat lumbar puncture showed an elevated opening pressure at 37 cmH_2O with normal constituents (WCC 1, protein 0.44, glucose 2.9 with paired serum 4.3) and negative gram stain, viral studies and cytology. Abnormal results included raised eosinophils, CRP 6, ESR 39 mm/hr, low iron indices and elevated IgG4 (1.15 g/L). Infective screen including TB, syphilis and HIV was normal, as was ACE. Repeat MRI demonstrated progressive pachymeningeal thickening overlying the right cerebral convexity with progressive dural venous sinus narrowing without thrombosis on MRV. Treatment commenced with acetazolamide 500mg twice daily increasing to 750mg three times daily, alongside oral iron supplementation.

Discussion

This case illustrates hypertrophic pachymeningitis causing papilloedema secondary to dural venous sinus narrowing. Leading differentials include IgG4-related disease and neurosarcoidosis. Key takeaway points include the importance of serial neuroimaging in slowly progressive disease, recognising delayed neuro-ophthalmic manifestations and the necessity of tissue diagnosis in guiding immunosuppressive therapy.

13) Multi-modal imaging in a rare presentation of bilateral optic nerve swelling and choroidal osteomas

Anika Rao[1], Irina Cristescu[2], Hibba Quhill[2], Umiya Harley[2], Susan P Mollan[3,4], Gabriele Berman[5]
1. Department of Clinical Education, Leicester Royal Infirmary, University Hospitals of Leicester NHS Trust, Leicester, LE1 5WW, UK.
2. Ocular Oncology Service, Department of Ophthalmology, Royal Hallamshire Hospital, Sheffield S10 2JF, UK.
3. Metabolism and Systems Science, School of Medical Sciences, University of Birmingham, Birmingham, B15 2TT, UK.
4. Ophthalmology Department, Queen's University, Kingston, Ontario, Canada
5. Birmingham Neuro-Ophthalmology, University Hospitals Birmingham NHS Foundation Trust, Birmingham, B15 2GW, UK.

Case report

A 21-year-old woman with a past medical history of Camurati-Englemann Disease (CED) and choroidal osteomas was referred to neuro-ophthalmology with bilateral optic nerve swelling. She reported headaches, transient visual obscurations and pulsatile tinnitus. Visual acuity (VA) was 6⁄6 in both eyes, with preserved colour vision and a right paracentral scotoma on Humphrey visual fields. Fundoscopy and optical coherence tomography (OCT) showed mild bilateral optic nerve swelling (retinal nerve fibre layer 96/128 microns), subretinal macular fluid (SRF) in right eye without choroidal neovascular membrane (CNVM). Optos and ultrasound imaging confirmed bilateral posterior pole lesions consistent with choroidal osteomas. Prior neuroimaging demonstrated extensive cranial hyperostosis and optic canal narrowing. Lumbar puncture opening pressure was 33 cm H_2O. Acetazolamide was initiated but discontinued due to intolerance, although VA and symptoms remained stable with mild improvement in disc swelling. Six months later she re-presented with worsening VA (6⁄12 bilaterally), progression of right eye scotoma and increased papilloedema on OCT. Low dose acetazolamide was not tolerated. Repeat neuroimaging excluded venous sinus thrombosis and optic canal compression, and neurosurgical review deemed implantable intracranial pressure monitor unfeasible due to marked hyperostosis. Three months later she reported further reduction in right eye vision and OCT demonstrated increased SRF now extending sub-foveally. Intravitreal bevacizumab was given monthly for three months, for the active choroidal osteomas without anatomical response. Furosemide 20 mg once daily was initiated with subsequent reduction in papilloedema. Due to increasing SRF involving both eyes, she was referred to ocular oncology, where photodynamic therapy (PDT) 83 seconds at 50mJ/cm right eye achieved complete SRF resolution at six months but demonstrated outer retinal changes. At 2.5 year follow-up, VA was 6⁄24 with mild bilateral optic nerve swelling (on furosemide 20 mg), bilateral GCL thinning, resolution of SRF and enlargement of choroidal osteomas.

Discussion

Visual decline in our case was multifactorial secondary to retinochoroidal (subretinal fluid, osteoma) and optic nerve involvement (papilloedema due to raised ICP in context of cranial hyperostosis). Intravitreal bevacizumab proved ineffective in treatment of SRF, and PDT, while resolving SRF was followed by outer retinal changes, underscoring the need for careful patient counselling and informed consent. Delivery of care across multiple centres added to the management complexity. This is the first reported case of CED with bilateral choroidal osteomas and demonstrates the value of multimodal imaging and multidisciplinary input in CED.

14) A recurrence after decades of silence

Mubarika Sami, Lily Lai, Charlotte Funnell
Epsom and St Helier Hospitals NHS TRUST.

Introduction

We report a case of MOGAD (myelin oligodendrocyte glycoprotein antibody associated disease) optic neuritis which recurred after an unusually protracted interval of 40 years. The previous longest interval between two episodes reported is 17 years.

Case report

A 58-year-old male presented with subacute reduced vision in his left eye and pain on eye movements. He recalled similar symptoms when he was 18 years old which improved with steroids. There were no further episodes until the current one. On examination his left visual acuity was 0.5 LOGMAR. He had a left RAPD. The colour vision was reduced to 9/15 Ishihara plates and there was a left inferior scotoma on automated visual fields. His left optic disc showed Frisen grade 4 swelling. He was treated with PO methylprednisolone 500 mg OD for 5 days with proton pump inhibitor cover. His vision rapidly improved. The visual fields and Ishihara were normal and there was no RAPD. The optic disc swelling resolved showing a very good steroid response. His MRI head and orbits with GAD showed a long,

enhanced segment of the left intraorbital and intracanalicular optic nerve. There were no brain lesions. Blood investigations included FBC, serum inflammatory markers, CRP, ANA, ACE, syphilis serology, Quantiferon Gold were all normal. Aquaporin 4 antibody and anti MOG antibody were also requested. Three weeks later his right vision was similarly reduced with pain on eye movement and was subsequently treated with PO methylprednisolone 500mg OD for 5 days with PPI cover. The vision improved in the right eye but there was residual optic disc swelling. Immunology results showed negative Aquaporin 4 antibody while MOG antibody was reported weakly positive. Neurology investigations included MRI spinal cord which showed no lesions and lumbar puncture with no CSF oligoclonal bands. He was started on PO Prednisolone 1mg/kg/day and gradually weaned off over 4 months. During this time his vision remains stable and the right optic disc swelling resolved. Due to the long period of disease inactivity, long term treatment was not commenced with a second line immunosuppressant.

Discussion

The MOG Ig antibody was identified in 2007 in patients with ADEM and is a transmembrane protein expressed on the myelin sheath's outer surface hence it is likely to be targeted by antibodies. It is distinct from Aquaporin 4 antibody discovered in 2004 which targets astrocytic foot plates. MOG Ig antibodies also cause encephalitis and transverse myelitis.

15) Importance of Early Detection and Treatment of Periorbital Involvement in Chronic Recurrent Multifocal Osteomyelitis in Children

Ji Yun Bog
Nottingham University Hospitals NHS Trust

Case report

We report the case of a 13-year-old girl with chronic recurrent multifocal osteomyelitis (CRMO) presenting with periorbital involvement and compare her management with published cases. The patient presented to the Queen's Medical Centre Eye Unit in Nottingham, UK, with periorbital pain, lid swelling, and ophthalmoplegia. Admission and treatment were delayed by five days following initial presentation, prompted by CT and MRI findings showing marked periorbital and superior orbital inflammation with front dural enhancement, and likely frontal sinus wall osteomyelitis. Intravenous cefuroxime and metronidazole were commenced, followed by right frontal sinus trephination and examination under anaesthesia of the nasal cavity. She was subsequently discharged; however, she was re-admitted in 36 days due to interval MRI scans demonstrating new right periorbital swelling with increased frontal sinus and dural enhancement, indicating worsening infection. She was treated with a 12-week course of IV meropenem, and underwent a draf 2a procedure with suprabrow incision for bone curettage with a combined approach under oculoplastics and ENT following a skull base multidisciplinary review. Microbiology and histopathology results were unremarkable beyond chronic osteomyelitis, and whole genome sequence identified no pathogenic mutation explaining the presentation. The patient's blood and swab cultures have remained negative for organisms throughout her admissions.

Discussion

CRMO is a rare inflammatory disorder of non-infectious nature with periods of exacerbations and remissions. It was long believed to be a benign, self-limiting disease, but recent studies show that physical damage may persist in up to 50% of patients. It is mainly a diagnosis of exclusion based on clinical data, but usually requires biopsy and histology to confirm the diagnosis. It can manifest as an isolated bone lesion, but can affect multiple sites; periorbital involvement is rare with limited evidence on diagnosis and management. Delayed diagnosis may result in irreversible vision and ocular functional loss. In this case, despite a known CRMO background, treatment and admission were delayed due to normal early clinical findings, highlighting that CRMO can present in well-appearing

patients without objective examination or laboratory findings. Imaging for bony inflammation is important when assessing patients with eye findings and a known CRMO diagnosis. Current literature supports diverse therapeutic approaches, with NSAIDs as common first choice, ranging between biologics, corticosteroids, and bisphosphonates. Further research is needed to assess the safety and efficacy of different drugs, especially in children and in rare cases of periorbital involvement of CRMO, to prevent long-term physical and psychological damage.

16) When You Hear Hoofbeats: TB Pachymeningitis Masquerading as Idiopathic Intracranial Hypertension

Dr Mohamed Bakhit 1, Mr Lewis Thorne 2, Dr Katherine Miszkiel 1,2, Dr Ailbhe Burke1,2
1Moorfields Eye Hospital, London
2 National Hospital for Neurology and Neurosurgery, Queens Square

Introduction

Idiopathic intracranial hypertension (IIH) is a common diagnosis in female patients with high body mass index (BMI) presenting with raised intracranial pressure (RICP) and papilloedema, and secondary causes must always be excluded. Tuberculous pachymeningitis (TB-PM) is a rare manifestation of central nervous system tuberculosis, capable of mimicking IIH both clinically and radiologically.

Case report

A 50-year-old woman presented to Moorefield's Eye Hospital A&E with a two week history of bilateral visual deterioration to 6/60 alongside worsening of chronic headache for which previous unenhanced MRIs were reported as normal. Examination revealed bilateral haemorrhagic optic disc swelling with gross subretinal fluid at the fovea. Initial investigations were judged consistent with IIH including CT head and CT venogram, CSF opening pressure high at 36 cm CSF & normal constituents. She was prescribed acetazolamide and admitted under Neurosurgery Queen Square for temporising lumbar drain to preserve vision while secondary causes were sought. Urgent contrast enhanced MRI was initially mis-reported as normal bar en-plque meningioma but on re-review demonstrated extensive dural thickening and enhancement along the right tentorium and posterior falx. Bloods revealed positive Quantiferon-TB test. Whole body FDG PET revealed a small avid left supraclavicular node & biopsy showed granulomatous inflammation on consistent with tuberculous pachymeningitis. No acid fast bacilli were seen in CSF or on biopsy but optimal histology was not possible due to single small sample. Anti-tuberculous therapy led to complete recovery with resolution of headache, restoration of vision, disappearance of the gross macular subretinal retinal fluid and regression of dural thickening on follow-up MRI.

Discussion

This case highlights the diagnostic challenge of distinguishing secondary causes of raised ICP from idiopathic IIH as well as of reviewing imaging directly. The presence of atypical imaging findings particularly dural thickening and persistent subretinal fluid, should prompt consideration of inflammatory or infective pachymeningitis. Despite negative acid-fast staining, histopathology and clinical response confirmed the diagnosis of TB-PM.

17) Sarcoidosis: When the Third Time's the Charm in Diagnosis

Dr Muhammad Sabeeh Ahmed, Dr Alibhe Burke, Dr Rogan Fraser, Dr Ashir Iqbal, Dr Dalia Ludwig

Case report

A 55-year-old woman presented to Moorfields A&E with several weeks of progressive, painful right visual impairment with right relative pupillary defect, optic disc swelling, & polyarthralgia. Giant cell arteritis was suspected, high dose steroids started and she went on to local tertiary care centre for Rheumatology input. Under local Rheumatology temporal artery ultrasound was negative but ankle & knee ultrasound revealed abnormal intrarticular fluid consistent with inflammatory athropathy. They

ruled out GCA & weaned off steroids after two weeks total treatment. No alternative diagnosis was proposed. Extensive bloods were normal bar mildly raised ESR (35 mm/h) & evidence of prior Hepatitis B infection. She relapsed off steroids & re-presented to Moorfields with superior altitudinal visual field defect. Dr Burke admitted acutely to NHNN for urgent investigations, UCLH Rheumatology opinion. CSF examination was normal bar positive & matched oligoclonal bands. Brain & orbits MRI demonstrated concentric enlargement and enhancement of the right optic nerve sheath. Chronic Hepatitis B infection has been reported as a driver of large joint arthropathy** as well as of optic neuritis* but not optic perineuritis. This was the second putative diagnosis considered but ultimately further bloods and Infectious Diseases input allowed us to rule out this diagnosis. FDG PET–CT revealed intensely avid mediastinal and bilateral hilar lymph nodes, mildly avid pulmonary nodules, and an active right supraclavicular node. Biopsy of the latter showed non-necrotizing granulomatous inflammation felt to be typical for sarcoidosis. Rheumatology took over management including initiating subcutaneous methotrexate as their preferred steroid-sparing immunomodulatory treatment in joint sarcoidosis and monitoring for Hepatitis B reactivation while immunosuppressed.

Discussion

In our patient, the combined imaging, CSF, biopsy and clinical data fulfill accepted diagnostic criteria for probable neurosarcoidosis. This case illustrates again the challenges in diagnosing sarcoidosis as well as the importance of careful testing prior to long-term immunosuppression to avoid subsequent infective complications. Early institution of immunomodulatory therapy is essential to mitigate the risk of permanent optic atrophy & of wider neurological morbidity.

18) From Incidental Finding to Acute Blindness: A Rare Cause of Acute Optic Neuropathy with Remarkable Recovery

Michael Wahba, Mr Hugh Jewsbury, Dr Iliana Georganta
University Hospital Wales- Cardiff University

Case report

A 66-year-old male, with a previous history of Mantle Cell Lymphoma (MCL) treated with chemoimmunotherapy (now on rituximab), T2DM, CKD, and hypertension, was incidentally found to have bilateral optic disc swelling on routine retinopathy screening by his optometrist. He was asymptomatic except for mild visual “ache” and intermittent transparent scotomata. His initial assessment revealed preserved visual acuities (RE 6/6, LE 6/9), bilateral disc oedema, and thickened retinal nerve fibre layer (RNFL) on OCT. CT head, lumbar puncture, and laboratory tests were unremarkable. At 3-week follow-up, findings remained stable, suggesting a benign process. However, a few weeks post his initial presentation the patient deteriorated acutely. He presented with severe vision loss described as "dark at all times," episodic complete blindness, peripheral visual field constriction, and morning headaches. Visual acuity had collapsed to counting fingers bilaterally. Urgent MRI revealed concentric enhancement and restricted diffusion of intraorbital optic nerve sheaths, followed by CSF flow cytometry confirming monoclonal B-cells with characteristic MCL immunophenotype (CD5+/CD19+/CD20+, CD23−), establishing leptomeningeal and optic nerve infiltration. High-dose intravenous methylprednisolone therapy was initiated, with a dramatic therapeutic response evident within hours of the first infusion. The patient reported substantial subjective improvement in visual symptoms within 24 hours, and by day 3 of treatment, he stated that vision had improved approximately 80% with marked clinical recovery in visual acuity. He was then transitioned to oral prednisolone with a scheduled 10mg weekly taper and concurrently initiated on Zanubrutinib (Bruton tyrosine kinase inhibitor) for targeted lymphoma therapy.

At 6-week follow-up, structural and functional recovery was confirmed. OCT demonstrated resolution of RNFL oedema, visual acuities had recovered to 6/5 right eye and 6/9 left eye, and colour vision was

fully restored to 15/15 bilaterally. Resolution of the previously striking optic nerve sheath enhancement was also noted on MRI.

Discussion

This case highlights a profoundly unusual manifestation of Mantle Cell Lymphoma, where secondary intraorbital optic nerve infiltration presented as seemingly benign bilateral papilledema. This case underscores the diagnostic challenge in immunocompromised oncology patients due to the deceptively reassuring presentation considering the absence of conventional signs. Optic nerve involvement may represent the initial clinical manifestation of CNS infiltration by MCL, therefore Ophthalmologists serve as frontline clinicians uniquely positioned to detect CNS malignancy through recognition of atypical optic disc pathology. Early detection could facilitate prompt systemic treatment initiation to prevent disease progression, and as demonstrated in this case can result in complete ophthalmic recovery.

19) Intermittent lid retraction and vertical diplopia secondary to a calcified cavernous sinus lesion

Sivapathasuntharam, C. Ananthavarathan, P., Khaleeli, Z.

Case report

A 26 year old man was referred for a third opinion with intermittent diplopia and right upper-lid retraction. His symptoms began more than a year earlier with vertical diplopia whereby one image moved upward. Five months later, he developed intermittent right upper-lid elevation occurring 10 to 30 times a day. Episodes developed gradually, were brief (seconds to minutes) and were brought on by positional change and Valsalva manoeuvres, although they also occurred at rest. On examination, visual acuity was 6/5 in both eyes. There was no proptosis, his conjunctiva was white, and anterior and posterior segment examinations were normal. His pupils were equal and reactive without a RAPD. His ocular motility was full and this was confirmed on orthoptic assessment with a normal Hess chart. Sensation in V1 was intact. The patient submitted photographs and videos, which demonstrated right upper-lid retraction. An orbital ultrasound did not show evidence of varices. Neuro-imaging revealed a heavily calcified mass in the right cavernous sinus, appearing to arise from the dorsum sella and encasing the adjacent internal carotid artery.

Discussion

The patient's intermittent lid retraction and vertical diplopia most likely represents overaction or irritation of the superior division of cranial nerve III due to the calcified cavernous sinus lesion. Differential diagnoses for a calcified cavernous sinus mass include tumours arising from bone such as chordomas and chondrosarcomas, as well as tumours arising from nerve tissue including an atypical meningioma or a nerve sheath tumour.

20) Patients Aren't Textbooks: Navigating Diagnostic Uncertainty

Saba Ishrat[1], Akbar Hussain[2], Gulmeena Hussain[1], Wendy Adams[1]
[1] Sunderland Eye Infirmary, Sunderland, UK
[2] The University of Sunderland, Sunderland, UK

Case report

A 57-year-old lady presented with a two-week history of painless blurred vision in her right eye. Visual acuity was 6/7.5+2 in the right eye and 6/7.5+1 in the left eye, with a right-sided grade 1 relative afferent pupillary defect. Fundus examination revealed marked right optic disc swelling (grade 4) with circumferential peripapillary haemorrhages and a small vitreous haemorrhage; the left eye was normal. Colour vision assessment with Ishihara plates demonstrated 16/17 in the right eye and normal in the left. MRI brain and orbits with contrast and MR-venography were unremarkable. Humphrey visual field testing showed a right inferior altitudinal defect, and an initial diagnosis of atypical NAION was made. As disc haemorrhages and vitreous haemorrhage are unusual features of NAION, fundus

fluorescein angiography (FFA) was performed to exclude other causes of optic disc oedema. Three weeks later, repeat blood tests revealed rising inflammatory markers (ESR 52mm/hr; CRP 69 mg/L), coinciding with the onset of new headaches. Given concern for an atypical presentation of giant cell arteritis (GCA) - despite the patient being younger than the typical demographic - she underwent an urgent temporal artery biopsy and was commenced on oral steroids. Biopsy results are awaited.

Discussion

This case demonstrates the difficulty in differentiating atypical NAION from GCA when initial presentation does not fulfil classic diagnostic criteria. Although early symptoms aligned with NAION, several atypical findings (pronounced disc swelling, extensive peripapillary haemorrhages, and a small vitreous haemorrhage) raised suspicion for other causes. These features prompted FFA to rule out alternative optic neuropathies such as inflammatory, infiltrative or vascular causes. The patient's age was atypical for GCA, contributing to the initial lower clinical suspicion. However, GCA can present without systemic symptoms and may evolve over time. Later development of headaches and significant rise in ESR and CRP were key turning points that shifted diagnostic thinking toward occult GCA. This highlights the value of repeated inflammatory markers and ongoing symptom review, as normal or mildly raised markers early on do not exclude GCA. Prompt initiation of corticosteroids was appropriate, as early treatment reduces the risk of irreversible visual loss, particularly in the fellow eye. Temporal artery biopsy remains the diagnostic gold standard, though clinicians must remain aware of potential skip lesions and potential false negatives. This case reinforces the need for vigilance when clinical features fall outside classic patterns and emphasises the importance of reconsidering the diagnosis when clinical features evolve.

21) A rare presentation of complex ophthalmoplegia with hemiparesis: Nine Syndrome with Peduncular Hallucinosis

Dr Madiwilage Gunarathna, Dr Parisa Moshfeqa, Dr Subramaniam Nagasayi
Hywel Dda Health Board

Introduction

The brainstem and midbrain contain multiple nuclei and white-matter tracts responsible for ocular motility, facial motor control, and sensorimotor function. Concurrent pathological involvement of these structures can give rise to a group of numerically designated neuro-ophthalmic syndromes. Nine Syndrome is characterized by the triad of one-and-a-half syndrome, ipsilateral facial nerve palsy, and contralateral hemiparesis or ataxia, reflecting combined damage to the abducens nucleus or paramedian pontine reticular formation (PPRF), the medial longitudinal fasciculus (MLF), the facial nerve fascicle, and adjacent long tracts within the pontine tegmentum.

Case report

We present an extremely rare case of Nine Syndrome occurring simultaneously with peduncular hallucinosis (PH) in a 69-year-old man with vascular risk factors who presented with acute diplopia, right facial weakness, partial ptosis of the left eye, and left-sided hemiparesis. Oculomotor examination of the left eye showed partial ptosis, impaired adduction, and nystagmus on abduction. Examination of right eye revealed complete adduction failure and abduction failure establishing the clinical picture of right-sided one-and-a-half syndrome. Vertical movements of the eyes were intact and both pupils were normal size and exhibited appropriate reactivity to light. The cerebellar examination and rest of the systemic examinations were unremarkable. During admission, he experienced vivid, well-formed visual hallucinations involving animals, which were recognized as unreal, occurred in full consciousness, and were not associated with psychiatric symptoms. Thus, the new onset of hallucinations in a patient with no prior psychiatric history, presenting with acute stroke, strongly supports the diagnosis of peduncular hallucinosis. Routine laboratory tests and initial CT and MRI imaging were unremarkable. However, progressive neurological deterioration prompted repeat

MRI, which demonstrated a linear focus of restricted diffusion in the inferior pons, confirming an acute pontine infarct involving the paramedian region. The constellation of right one-and-a-half syndrome, ipsilateral lower motor neuron facial palsy, and contralateral hemiparesis supported the diagnosis of Nine Syndrome. The contralateral ptosis was attributed to pseudoptosis secondary to facial nerve dysfunction rather than primary oculomotor involvement. The patient received standard stroke therapy, including antiplatelets, lipid lowering treatment, and rehabilitation, with his regular anticoagulation. His ocular motility and limb weakness gradually improved over one week, and the hallucinations resolved spontaneously without pharmacological intervention.

Discussion

This case highlights an exceptionally rare co-occurrence of Nine Syndrome and peduncular hallucinosis and value of meticulous neuro-ophthalmic examination in localizing pontine lesions. Early recognition allows prompt targeted management

22) Severe Orbital Inflammation and Crystalline Lens Displacement in a Toddler: An Unusual Diagnostic Challenge

Catharine Kwok, West Hertfordshire Teaching Hospitals NHS Trust
Paul Chua, NHS University Hospitals of Liverpool Group
Yamini Krishna, NHS University Hospitals of Liverpool Group
Heinrich Heimann, NHS University Hospitals of Liverpool Group
Damien Yeo, NHS University Hospitals of Liverpool Group & Alder Hey Children's NHS Foundation Trust

Case report

A 17-month-old male presented to A&E with a one-week history of progressive right-sided eyelid swelling and conjunctival chemosis, alongside an upper respiratory tract infection. There was no history of trauma or relevant family history. The left eye was normal. Examination under anaesthesia (EUA) of the right eye was performed which revealed extensive chemosis of the eyelid and conjunctiva, a hazy oedematous cornea and inflammatory debris in the anterior chamber. Intraocular pressure was elevated at 36.1mmHg (left eye 16.2mmHg). B-scan ultrasonography showed a lens-shaped structure in the vitreous with a hypo-echoic centre. Following the EUA, intravenous antibiotics (for presumed orbital cellulitis) were switched to corticosteroids and ocular antihypertensives. CT imaging of the orbits showed a displaced hyperdense opacity and pre-septal inflammation, suggesting a dislocated crystalline lens. Non-accidental injury was excluded by the safeguarding team. There was a clinical improvement in swelling and uveitis over the following days. Repeat EUA and anterior chamber tap two weeks later excluded malignancy. A vitrectomy was planned to remove the lens. On entering the eye, the lens appeared pulled backwards into a stalk of ciliary body and retina, with bare choroid and sclera visible. The lens was left in situ due to high risk of haemorrhage. Vitreous biopsy results were non-specific. Despite treatment and close monitoring, the eye progressed to phthisis bulbi, necessitating evisceration for pain control and cosmesis. Analysis of the eviscerated specimen identified marked degenerative changes, including distorted iris, ciliary body and retina, uveal and retinal pigment epithelial proliferation and clumping and retinal gliosis. The retina was replaced by a chronic inflammatory cell infiltrate comprising multinucleated giant cells surrounding cholesterol clefts and foamy macrophages. Retinal telangiectasia with exudation and choroidal tissue with ectatic vessels were also noted. These confirmed Coats' disease.

Discussion

Coats' disease is a rare idiopathic retinal disease characterised by telangiectasia and aneurysmal vessels with intra or subretinal exudation. This case demonstrates an atypical and advanced presentation. Differentiation from retinoblastoma is critical, as most may present with leukocoria and retinal detachment. In this case, initial EUA and cytology excluded retinoblastoma. Unilateral dislocation of a crystalline lens is associated with blunt force trauma; safeguarding measures were

taken and excluded non-accidental injury. To our knowledge, this is the first reported case of Coats' disease presenting with acute dislocation of the crystalline lens with significant inflammation of the eye and orbit. Recognition of atypical presentations may aid earlier diagnosis and potentially better outcomes.

23) Unusual Bilateral Optic Disc Swelling with Serous Macular Degeneration

Ayman Awadghanem, Anugya Agrawal, Vernon Geh, Aman Chandra, Niral Karia, Beatrice Gallo
Karthika Ullattil

Case report

A 78-year-old woman presented with reduced vision in both eyes. Examination showed bilateral optic disc swelling, vitritis, and serous macular detachment. Her visual acuity had declined to 6/18 on the right and 6/120 on the left. MRI of the brain and spine with contrast was unremarkable, and CT venography confirmed normal venous drainage. Lumbar puncture demonstrated a normal opening pressure of 13 cmH_2O, with only mildly raised lymphocytes ($14/mm^3$), additionally CSF analysis revealed monoclonal bands, and serum electrophoresis identified an IgG lambda paraprotein. Over the following weeks, she developed increasing fatigue, unintentional weight loss, and generalised weakness. Further investigations identified a lytic lesion in the femur and hilar lymphadenopathy, and subsequent haematological work-up confirmed multiple myeloma. Cross-sectional imaging also showed nodular hypertrophy of the right adrenal gland, raising the possibility of organomegaly as part of a broader systemic process. The combination of monoclonal gammopathy, adrenal enlargement, lytic bone disease, and progressive systemic symptoms raised strong clinical suspicion for POEMS syndrome. Although ocular involvement is not included in the traditional POEMS acronym, it is increasingly recognised that patients may present with bilateral optic disc oedema, vitritis and, in some cases, serous retinal detachment. Importantly, the optic disc swelling in POEMS often occurs despite normal intracranial pressure. This has been linked to elevated vascular endothelial growth factor (VEGF) levels, which may drive vascular leakage within the optic nerve head. The term "VEGF-induced papillopathy" has been suggested to capture this distinctive mechanism.

Discussion

This case illustrates the range of ocular findings that can occur in the context of a monoclonal gammopathy and highlights the need to consider plasma-cell disorders and POEMS syndrome when encountering unexplained bilateral optic disc swelling, particularly when routine neuroimaging and CSF pressures are normal

24) Structural-Functional Mismatch in Chiasmal Compression: Tuberculum Sella Meningioma Presenting With a Dense Unilateral Temporal Field Defect Despite Preserved mGCL

Lawrence Langley, Cara Heppel, Sui Wong, Shivaa Ramsewak, Jonathan Virgo
Guy's and St Thomas' Hospital, London

Case report

A 39-year-old woman presented with two months of progressive left visual loss with an associated holocranial headache. Visual acuity was 6/6 in the right eye (RE) and 6/60 in the left eye (LE). 24-2 Humphrey visual fields (HVF) demonstrated a dense temporal defect in the LE respecting the vertical midline. The remaining cranial nerve examination was normal. Fundus examination and OCT imaging of both eyes revealed normal optic discs and a symmetrical retinal structure. Macular ganglion cell thickness was healthy and symmetrical without any suggestion of nasal thinning to correspond to the severe temporal field loss. MRI brain and orbits with contrast showed an avidly enhancing suprasellar lesion elevating and compressing the left aspect of the optic chiasm, consistent with a tuberculum sella meningioma. The patient underwent an Axiem-guided extended endoscopic transsphenoidal/ trans-planum resection with fascia lata repair and nasoseptal flap at King's College Hospital five

months later. The tumour was dissected off the optic nerves, chiasm and both anterior cerebral arteries. Immediate postoperative visual improvement was reported by the patient. Recovery was complicated by cerebrospinal fluid leak and diabetes insipidus. Histology revealed a WHO Grade I meningioma with low risk on the integrated meningioma score. At three months post-resection, best corrected visual acuity (BCVA) was 6/6 (RE) and 6/6-2 (LE) with complete subjective normalisation. OCT revealed the development of binasal macular ganglion cell thinning and on perimetry there was a mild superior bitemporal hemianopia.

Discussion

Tuberculum sella meningiomas are typically slow-growing WHO Grade I tumours, and dense field defects usually coincide with established retrograde ganglion cell loss on OCT. This case is unusual as it presented without macular ganglion cell thinning despite a dense unilateral temporal hemianopia, also known as a Junctional scotoma of Traquair. This mismatch suggests a long-standing but indolent tumour that only recently reached the threshold for axonal compromise, producing rapid functional deterioration before measurable retinal degeneration occurred. This case highlights that a normal OCT does not exclude compressive pathology and reinforces the need to pursue neuroimaging even in the absence of structural GCL changes.

25) A case of autosomal recessive spastic ataxia of Charlevoix-Saguenay (ARSACS)

Luca Seregni, Laura dell'Arti
Fondazione IRCCS Policlinico, Milan, Italy

Introduction

Autosomal recessive spastic ataxia of Charlevoix-Saguenay (ARSACS) is a rare neurodegenerative disorder characterized by progressive cerebellar ataxia, spasticity, and peripheral neuropathy. It is caused by mutations in the SACS gene on chromosome 13q12.12, which encodes the mitochondrial protein sacsin. ARSACS exhibits distinctive retinal abnormalities on optical coherence tomography (OCT): peripapillary retinal nerve fiber layer (pRNFL) thickening, foveal hypoplasia, and characteristic sawtooth appearance of inner retinal layers.

Case Report

A 23-year-old Pakistani male with parental consanguinity presented with childhood-onset progressive gait disturbance, ataxia, fine motor impairment, and hand tremor. Genetic testing confirmed homozygous SACS variant c.4716_4718del, p.(Ile1572del). Ophthalmologic examination (December 2025) revealed best-corrected visual acuity 20/20 bilaterally, normal color vision (17/17 Ishihara), and intraocular pressure 16 mmHg in both eyes. Anterior segment was unremarkable. Fundus photography and dilated examination documented increased visibility of retinal nerve fiber, peripapillary radial retinal folds, normal macular appearance, and preserved foveal reflex. The retinal vasculature and periphery were within normal limits. Spectral-domain OCT demonstrated marked pRNFL thickening (173 µm OD, 168 µm OS), exceeding the upper limit of reference range 121 µm. Macular scans revealed uniform inner retinal layer thickening with marked prominence along the papillomacular bundle. Prominent retinal folds involving these inner layers create the characteristic sawtooth appearance. The patient exhibits mild foveal hypoplasia (Grade 1b per Thomas et al.) featuring shallow foveal pit and preserved outer segments. OCT angiography showed absent foveal avascular zone in right eye and markedly reduced in left eye. Automated perimetry (Humphrey 30-2 SITA) revealed diffuse mild sensitivity reduction without topographically consistent defects.

Discussion

This case exemplifies classical ophthalmic features associated with ARSACS which are a key part of phenotyping the disease prior to genetics. The patient achieved excellent visual acuity and was without nystagmus. All diagnostic OCT hallmarks of ARSACS were present: pRNFL thickening, sawtooth pattern, and foveal hypoplasia. Visual field defects were mild and subclinical, consistent with prior

ARSACS series. These ophthalmic features alongside the characteristic neurologist phenotype are key clues to the underlying diagnosis of ARSACS prior to a confirmed SACS mutation. OCT remains the key ophthalmic evaluation cornerstone for identifying ARSACS among progressive ataxia patients.

26) Beyond Eyes and Reflexes

Betul Ozdemir Ozturk
Emergency Department, Barking, Havering and Redbridge NHS Trust University Hospital

Introduction

Holmes-Adie syndrome (HAS) is a neuro-ophthalmologic disorder characterized by tonic pupils and deep tendon areflexia. Despite its potential systemic significance, it is often underrecognized in clinical practice. This narrative review aims to summarize the clinical features, demographics, and associated conditions of HAS cases reported between 2000 and 2025.

Methods

A comprehensive review of 57 case reports published between 2000 and 2025 was conducted. Data were extracted regarding patient demographics, ocular and systemic manifestations, and diagnostic approaches, including dilute pilocarpine testing for denervation hypersensitivity.

Results

Most patients were female (75%), with common presentations including blurred vision (38.6%), pupil asymmetry (31.6%), and photophobia (17.5%). Isolated Adie's pupil was reported in 28.1% of cases, while 40.4% fulfilled the criteria for Holmes-Adie syndrome. Rare systemic associations included autoimmune disorders (e.g., Sjögren's syndrome, Vogt-Koyanagi-Harada syndrome, en coup de sabre scleroderma), infectious diseases (including COVID-19), paraneoplastic syndromes, and post-vaccination neuro-ophthalmologic events.

Discussion

Diagnosis is supported by dilute pilocarpine testing demonstrating denervation hypersensitivity. Although generally benign, HAS is frequently overlooked. Awareness of its subtle ocular and systemic manifestations is essential for early recognition, multidisciplinary evaluation, and informed management of associated systemic conditions. These findings emphasize that, while often subtle, Holmes-Adie syndrome has significant clinical and systemic implications.

27) Holmes-Adie Syndrome Through a Neuro-Ophthalmologic Lens: Clinical Patterns and Associations

Betul Ozdemir Ozturk
Barking, Havering and Redbridge University Hospital NHS Trust

Introduction

Holmes-Adie syndrome (HAS) is a neuro-ophthalmologic disorder characterized by tonic pupils and deep tendon areflexia. Despite its potential systemic significance, it is frequently underrecognized. This narrative review aims to summarize recent case reports (2000–2025), focusing on clinical features, demographics, and associated systemic conditions, to enhance awareness and guide multidisciplinary evaluation.

Methods

A comprehensive literature search of PubMed and Google Scholar was conducted using the keywords "Holmes-Adie syndrome," "Adie pupil," and "tonic pupil." Case reports published between 2000 and 2025 were included. Studies involving Ross syndrome, Harlequin syndrome, or iridocorneal endothelial (ICE) syndrome were excluded. Demographics, clinical presentation, ocular and neurological findings, associated conditions, and diagnostic methods were extracted and analysed.

Results

Fifty-seven case reports were included. The majority of patients were female (75%) with a mean age of 32 years. Common presentations included blurred vision (38.6%), pupil asymmetry (31.6%), and photophobia (17.5%). Adie's pupil occurred in isolation in 28.1%, whereas 40.4% met criteria for Holmes-Adie syndrome. Rare systemic associations included autoimmune disorders (Sjögren's syndrome, Vogt-Koyanagi-Harada syndrome, en coup de sabre scleroderma), infectious diseases including COVID-19, paraneoplastic syndromes, and post-vaccination neuro-ophthalmologic events. Denervation hypersensitivity confirmed by dilute pilocarpine testing supported the diagnosis.

Discussion

Holmes-Adie syndrome extends beyond ocular manifestations, frequently involving autonomic dysfunction and systemic associations. Awareness of subtle presentations is crucial to avoid missed diagnoses and to identify potential underlying systemic conditions. Emerging associations with COVID-19 infection and vaccination highlight the need for vigilance in contemporary clinical practice. Early recognition allows for appropriate evaluation, management of comorbidities, and patient counseling.

28) The value of eye tracking in the management of neurodegenerative disease: use for detection and diagnosis

Mr Daniel Goldstone
University of Bristol

Introduction

Neurodegenerative diseases cause progressive and irreversible loss of neurons in the brain and central nervous system with healthcare costs over $300B worldwide. There are many different types of neurodegenerative disease, including Alzheimer's Disease and Huntington's Disease, and they impact different brain areas causing clinical symptoms and impacting eye movements. They cause symptoms such as memory loss, cognitive issues, movement disorders, incontinence and many more. This study investigated the link between eye movements and neurodegenerative disease for use in detection and diagnosis.

Methods

The study sample included 18 Normal control and 17 Disease patients of similar ages to complete eye tracking tasks using the BulbiCAM device. Eye movements measured included: pro-saccades, memory-saccades, anti-saccades, smooth pursuit, fixation time and pupil light
response movements. Peak velocity, accuracy, error rates, latency and gain were measured; measurements were repeated a mea was taken.

Results

Neurodegenerative disease was correlated with: slower peak velocity, increased latency, lower accuracy, higher error rates and reduced gain across tests. Results remained after age was considered. There were statistically significant results when compared to normal controls showing decreased peak velocity for dementia patients, decreased smooth pursuit gain for dementia patients and reduced fixation time for all neurodegenerative disease patients ($p<0.05$).

Discussion

The results show statistically significant differences in eye movements between normal control and disease groups. Simple eye movements may be used to aid in diagnosis of these conditions. Future experiments can recruit more participants to evaluate if more diseases can be distinguished or disease severity can be monitored.

29) The utility of the macular ganglion cell layer in pituitary patients

Shveta Bansal, Safiah Casooji

Abstract

We present a case series of 3 patients with pituitary masses highlighting their visual field and ganglion cell layer findings on OCT imaging. 1st patient is a typical presenting showing visual field (VF) and corresponding ganglion cell layer (GCL) loss. 2nd patient is atypical with symptoms of visual disturbance but no significant field loss. OCT shows GCL loss. Pituitary mass was found following neuro imaging. 3rd patient was a functional patient who showed VF loss with 30-2 field however no change on GCL or on neuro imaging. The importance of GCL in particular when VF can't be relied on is highlighted.

30) The Effect of Smoking on Retinal and Optic Disc Microvasculature in Thyroid-Associated Ophthalmopathy

Eda Gumrukcuoglu

Burçin Çakır

Introduction

To evaluate the effects of smoking status on macular, optic disc (OD), and peripapillary vascular density (VD) in patients with thyroid-associated ophthalmopathy (TAO) using optical coherence tomography angiography (OCTA).

Methods

Patients with TAO with a clinical activity score below 3 and healthy individuals were included in the study. Visual acuity (VA), eye examination, and OCTA analysis were performed. VD was evaluated in the superficial and deep layers of the macula; total, superior, inferior, central, inner, superior inner, inferior inner, outer, superior outer, inferior outer, and ETDRS regions. In the OD, VD was analyzed in the total, superior, inferior, intra-disc, and peripapillary regions, including superior and inferior peripapillary areas. The foveal avascular zone (FAZ) was evaluated in terms of area, perimeter, and circularity index. According to these parameters, TAO patients were divided into two groups as smokers (Group 1) and non-smokers (Group 2), and statistically compared with healthy non-smoking individuals (Group 3).

Results

The mean ages of Groups 1, 2, and 3 were 47.94 ± 11.08, 49.76 ± 20.4, and 45.25 ± 7.3, respectively, and no significant difference was found between the groups ($p = 0.71$). The mean VA (logMAR) of Groups 1, 2, and 3 were 0.047 ± 0.11, 0.17 ± 0.009, 0.0 ± 0, respectively, and a significant difference was found between the groups ($p = 0.009$). A statistically significant difference was observed among the three groups in the macular superficial superior segment VD and in the OD papillary area total, superior, inferior, intra-disc, and superior peripapillary VD ($p = 0.043$, $p = 0.030$, $p = 0.018$, $p = 0.021$, $p = 0.034$, $p = 0.021$, respectively). In the analysis between Groups 1 and 2, a difference was found in OD superior peripapillary VD (post-hoc $p = 0.006$). No statistically significant difference was observed among the three groups in FAZ area, perimeter, and circularity index values.

Discussion

In patients with TAO, segmental VD values in the macula, OD, and peripapillary regions were lower than in the control group, independent of smoking. It was determined that smoking increased the effect of TAO and caused a decrease in OD superior peripapillary VD.

31) Landmark trials in Neuro-Ophthalmology: rAAV2/2-ND4 in Leber Hereditary Optic Neuropathy

Sam Sherratt-Mayhew, Midland Metropolitan University Hospital, Sandwell and West Birmingham Hospitals NHS Trust, Birmingham, B66 2QT, UK.

Charles Page, Midland Metropolitan University Hospital, Sandwell and West Birmingham Hospitals NHS Trust, Birmingham, B66 2QT, UK.
Michael Lowe, Department of Neurology, Institute of Neurological Sciences, Queen Elizabeth University Hospital, Glasgow, G51 4TF, UK.
Gabriele Berman, Birmingham Neuro-Ophthalmology, University Hospitals Birmingham NHS Foundation Trust, Birmingham, B15 2GW, UK
Susan Mollan, Department of Ophthalmology, Queens University, Kingston, Ontario, ON K7L 3N6, Canada

Introduction

The growing volume of clinical trials in ophthalmology has made it increasingly difficult for clinicians to stay informed. To address this, Eye has commissioned visual summaries that allow rapid appraisal of key findings. We summarised four major studies - RESCUE, REVERSE, RESTORE and REFLECT - which have recently evaluated the efficacy of rAAV2/2-ND4 gene therapy in improving visual acuity in patients with Leber Hereditary Optic Neuropathy (LHON). This project aims to provide a concise, structured and memorable synthesis of these four phase III trials, presenting essential information for clinicians regarding the therapeutic efficacy of rAAV2/2-ND4 in LHON.

Methods

Two independent reviewers extracted core trial data, including study design, participant characteristics, interventions and outcomes. Information was organised using a standardised visual framework that prioritised simplicity, clarity and accessibility. Iterative feedback from ophthalmology experts ensured both accuracy and clinical relevance. Figures were created using BioRender, selected for its professional quality and suitability for scientific communication.

Results

The RESCUE trial found no significant visual improvement in either eye among participants with less than six months of vision loss. REVERSE demonstrated bilateral visual gains in patients with six to twelve months of vision loss. RESTORE confirmed the durability of these improvements in long-term follow-up. REFLECT reported significantly greater visual improvement in treated participants compared with an external control cohort. All trials found acceptable safety profiles, however there was an increase in ocular inflammation in the intervention arm of the REFLECT trial that has currently delayed licensing of the therapy.

Discussion

Across these trials, rAAV2/2-ND4 gene therapy was associated with measurable visual benefit, particularly in patients within the dynamic disease phase (>6 months since onset). The variable natural course of LHON presents challenges to trial interpretation and design. Substantial evidence from four phase III studies supports the efficacy of rAAV2/2-ND4 gene therapy in improving vision in LHON patients, particularly when administered beyond six months from disease onset. Ongoing research continues to investigate predictors of visual outcomes and refine patient selection for treatment.

32) Illuminating the overlooked: An audit into comorbidity identification in idiopathic intracranial hypertension patients

Hadjiforados L, Heward G, Cooper S

Introduction

Idiopathic intracranial hypertension (IIH) is a chronic neurological condition predominantly affecting women of reproductive age with obesity. While ophthalmological manifestations often dominate clinical attention, IIH's strong association with systemic comorbidities, such as Type 2 diabetes mellitus (T2DM), hypertension, dyslipidemia, and obstructive sleep apnea (OSA), necessitates a broader, more integrated approach to patient care. This audit evaluates the effectiveness of specialist IIH clinics in

identifying obesity-related comorbidities among patients with IIH compared to mixed neurology clinics.

Methods

A retrospective review of clinical records was conducted for patients diagnosed with IIH and a BMI >30. Two groups were identified over a 12-month period: patients seen in mixed neurology clinics (n = 20) and those assessed in dedicated IIH clinics (n = 20). Screening for comorbidities included blood pressure (BP) for hypertension, HbA1c and lipid profile testing for T2DM and dyslipidaemia, and completion of the STOP-BANG questionnaire to assess risk of OSA. General practice records were reviewed to determine whether these comorbidities had been screened for within the preceding six months.

Results

Out of the 20 patients seen in the first dedicated IIH clinics, BMI was recorded in 9/20, BP and HbA1c were recorded in 4/20 for each, and a lipid profile was recorded in 3/20. Screening for OSA was completed in all. Subsequently, rates of screening improved as an orthoptist joined the service. Among the 20 patients seen in the mixed neurology clinic, BMI was recorded in 11/20. HbA1c and OSA were screened for in only one patient. BP and lipid profile were not assessed in this group.

Discussion

Dedicated IIH clinics demonstrated greater consistency in identifying obesity-related comorbidities, particularly OSA, compared with mixed neurology clinics. Logistical challenges affected both settings, with limited consultation time occasionally resulting in incomplete screening. The presence of an orthoptist in the IIH clinic improved workload distribution and consultation flow. Introducing a pre-consultation questionnaire enhanced efficiency and structure. Dedicated IIH clinics also allowed for more time to focus on discussions on weight management. Specialist IIH clinics provide a more comprehensive framework for holistic care, facilitating earlier identification and management of comorbidities that may impact overall health. Integrating structured screening and multidisciplinary input into neurology pathways is a crucial step toward patient-centred care that extends beyond the optic nerve.

33) Assessing Adherence to IIH Diagnostic Guidelines: A Retrospective Audit of Imaging and Management Delays

Dr Rhys Gitau-Jones, Ms Victoria Nowak
Musgrove Park Hospital, Somerset Foundation Trust

Introduction

Idiopathic Intracranial Hypertension (IIH) is a condition characterized by increased intracranial pressure without an identifiable cause, often presenting with papilloedema. Prompt diagnosis and management are critical to prevent vision loss. This audit aimed to evaluate the timeliness of neuroimaging and lumbar puncture (LP) in patients presenting with papilloedema or suspected IIH at Musgrove Park Hospital. Secondary aims included assessing factors contributing to delays and reviewing management approaches.

Methods

A retrospective audit was conducted on patients attending the neuro-ophthalmology service between 2023 and 2025. Patients were identified via hospital databases and included if presenting initially with papilloedema or suspected IIH. Exclusion criteria were prior IIH diagnosis elsewhere, missing records, or incomplete investigations. Data collected included dates of presentation, neuroimaging (CT/MRI), venography (CTV/MRV), and any documented reasons for delays. The audit adhered to consensus guidelines established in 2018, which recommend brain imaging within 24 hours and LP to confirm diagnosis.

Results
Fifty-seven patients were included, predominantly female (95%), with 82% initially diagnosed with papilloedema or possible IIH. All patients underwent neuroimaging; 65% received their first scan within 24 hours. The mean time to CT was 1.2 days, while MRI was slower, with a mean of 27 days.
Discussion
The audit revealed suboptimal adherence to the recommended 24-hour imaging standard, with delays not fully explained by patient acuity. Contributing factors included preference for MRI over CT initially, outpatient imaging requests for low-risk cases, and unfamiliarity with referral pathways. Limitations included a small sample size, retrospective data quality issues, and outliers affecting mean values. Recommendations focus on updating registrar training, establishing clear referral pathways and hospital protocols for acute papilloedema. These measures aim to improve timely diagnosis and management, reducing the risk of vision loss in IIH patients.

34) Beyond Pressure: Resolution of Papilloedema After Anaemia Correction in Patients on the IIH Pathway
Mr Hassan Ahmad, Ms Shveta Bansal
Introduction
Idiopathic intracranial hypertension (IIH) is characterized by raised intracranial pressure in the absence of an identifiable structural lesion and typically affects young women of childbearing age. Anaemia, particularly iron deficiency anaemia, has been increasingly reported as a potentially modifiable factor in patients with IIH, but data remains limited and heterogeneous.
Methods
This retrospective case series describes three female patients managed on an IIH pathway who were found to have significant anaemia (one aplastic anaemia, two iron deficiency anaemia) and in whom targeted haematological treatment was instituted alongside standard IIH therapy. Demographic data, visual function, fundus and OCT findings, cerebrospinal fluid opening pressure, neuroimaging, haematological parameters, treatment course and longitudinal outcomes were extracted from electronic records.
Results
All three patients presented with symptoms and signs consistent with IIH, including headache, papilloedema and, in one case, abducens nerve palsy, with opening pressures of at least 26–35 cm H_2O and normal neuroimaging aside from transverse sinus stenosis in one patient. Each patient was treated with acetazolamide and monitored in a neuro ophthalmology service, but papilloedema persisted or only partially improved until the anaemia was actively corrected with iron supplementation, transfusion or treatment of aplastic anaemia. Following stabilisation or normalisation of haemoglobin, two patients demonstrated complete resolution of papilloedema with preserved visual acuity and visual fields, allowing successful withdrawal of acetazolamide and discharge. The third patient showed rapid resolution of disc swelling but was left with optic atrophy and ganglion cell layer loss, highlighting the risk of delayed recognition despite subsequent haematological optimisation.
Discussion
This case series supports anaemia as an important and potentially reversible contributor to the disease course in patients treated for IIH. In all three cases, meaningful structural improvement of the optic discs coincided with correction of the underlying anaemia, whereas papilloedema had remained stable despite standard IIH therapies alone. Routine screening for anaemia, close collaboration with haematology and timely correction of haematological abnormalities should be considered integral to IIH assessment and management to optimise visual outcomes and potentially reduce the need for prolonged acetazolamide therapy or invasive CSF diverting procedures.

35) Vigabatrin Optic Neuropathy Presenting via Glaucoma Referral Pathways: A Multicentre Case Series from Three UK Hospitals

A. Bushnag, G. Reynolds

Purpose

To describe the clinical, perimetric and optical coherence tomography (OCT) features of adult vigabatrin optic neuropathy detected in glaucoma clinics, and to compare real world monitoring with the Royal College of Ophthalmologists 2008 guidance on vigabatrin ocular toxicity24. Emphasis is made on the differentiating clinical and imaging signs between Glaucomatous optic Neuropathy (GON) and Vigabatrin Optic Neuropathy (VON)

Methods

Multicentre retrospective case series from three eye units (Bristol eye hospital, Royal united hospitals Bath, and Cheltenham general hospital) of six adults exposed to vigabatrin who attended glaucoma clinics between 2003 and 2025 after community optometrist referral for suspected glaucoma. Clinical review and diagnosis was conducted by a consultant glaucoma specialist (GR), with recording of epilepsy diagnosis, vigabatrin dose and duration, visual acuity, IOP, central corneal thickness (CCT measured at glaucoma clinic), disc appearance, visual fields, OCT retinal nerve fibre layer (RNFL) and macular parameters, and any electrophysiology. Screening and follow up were compared against the Royal College recommendations.

Results

Preliminary data from six patients identified a mean age of 64.2 years (range 49–78) and mean vigabatrin duration 12.2 years (6–30). 4 patients had treatment prior to 2003, with two remaining on vigabatrin at the time of presentation. Most had mid teens IOP at presentation (mean 15.8 mmHg right, 16.6 mmHg left) and four of six had been labelled or treated as glaucoma within secondary care before recognition of vigabatrin optic neuropathy. All demonstrated a characteristic pattern of bilateral, relatively symmetric, predominantly nasal or concentric peripheral visual field loss with relative central and temporal sparing, with characteristic nasal OCT RNFL thinning and/or optic disc pallor in the context of vigabatrin exposure.

Discussion

Vigabatrin optic neuropathy continues to represent an underrecognized cause of visual field loss in the adult population. Despite identification of its toxic effect on the optic nerve over 25 years ago, there remains a cohort of individuals who were not identified as having toxicity during this period. Due to increased availability of community visual fields testing, such patients can present via glaucoma referral pathways and may be misdiagnosed as primary open angle glaucoma. Without awareness of the key characteristic visual field and OCT findings, such patients remain misdiagnosed and overtreated for presumed normal-tension glaucoma, with a risk for unnecessary surgical intervention. This case series highlights distinguishing features, describes the misclassification within glaucoma clinics, and identify gaps between national vigabatrin screening guidance and actual practice.

36) Venous Sinus Stenting in Idiopathic Intracranial Hypertension: the impact of Extrinsic versus Intrinsic Venous Sinus Stenosis

Sara Syed1, Safiah Casooji2, Siddhartha Wuppalapati2, Shveta Bansal2
1 - Manchester Royal Eye Hospital, Manchester
2 - Lancashire Teaching Hospitals NHS Foundation Trust, Preston

Introduction

Idiopathic Intracranial Hypertension (IIH) is a potentially sight-threatening condition. Interventions to reduce intracranial pressure (ICP) include CSF diversion by surgical shunts, or, less invasively, venous

sinus stenting (VSS). Cerebral venous sinus stenosis, commonly seen on imaging, can be extrinsic (external compression from brain parenchyma), or intrinsic (internal narrowing of the sinus itself).

Purpose

To review the effect of VSS on visual function and to investigate whether VSS outcomes differ between patients with extrinsic versus intrinsic venous sinus stenosis.

Methods

We conducted a retrospective review of patients with IIH undergoing VSS at a tertiary hospital. Data collected included pre- and post-stenting symptoms, visual acuity (VA), optic disc swelling, visual fields and recurrence. A neuro-radiologist categorised cases into extrinsic or intrinsic venous sinus stenosis.

Results

13 patients who underwent VSS between 2016 and 2024 met the study criteria. Most were female (12/13; 92%), with a mean age of 31 years. 6 patients (46%) had intrinsic stenosis, 7 (54%) had extrinsic stenosis. Pre-stenting, 11 patients (85%) were clinically obese, all were on Acetazolamide and all had headaches. 2 intrinsic stenosis patients reported pulsatile tinnitus. 8/13 patients had VA of 0.2logMAR or worse (range -0.1 to 0.8). All patients had disc swelling in one or both eyes (Grade 1-3 Frisen grading). Humphrey 30-2 visual field (HVF) testing showed pathology in 6 eyes of 3 patients (enlarged blind spot or diffuse loss), with a reduced visual field index (VFI) of 12-94%. Post-stenting, headaches resolved in 8/13 patients (62%). Of the remaining 5 patients, 3 had extrinsic stenosis and 2 had intrinsic. Tinnitus resolved in one of the two patients with tinnitus. VA was stable or improved in 9/12 patients (75%). The remaining 3 patients (6 eyes) had reduced VA but showed improved or resolved disc swelling (2 extrinsic stenosis; 1 intrinsic). 4 patients (31%), all with extrinsic stenosis, had persistent disc swelling, confirmed on OCT retinal nerve fibre layer analysis. HVF comparison (possible in 4 patients) showed improvement in 7/8 eyes and stability in 1. VFI improved in all 8 eyes. IIH recurred in 5 patients (48%), 3 with extrinsic stenosis and 2 with intrinsic.

Discussion

VSS generally improved symptoms and visual function for the majority. Whilst overall outcomes show no clear difference between extrinsic and intrinsic stenosis groups, persistent optic disc swelling was only seen in the extrinsic stenosis cohort. This may reflect the greater effect of VSS on lowering ICP in patients.

37) Optic disc elevation due to vitreopapillary traction: a potential contributor to overdiagnosis of Idiopathic Intracranial Hypertension

Hana Abukar, Jonathan Marler, Safina Rashid, Louis Clearkin
Royal Free London NHS Foundation Trust

Introduction

False-positive misdiagnosis of Idiopathic Intracranial Hypertension (IIH) is frequent, potentially harmful and delays a true diagnosis. Misattribution of optic disc appearance is a recognised as a source of error. We evaluated surety of designation of optic disc appearance in a large UK neuro-ophthalmology service.

Methods

During 2024-25 all patients designated as IIH were seen by a single clinical team, undergoing standardised evaluation, informed by current UK consensus guidelines .

The contribution of each clinical indicator of IIH to misattribution was estimated for each individual studied.

Results

Overlooked vitreopapillary (VPT) traction was frequently identified as a factor that undermined plausibility of initial IIH designation - in more than 10% of the patient cohort.

Discussion
Analysis of key clinical indicators may allow improved credibility in IIH diagnosis. Diagnostically relevant clinical, OCT, radiological & ultrasound indicators of VPT are presented and an estimation of their influence on the likelihood of diagnosis in IIH. This may increase patient's ability to determine their own preference for intervention.

38) Ocular Myasthenia Gravis and Thymoma on CT Thorax: A Retrospective Audit
Dr Mai Nur Sariah Mair Nasser, Dr Sarah Cooper, Dr Raluca Vasilescu
Introduction
Ocular myasthenia gravis (OMG) patients frequently present through Eye A&E, orthoptics clinics, or after initial review in emergency care or optometry. Early clinical recognition and prompt acetylcholine receptor antibody (AChR-Ab) testing are essential for diagnosis. Importantly, thoracic imaging is required to exclude malignant thymoma, which occurs in approximately 10–15% of patients with myasthenia gravis. Thymoma carries significant implications for morbidity and mortality. Delays in requesting CT thorax may postpone thymoma diagnosis, surgical intervention, and optimisation of MG management.
Aim
To analyse whether there were delays in thymoma diagnosis in a cohort of patients presenting with acetylcholine receptor antibody (AChR-Ab) positive myasthenia gravis to Eye A&E department. Secondary aims included raising awareness of the importance of early thoracic imaging in patients with positive AChR-Ab.
Methods
A retrospective review of patients presenting to the same Eye A&E department with ocular symptoms of MG was undertaken. Twenty who were found to be AChR-Ab positive were analysed in detail. All patients who have been diagnosed with thymoma over a 7-year period from the same Eye A&E department were included. Demographic data, antibody testing, symptom profile, imaging timelines, and final CT/MRI thorax diagnoses were collected. Time intervals between presentation, antibody testing, result availability, imaging request, and imaging completion were analysed.
Results
The cohort comprised 8 females and 12 males, aged 52–89 years (median 71.85; mean 73.5). Ptosis and diplopia occurred in 16 patients each. Median time from antibody test request to result was 25.5 days (range 6–247). Median time from diagnostic confirmation (AChR-Ab positivity) to CT thorax request was 30 days (range 0–405). Four patients had CT thorax requested before antibody results were available; one had no CT data recorded. Median time from CT request to scan completion was 22 days. Imaging was ordered by ophthalmologists in 8 and by neuro-ophthalmologists in 11 cases. Thymoma was identified in 4 of 20 patients (20%).
Discussion
There are significant variability and delay in thoracic imaging after diagnostic confirmation of ocular MG. Given the importance of diagnosing thymoma (20%), earlier and more consistent CT thorax requesting is warranted. Implementing standardised diagnostic pathways, Electronic Patient Record prompts at the time of positive AChR-Ab, and cross-specialty education may reduce delays and improve patient outcomes. The findings support developing a local clinical guideline to ensure timely thymoma exclusion in OMG.

39) The Royal Victoria Infirmary Blues: Optic Nerve Involvement in Patients with Ocular Syphilis in North East England, 2003-2025
Emma Spowart, Aashna Jain, Sinéad Connolly, Ranjeet Pandit
Newcastle Eye Centre, Royal Victoria Infirmary, Newcastle-upon-Tyne, England

Introduction
Following a recent increase in case numbers of ophthalmic syphilis in the north east of England seen at the Newcastle Eye Centre, we present a summary of those with optic nerve involvement.
Methods
Confirmed cases of ocular syphilis with neuro-ophthalmic presentations between 2003 and 2025 were reviewed. Data were collected on demographics; symptoms and signs; ocular comorbidities; HIV status; systemic symptoms; investigation and treatment before diagnosis; and visual outcomes at most recent follow up. Data collection and processing were in accordance with Caldicott principles and approved by information governance. Data collection and statistical analyses were performed in Microsoft Excel using t-test for numerical variables and chi-squared tests for categorical variables.
Results
Thirty-nine patients presented with ocular syphilis with optic nerve involvement. There has been a sharp increase in patient numbers over time: 19 patients between 2002-2023 (average 0.8, range 0-3 per year), 8 patients in 2024, and 12 patients in 2025. Seven patients had coexisting HIV infection; 29 had no ocular co-pathologies; 2 had cataract, 2 had amblyopia, 4 had retinal pathologies, 1 had glaucoma, and 1 keratoconus.
Systemic symptoms were present in 31 patients, most commonly rash (18), oral/genital ulcers (8), fevers/night sweats (7), hearing loss/tinnitus (7), headache (4), weight loss (5), joint pains (2), rectal bleeding (2), and confusion/disinhibition (2). Four patients were referred from infectious diseases or sexual health with an already confirmed diagnosis of syphilis, and 5 from other ophthalmology departments. 17 patients had previously presented to other healthcare services with systemic symptoms of syphilis. 3 patients underwent lymph node biopsy and 1 gastroscopy. Five patients were initially treated as GCA, all with high dose steroids, and 2 had temporal artery biopsies. Visual acuity at presentation ranged from 6/4 to HM (-0.18-2.10 LogMAR, mean 0.52 LogMAR). 21 patients had anterior segment inflammation, 24 had vitritis, and 17 had retinal changes. 9 had only optic nerve signs: optic atrophy in 2 patients, and optic disc swelling in 37, 1 with disc neovascularisation. At follow up, mean VA was 0.35 LogMAR, with 10 patients losing vision and 19 patients gaining vision.
Discussion
This case series describes different neuro-ophthalmic presentations of syphilis, and highlights the importance of considering it as a differential diagnosis and testing for it at an early stage to avoid unnecessary investigations and ensure timely treatment to reduce the risk of vision loss and systemic comorbidities.

40) What are the benefits of using acetazolamide in Idiopathic Intracranial Hypertension? Insights from a subanalysis of the IIH:Weight Trial
Michael C Lowe: Department of Neurology, Institute of Neurological Sciences, Glasgow, UK
Yousef Hyder: Translational Brain Science, Department of Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, UK; Department of Neurology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK
Andreas Yiangou: Translational Brain Science, Department of Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, UK; Department of Neurology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK
James Mitchell: Translational Brain Science, Department of Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, UK; Department of Neurology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK; Department of Military Rehabilitation, Defense Medical Rehabilitation Centre, Loughborough, UK
Kristian Brock: Cancer Research Clinical Trials Unit, University of Birmingham, Birmingham, UK

Susan P Mollan: Ophthalmology Department, Queen's University, Kingston, Ontario, Canada; Translational Brain Science, Department of Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, UK
Alexandra J Sinclair: Translational Brain Science, Department of Metabolism and Systems Science, College of Medicine and Health, University of Birmingham, Birmingham, UK; Department of Neurology, Queen Elizabeth Hospital, University Hospitals Birmingham NHS Foundation Trust, Birmingham, UK

Introduction

Acetazolamide is a commonly used treatment in idiopathic intracranial hypertension (IIH), supported by the results of the IIH Treatment Trial (IIH-TT) demonstrating improved visual outcomes. However, patients may have difficulty tolerating adverse effects. The IIH Weight Trial (IIH-WT) was designed to assess the efficacy of weight management interventions in IIH, demonstrating a correlation between weight loss and intracranial pressure (ICP) and a significant benefit of bariatric surgery. Some patients in this trial were using acetazolamide; we performed a sub-analysis to determine if use of acetazolamide influenced outcomes in this study.

Methods

Participants (n=66) were randomised to bariatric surgery (n=30) or CWI (n=36). Randomisation was stratified for acetazolamide use, but this was determined by the treating physician. Primary outcome was ICP. Secondary outcomes included weight, visual and headache measures. We employed hierarchical regression to analyse outcomes, corrected for baseline differences, between patients taking (n=19) or not taking acetazolamide (n=47). Most participants in the bariatric surgery group stopped acetazolamide by 1 year. A second analysis was therefore performed for the CWI group alone.

Results

There were no significant differences in weight loss or ICP with acetazolamide use. There was a significant within-group improvement in perimetric mean deviation (PMD) from baseline to 12 (+1.1±0.4dB, mean±SE p=0.009) and 24 (+1.9±0.5dB, p<0.001) months in participants not taking acetazolamide in the whole cohort analysis. A similar significant improvement was seen in the CWI group analysis. A within-group improvement was not seen in the acetazolamide group. Headache diary data showed significant within-group reduction in monthly headache days (MHDs) in the acetazolamide group from baseline to 12 and 24 months in the whole cohort and CWI analyses. A significant between-group difference was found favouring acetazolamide at both timepoints (-6.2±3 MHDs at 12 months; -7.5±3.1 at 24 months) in the whole cohort analysis. A similar trend, though not reaching significance, was seen in the CWI analysis.

Discussion

Acetazolamide use did not influence weight loss or ICP in the IIH-WT. Surprisingly, participants managed 'conservatively' (CWI, no acetazolamide) had significant improvement in PMD, whilst participants taking acetazolamide did not. There were improvements in headache with acetazolamide. These results contrast those of the IIH-TT, where improvements in PMD, but not headache outcomes was seen with acetazolamide. This should be interpreted with caution given the small and exploratory nature of this analysis. Our results support that some patients with IIH can be managed conservatively, without poorer outcomes.

41) Anton syndrome: a review of clinical presentation features

Poppy Cross, Fiona Rowe
University of Liverpool

Abstract

Anton syndrome is a rare neuropsychiatric phenomenon characterised by cortical blindness, anosognosia and confabulation. This systematic review collates the clinical and psychological findings from 67 studies to explore the presentations, underlying neurological mechanisms and outcomes

associated with Anton syndrome. The papers were identified through PubMed and Scopus searches using defined inclusion and exclusion criteria and data was extracted based on cause, neuroimaging, examination findings, management and outcomes. The patterns that presented themselves throughout the review included bilateral occipital stroke as the most common cause of Anton syndrome with great variability in outcomes depending on the extent of disease. The pathophysiological mechanisms suggested centre around a disconnection phenomenon between the primary visual cortex and a centralised consciousness awareness system. Despite their bilateral blindness, the patients had preserved pupillary reflexes and fundoscopy, observed with absence of the menace reflex, supporting a cortical diagnosis. Many patients experienced confabulations and anosognosia, posing diagnostic challenges due to impaired self-awareness. This syndrome has significant psychological ramifications as well as physical, causing distress and confusion when addressing their visual deficit and in recovery when adapting to their condition. There were several rehabilitation techniques identified including, alarming visual stimulation training, visual compensatory training and other supportive care. These findings highlight Anton syndrome to be implicated in the fields of neurology and neuropsychology as it can offer insights into self-awareness, sensory misperceptions and neurological deficits.

42) Retinal nerve fibre and macular ganglion cell layer findings due to delayed foreign body reaction post cerebral aneurysmal clipping: 2 case reports

Eleni Konstantinidou[1], Arani Nitkunan[2], Dariush Ahrabian[2], Pawanjit Minhas[2], Andrew Mackinnon[2], Heidi Laviers[1]

1 Moorfields Eye Hospital at St George's Hospital

2 St George's Hospital, London, UK

Introduction

We present two cases of progressive optic neuropathy following anterior-communicating artery aneurysm clipping, in whom optical coherence tomography (OCT) demonstrated a similar distribution of retinal nerve fibre layer (RNFL) and macular ganglion cell layer (mGCL) loss, despite different clinical courses and imaging timelines.

Case 1

A 64-year-old female presented with episodes of painless right visual loss 6 months after aneurysm clipping for subarachnoid haemorrhage. Each relapse was steroid-responsive, evolving to relapsing inflammatory optic neuropathy. Serial magnetic resonance imaging (MRI) showed an enhancing soft-tissue mass encasing the clipped aneurysm, indenting the chiasm and extending into the optic tracts, with gradual evolution from oedema to optic nerve and chiasmal atrophy. Aneurysm recurrence was excluded on vascular imaging. OCT showed diffuse RNFL thinning in the right eye and progressive temporal/nasal thinning in the left, with mGCL loss consisting of diffuse right-eye loss and a vertical, nasal pattern in the left eye that was not present pre-clipping but became established over the follow-up period.

Case 2

A 55-year-old female presented with painful right visual loss and bitemporal field loss one year after aneurysm clipping. MRI demonstrated an enhancing mass encasing the clip, involving the aneurysm sac, chiasm and optic tracts, which was interpreted as inflammatory. High-dose steroids led to radiological improvement and functional stabilisation was achieved with low-dose maintenance therapy. OCT showed diffuse mGCL loss in the right eye and paramacular nasal mGCL loss in the left eye, with temporal RNFL thinning in both eyes, respecting the vertical meridian and remaining remarkably stable over serial scans.

Discussion
In both cases, no muslin wrapping or other irritant materials was used on the clip. A retrospective Japanese review has found that this is a rare (12/2327) delayed complication of aneurysm clipping which is unlike conventional "muslin-induced" or coating related foreign-body reactions. These cases illustrate a reproducible OCT phenotype of post-aneurysm-clipping optic neuropathy, namely diffuse mGCL and RNFL loss in the eye ipsilateral to surgery, with vertical, nasal mGCL loss and temporal RNFL thinning in the fellow eye, consistent with chiasmal and optic tract involvement. The close spatial correlation between this pattern and the enhancing peri-clip tissue supports a chronic inflammatory aetiology rather than pure mechanical compression or recurrent aneurysm. Recognition of this characteristic structural pattern could help differentiate inflammatory peri-clip optic neuropathy from alternative causes of chiasmal dysfunction and supports consideration of sustained immunosuppression alongside neurovascular surveillance.

43) Optic Disc Topography in Adults with Optic Neuropathy: Findings from the National Eye Survey of Trinidad and Tobago

*Tsz Lun Ernest Wong[1], *Anushka Irodi[2], Felyx Wong[1], Debra Bartholomew[3], Vedatta Maharaj[4,5], Subash Sharma[5], Deo Singh[6], Samuel Ramsewak[7], Rupert Bourne[8], Tasanee Braithwaite[9,10]
1. Section of Ophthalmology, Faculty of Life Sciences and Medicine, King's College London, London, UK.
2. School of Clinical Medicine, University of Cambridge, Cambridge, UK.
3. Ophthalmology Department, Port of Spain General Hospital, Trinidad, Trinidad and Tobago.
4. Richmond University Medical Center, Staten Island, NY, USA
5. Department of Optometry, The University of the West Indies, St Augustine Campus, St. Augustine, Trinidad and Tobago
6. Caribbean Eye Institute, Valsayn, Trinidad
7. Medicine, University of the West Indies at Saint Augustine Faculty of Medical Sciences, Saint Augustine, Trinidad and Tobago
8. The Vision and Eye Research Institute, Anglia Ruskin University, Cambridge, UK.
9. School of Life Course and Population Sciences, King's College London, London, UK
10. Centre for Translational Medicine, King's Health Partners, London, UK
*These authors share first-authorship

Introduction
Accurate characterisation of optic disc morphology using optical coherence tomography (OCT) aids differentiation of disease from normal anatomical variation, and supports optic neuropathy diagnosis and classification, but no population-representative data have been reported previously in a Caribbean population.

Methods
This national, population-based, cross-sectional survey using random multi-stage sampling in 120 clusters, with probability proportionate-to-size methods, identified 4263 participants aged ≥40 years in 2014.(Braithwaite 2017, 2020) Comprehensive assessment included dilated biomicroscopy and optical coherence tomography (Topcon 3D 2000), and Humphrey Visual Field Tests (24-2) in a subgroup. Disc and visual field parameters were summarised using median and interquartile range (IQR). If both eyes were affected, the eye with the worse best-corrected visual acuity (BCVA) was analysed; otherwise, the right eye was included.

Results
65.4% (n=2,790) adults attended comprehensive clinic assessment, and OCT imaging was available for 2364. 740 participants had optic neuropathy, of which 20%(N=147) had known glaucomatous optic neuropathy, 22%(N=164) had undiagnosed glaucoma, 56%(N=413) were glaucoma suspects, 2% (N=16) had non-glaucomatous optic neuropathy. These included 54%(N=398) women; mean age 62-

years; 51%(N=372) were African, 37%(N=272) Indian, 11%(N=84) Mixed and 1%(N=6) other ethnicity. 67%(N=493) of measurements were taken from the right eye. Median BCVA (IQR) in the worse seeing eye was 0.1(-0.04, 0.32) logMAR.
The median (IQR) optic disc parameters in the whole population sample (right eye) versus the optic neuropathy group were as follows: disc area 2.45(2.15, 2.79) versus 2.61(2.27,2.99) mm2; cup area 0.82(0.5, 1.22) versus 1.25(0.82, 1.72) mm2; rim area 1.56 (1.3, 1.9) versus 1.33(1.04, 1.62) mm2; cup volume 0.16 (0.07, 0.29) versus 0.27 (0.12, 0.44) mm3; rim volume 0.11(0.08, 0.18) versus 0.1 (0.06, 0.19) mm3; Cup:Disc area ratio 0.34 (0.22, 0.46) versus 0.48(0.34, 0.59); vertical disc diameter 1.88(1.73,1.97) versus 1.9(1.78,2.06) mm; vertical Cup:Disc ratio 0.60 (0.49, 0.70) versus 0.75(0.77,0.93).
Discussion
This is the first study to characterise optic disc parameters using OCT in a racially/ethnically diverse adult population including optic neuropathy in the Caribbean. We identify significant abnormal parameters driven by the high prevalence of glaucomatous optic neuropathy in Trinidad and Tobago.

44) Epidemiology and risk factors associated with optic neuropathy in the Caribbean: findings from the National Eye Survey of Trinidad and Tobago
*Felyx Wong[1], *Anushka Irodi[2], Tsz Lun Ernest Wong[1], Debra Bartholomew[3], Vedatta Maharaj[4,5], Subash Sharma[5], Deo Singh[6], Samuel Ramsewak[7], ☿Rupert Bourne[8], ☿Tasanee Braithwaite[9,10]
1. Section of Ophthalmology, Faculty of Life Sciences and Medicine, King's College London, London, UK.
2. School of Clinical Medicine, University of Cambridge, Cambridge, UK.
3. Ophthalmology Department, Port of Spain General Hospital, Trinidad, Trinidad and Tobago.
4. Richmond University Medical Center, Staten Island, NY, USA
5. Department of Optometry, The University of the West Indies, St Augustine Campus, St. Augustine, Trinidad and Tobago
6. Caribbean Eye Institute, Valsayn, Trinidad
7. Faculty of Medical Science, University of the West Indies at Saint Augustine Faculty of Medical Sciences, Saint Augustine, Trinidad and Tobago
8. The Vision and Eye Research Institute, Anglia Ruskin University, Cambridge, UK.
9. School of Life Course and Population Sciences, King's College London, London, UK
10. Centre for Translational Medicine, King's Health Partners, London, UK
*These authors share first-authorship
☿ These authors share senior authorship
Introduction
Glaucomatous optic neuropathy is a leading cause of vision impairment in the Caribbean, but there is little epidemiological data on the prevalence and cause of other optic neuropathies, including Strachan's syndrome, a triad of optic, sensorineural and painful sensory peripheral neuropathies, particularly associated with B vitamins deficiency, and sometimes reported amongst the Rastafari community observing a strict vegan diet. We aimed to determine the prevalence and causes of optic neuropathy in Trinidad and Tobago, and explore population-level risk factors.
Methods
This was a national, population-based, cross-sectional survey using random multi-stage sampling in 120 clusters, with probability proportionate-to-size methods that identified 4263 eligible participants aged ≥40 years(2014).(1) Diagnosed and undiagnosed disease were ascertained through comprehensive assessment including dilated biomicroscopy, and optical coherence tomography (Topcon 3D 2000).

Results

We assessed vision in 3,589 participants aged ≥40 years (84.2% response rate) with mean age 57.1 ± 11.8 years; 54.4% women; 42.0% African, 40.5% Indian, 16.5% mixed. 65.4% (n=2,790) attended comprehensive clinic assessment. 4.0% (142/3577) participants reported a history of diagnosed glaucomatous optic neuropathy. Other forms of optic neuropathy were reported in 0.14% (4/2,934) of cases (traumatic = 1; optic atrophy of unknown cause = 2; prior bilateral optic neuritis = 1). Additionally, 0.6% (17/2,792) reported a diagnosed neurological disease, and further 3.7% (109/2,934) reported a history of stroke. Examination identified 431 glaucoma or suspected glaucoma cases, 78.2% previously undiagnosed. 16 people (62.5% men) without glaucoma had optic disc pallor from varied causes; 69% were blind and 18.9% had moderate vision impairment in the worse-seeing eye, but 69% retained normal vision in the better-seeing eye. We explored potential population-level risk factors for optic neuropathy. Alcohol was consumed by 42.3% (1180/2792). Binge consumption (defined as women: >6 units/day, men >8 units/day (2)) in the last 30 days was reported in 3.2% (17/536) and 8.02% (52/648), respectively. In addition, 23.1% (644/2792) of participants were current smokers, while 4.9% (137/2792) reported a history of recent illicit drug use, predominantly marijuana. Only 0.15% (4/2646) belonged to the Rastafari movement and none had any neuropathies.

Discussion

This nationally population-representative data offers new insights into the burden of undiagnosed glaucomatous and non-glaucomatous optic neuropathy in the population of Trinidad & Tobago.

45) A Giant Dilemma

Dr Rogan Fraser[1], Dr Dalia Ludwig[2], Dr Dermot Mallon[3], Miss Lola Ogunbowale[1], Dr Ailbhe Burke[1,3]

1. Moorfields Eye Hospital, London
2. Department of Rheumatology, University College London Hospitals Trust (UCLH)
3. National Hospital for Neurology and Neurosurgery, Queen Square (NHNN)

Introduction

Moorfields Neuro-Ophthalmic Emergency service would like to share this small case series of patients with Giant Cell Arteritis (GCA) in whom there was diagnostic dissent for prolonged periods with high risk of preventable blindness overcome only through Neuro-Ophthalmology-led investigation and treatment. We share our findings in the hope they will be of use to UKNOS colleagues who find themselves facing similar challenges, particularly in areas without such ready access to Fluorescein and Indocyanine Green Angiography, whole body FDG-PET, multi-modal neuro-imaging and support – as needed – to image re-interpretation by sub-specialty trained radiologists. GCA remains a vision-threatening emergency in neuro-ophthalmic practice. Occult GCA, that is GCA with exclusively ophthalmic manifestations in the absence of classically described neurological or systemic symptoms, has been reported in approximately 20% of cases in case series. It is well known that inflammatory markers can also be normal or near normal in GCA. While vessel-wall imaging (such as MRA, CTA and temporal artery ultrasound), and PET-CT have emerged as useful, minimally invasive tools in diagnosing cranial GCA potentially obviating the need for temporal artery biopsy their robustness is affected by inter-observer variability in interpretation. This can cause diagnostic uncertainty and contribute to high-risk delays in diagnosis, in particular in patients who require Tocilizumab to achieve remission.

Methods

We present a small case series in whom diagnostic consensus for GCA was only achieved after a prolonged period of diagnostic uncertainty. Contributory factors to these challenges included significant inter-observer variability in interpretation of imaging findings, and variability in reported and elicited symptoms within and across specialties.

Discussion
This affirms the central role for multidisciplinary collaboration in the diagnosis of GCA. It also highlights a potentially vital ongoing role in selected patients for timely access to temporal artery biopsies given their superior specificity when an adequate sample is obtained. This is despite reassuring data around the robustness of vessel wall imaging in diagnosis. At Moorfields we are currently working on refining our own diagnostic pathway, and in collaboration with UCLH Rheumatology, hope to provide a robust update to practice with the aim of helping in such scenarios.

46) The Bus Stop Hypothesis: A Window into Perceived Clustering of Optic Nerve Sheath Fenestration Surgeries
Kennedy O, Jefferis J, Salvi S |
Royal Hallamshire Hospital, Sheffield
Introduction
Optic nerve sheath fenestration (ONSF) is an uncommon and technically demanding procedure used in the management of vision-threatening idiopathic intracranial hypertension (IIH). It is an alternative procedure to cerebrospinal fluid (CSF) shunting, or venous sinus stenting and at Sheffield teaching hospitals (STH) has become our preference. At STH, we have observed that patients requiring ONSF appear to present in clusters, analogous to several buses arriving at once after a protracted interval. Because ONSF is generally required on an urgent basis to prevent vision loss, any clustering may have significant implications for service delivery and training. Elective operations and cancer cases may require delaying, and training on infrequent, high risk surgeries with months of no exposure is problematic. This raises the question of whether ONSF should be performed at centralised national hubs.
Methods
Surgical records were reviewed for a single surgeon at STH from 2011 onwards. All ONSF procedures were identified, and timings between cases was analysed. Descriptive statistics, distribution modelling, and autocorrelation analysis were performed to examine whether clustering existed.
Results
Between 2011-2025, there were 94 theatre slots for ONSFs, a mean of 0.56 surgeries/month (variance 0.56), and 6.7 surgeries/year. The modal number of surgeries/month was 0. The largest period without any operations was 10 months, notably there were also two 9 month gaps between cases, only one of which was post COVID-19. The most cases in a single calander month was 3, occurring just 3 times in this period. Case distribution followed a Poisson distribution of randomly spread events; an autocorrelation function was performed which did not identify seasonal or cyclical patterns of case incidence.
Discussion
The combination of low absolute numbers, protracted periods between cases, and urgent presentation generate the perception of clustering. However, we did not find any statistically significant clustering of cases. Training for surgeries like this is challenging- a specialised fellowship lasting 12 months to develop expertise could begin with a period of 9 months without any cases. A fellow visiting for 3 months, could see no cases. Some months might need 3 urgent theatre slots for ONSFs – at STH which is also an oncology centre, this typically means moving and delaying several elective and/or oncology cases. Centralisation of treatment into national hubs has been effective for other neuro-ophthalmological conditions, for example, neuromyelitis optica disease. A national hub for ONSF that could concentrate expertise, provide swift access to services, and offer training could pave a way forward.

47) Ocular Motility findings in Ocular Myaesthenia Gravis

Hitesh Agrawal, Shveta Bansal, Nabila Shaikh
Affiliation Lancashire Eye Centre, Lancashire Teaching
Hospitals NHS Trust, England

Introduction

Ocular Myasthenia Gravis (OMG) presents with signs and symptoms related to weakness of extraocular and eyelid muscles, which are often the first to be affected before more widespread muscle groups. The diagnosis of OMG can easily be missed because patients may display subtle signs, investigations are often inconclusive, and the features can mimic other neurological conditions. We report a case series of patients referred to an adult ocular motility service over 18 months, diagnosed with OMG. The aim is to describe their presenting characteristics, investigative outcomes, response to treatment, and natural progression.

Methods

Suspected and confirmed OMG patients referred to an adult ocular motility service were prospectively collected over 18 months. Clinical features were documented at the time of diagnosis.

Results

Twenty patients presenting with clinical signs and symptoms were diagnosed with OMG, confirmed by either one or a combination of the following:

1. Positive blood tests for anti-acetylcholine receptor antibodies
2. Positive response to therapy with pyridostigmine +/- oral steroids

All patients presented with diplopia, and nine also exhibited ptosis. Eight were seronegative. The most commonly involved extraocular muscle was the inferior rectus, followed by the superior rectus and superior oblique. Orbicularis weakness was tested in eighteen patients, with positive findings in ten, which may serve as a useful clinical adjunct in diagnosis.

All patients were treated with pyridostigmine, and nine also received steroids or immunosuppressants.

Discussion

Vertical diplopia, especially if variable and accompanied by ptosis, should raise suspicion of OMG. The presence of orbicularis weakness appears to be a strongly suggestive clinical sign that can aid diagnosis.

48) Costs, Quality of Life Impacts, and Health System Factors in Optic Neuritis: A Systematic Review

Anushka Irodi[1], Cara Heppell[2], Olufunmilola Ogun[3], Clare Fraser[4], KD Frick[5], Axel Petzold[6,7,8] Tasanee Braithwaite[2, 9, 10]

1 School of Clinical Medicine, University of Cambridge, UK
2 King's College London, London, UK
3 University of Ibadan, Nigeria
4 Sydney Eye Hospital, Australia
5 Johns Hopkins Carey Business School, USA
6 Institute of Neurology, University College London, UK
7 National Hospital for Neurology and Neurosurgery, London, UK
8 Moorfields Eye Hospital NHS Foundation Trust, London, UK
9 Centre for Translational Medicine, King's Health Partners, London, UK
10 The Medical Eye Unit, Guy's and St Thomas' NHS Foundation Trust

Introduction

Optic neuritis (ON) affects 115/100,000 people in the UK, especially in the most economically productive years of life. The societal economic burden, and health systems infrastructure influencing ON health and vision outcomes, remain poorly characterised globally.

Methods
In this PROSPERO-registered study (CRD420251160964), we systematically searched MEDLINE, Embase, and Web of Science to 04.10.2025 for studies reporting health systems characteristics (governance, resource generation, service delivery, financing) or economic outcomes (costs, quality of life (QoL), disability). We screened in duplicate and extracted data using WHO Health Systems Building Blocks Framework.
Results
From 863 titles, we identified 15 health systems studies and 12 economic/QoL studies published between 1999-2025, representing 13 countries across all income levels.
Critical evidence gaps emerged: National clinical guidelines reported by one country (USA, subsequently retired); comprehensive surveillance by one country (Colombia, 99% coverage); out-of-pocket costs by three studies (India, Canada, USA); waiting times by one (UK); service integration by three (UK, Spain, USA); quality assurance by two (India, Spain); workforce capacity by none. Substantial inequities existed where data were available. MRI access ranged from 100% (Singapore, UK) to 5-13% (Thailand). In India, while subsidies enabled MRI access, 19% patients could not afford contrast, declined antibody testing due to costs exceeding monthly income, and 1/3 discontinued treatment for financial reasons. Even in high-income settings, barriers persisted: formulations of high-dose oral corticosteroids were difficult to obtain in Australian centres and stocked by <50% of Canadian pharmacies.
Economic burden data were sparse. The annual excess direct cost of ON was €1,271-€1,982 in a Danish registry (1,677 ON, of which 402 MS-ON, versus 6708 controls), with productivity loss €2,230-€7,233 per person-year. Transport costs, informal care, and cost-effectiveness analyses were not reported from any setting. One systematic review reported high costs associated with corticosteroid-related adverse events in the USA and UK. Three studies demonstrated persistent multi-domain QoL impairment 5-8 years post-ON despite visual acuity recovery. A systematic review concluded there is no psychometrically valid patient-reported outcome measure for ON.
Discussion
This review exposes profound knowledge deficits impeding evidence-informed ON care. The near-absence of guidelines, registries, and economic data prevents rational resource allocation and quality improvement. Marked access inequities, particularly for diagnostics and medications, suggest widespread suboptimal care, with out-of-pocket expenditure driving treatment discontinuation in resource-limited settings. Priorities include understanding international ON practice patterns, conducting multi-country economic evaluations and developing guidance to support equitable outcomes.

49) National Trends in Hospital Admissions for Optic Neuritis in England: A Retrospective Study
Yasmin Adelekan-Kamara, UCL GOS Institute of Child Health, London, UK; Samineh Yousefi, Imperial College London, Faculty of Medicine
Purpose
To examine national temporal trends in hospital admissions for optic neuritis in England between 2014 and 2024.
Methods
NHS Hospital Episode Statistics databases were reviewed to identify hospital episodes with a primary diagnosis of optic neuritis. International Classification of Diseases codes (H46) were used to extract relevant admissions. The primary outcomes were the rates of optic neuritis-related hospital admissions between 2014 and 2024.

Results
Between 2014 and 2024, there were 11,295 admissions with a primary diagnosis of optic neuritis, accounting for 0.006% of all admissions. The most common admission method was emergency admission (52.7%, 95% CI 51.3%–54.1%). The male to female ratio was 1:2. Adults aged 30 to 34 were most likely to be admitted (12.5%, 95% CI 11.9%–13.1%), followed by adults aged 25 to 29 (11.5%, 95% CI 11.0%–12.1%). The highest annual incidence of optic neuritis-related admissions was 2.2/100,000 population in 2022/23 (95% CI 2.1–2.4) and the lowest was 1.5/100,000 in 2017/18 (95% CI 1.4–1.6). Mean length of stay was consistently between 3 and 4 days across all years. The largest year on year change in optic neuritis-related admissions was an increase of 27.7% in 2021/22 following the Covid-19 pandemic.
Discussion
Optic neuritis disproportionately affects working-age adults, with potential impact on work productivity. Mean length of hospital stay corresponds closely to the typical duration of inpatient treatment for optic neuritis. The increase in optic neuritis related-admissions in 2021/22 may reflect a return to routine clinical practice from reduced hospital activity during the Covid-19 pandemic.

50) Delivery of care for idiopathic intracranial hypertension: report of the service model in Glasgow and audit of new patient referrals
Michael Lowe, Dawn Buchan, Pushkar Shah
Introduction
The prevalence of idiopathic intracranial hypertension (IIH) in the UK is increasing. Diagnosis and management of IIH requires collaboration between ophthalmologists, neurologists and neuro-radiologists; as well as occasionally neurosurgeons or neuro-interventionalists. Delivery of these services varies across the UK.
Methods
We present our 'IIH clinic' model in Glasgow, covering the West of Scotland region (around 2.5 million people). We include recent audit data from a retrospective review of new patients attending the clinic between January and August 2025. We hope to share insights from our experience and stimulate discussions about how care for IIH should be delivered in the UK.
Results
The IIH service in Glasgow is a regional clinic within the department of Neurology, delivered by a neurology-trained Consultant neuro-ophthalmologist with orthoptist support. New referrals are commonly received from ophthalmology or acute medical services following an inpatient diagnosis; patients must have had adequate investigation to exclude secondary causes before referral to the clinic. Between January and August 2025105 new patients were seen. 50 patients had a new diagnosis of IIH. 6 patients had a secondary cause for intracranial hypertension. 40 patients did not have IIH and had pseudopapilledema or normal optic discs. 1 patient had spontaneous CSF rhinorrhoea without papilledema. 8 patients had been referred back to the service, 4 of whom had evidence of relapse. Patients attending clinic are initially seen by an Advanced Practitioner Orthoptist who will take a history, assess vision, take optic coherence tomography images and perform automated kinetic perimetry. Assessments are repeated at every visit. Stable patients may be seen in an orthoptist-led clinic, which runs monthly and has capacity for 60 patients per year; approximately half are able to be discharged. All patients with IIH are offered support and referral to weight management services. 60% of newly diagnosed or relapsed patients were additionally managed with medication. Headache is also assessed and managed with medications which can be prescribed in primary care (access to hospital-based treatment is via referral to specialist headache clinics).

Discussion

Our service continues to see a high proportion of patients who do not have IIH, with rates consistent with our historical audit data suggesting no change over time.

We provide a comprehensive service where vision and optic nerve parameters can be monitored alongside clinical assessment and headache management, minimising the need for a patient to attend multiple appointments and ensuring a consistent message is communicated to the patient.

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