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REVIEW

Papilloedema associated with dural venous sinus thrombosis

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Papilloedema is a diagnostic term used exclusively to describe optic disc oedema associated with increased intracranial pressure. Septic cerebral venous sinus thrombosis has become an increasingly rare cause of papilloedema because of the widespread availability of antimicrobial agents; however, it is imperative for optometrists to maintain vigilance for this pathologic process. Presented is a case of a 77-year-old Caucasian male with a complaint of blurred vision and non-specific, diffuse headache. He had a right sixth cranial nerve palsy and bilateral disc oedema. Raised intracranial pressure was confirmed by lumbar puncture. Neuroimaging, including magnetic resonance imaging and magnetic resonance venography in conjunction with cytological assessment of the cerebral spinal fluid led to a probable diagnosis of mastoiditis causing multiple dural venous sinus thrombi of the superior sagittal and right transverse sinuses. Sequential evaluation of this complex case is displayed along with pertinent differential diagnoses for optic disc oedema and a review of current standards for diagnosis and management of papilloedema from dural sinus thrombosis.

Key words: dural venous sinus thrombosis, papilloedema

Papilloedema is a term exclusively used to describe optic disc oedema associated with increased intracranial pressure (ICP). The cause of disc elevation remains controversial but may be related to an increase in cerebrospinal fluid pressure, causing a direct compressive force within the optic nerve sheath to create the characteristic emergence of ill-defined disc margins and optic nerve congestion. In addition to this oedema, clinically, one can also observe secondary sequelae of nerve fibre layer ischaemia or cotton-wool spots, vessel engorgement, cessation of spontaneous venous pulsation and resultant peripapillary splinter haemorrhages within the adjacent nerve fibre layer.

The non-visual signs and symptoms of increased ICP can vary but typically include headache, nausea and vomiting. The headache tends to be diffuse and classically worse in the morning. Most notable is an alteration in the intensity of the headache depending on postural position, usually worse in resting or recumbence.[1–3] The mechanical cause of headaches related to increased ICP is thought to be a distention of the meninges and cerebral veins.[1] Patients with increased ICP may experience cerebral spinal fluid rhinorrhea due to pressure on the cribriform plate. Typically, this is reported in patients with elevated ICP associated with a mass lesion or previous trauma.[1] Clinically, a patient may also experience symptoms defined as Cushing's triad, which is characterised by raised systolic blood pressure, alteration in respiratory rhythm and bradycardia. Cushing's triad occurs from a predominant sympathetic reflex stimulated by ischaemia when the ICP exceeds the cerebral mean arterial blood pressure.[4] Raised ICP may also cause brain herniation, a potentially life-threatening complication.[5]

Visual manifestations of papilloedema may be quite subtle and even patients with significant papilloedema may be visually asymptomatic. The most common visual presenting symptom is transient visual obscuration. During these episodes, vision may vary from blurred to complete sudden loss of vision. The obscuration tends to be described as grey or black, lasting for only a couple of seconds and usually preceded by changes in posture with recovery of vision being complete and rapid.[1–3] The visual obscuration is related to transitory compression of the optic nerve.[1] Clinically, subjective visual field testing can elicit a multitude of defects but most often there is a concentric enlargement of the physiologic blind spot. Enlargement of the blind spot is secondary to two proposed mechanisms: compression and lateral displacement of the peripapillary retinal nerve fibre layer or acquired peripapillary hyperopia induced by the elevation of the retina with subretinal fluid.[1,2] Acute absolute loss of central vision is atypical, but has been reported in patients with late stage papilloedema secondary to an intracranial mass or meningitis, either acutely or under a chronic process.[1] Patients with markedly increased intracranial pressure can undergo a slowly progressive constriction of the visual field, which can result in permanent loss of central acuity[1] (Table 1).

With raised ICP, patients may also present with a complaint of incomitant diplopia. Most frequently, this diplopia results from a sixth cranial nerve palsy. Cranial nerve six is most vulnerable to increased ICP due to its extended route to the orbit, specifically, due to its sensitive position against the sphenoidal ridge and the petrous portion of the temporal bone with its transverse course over the branches of the basilar artery at the base of the skull.[1,3] Additionally, but much more rarely, patients may present with trochlear nerve palsies due to the compression from increased ICP on the dorsal midbrain or external ophthalmoparesis from compression of varying cranial nerves, which may resolve with normalisation of intracranial pressure.[6]

Disc description	Types	Clinical characteristics
Pseudo-papilloedema	Hyaloid artery remnant	Unilateral, superficial gliosis, may also have Mittendorf dot on posterior lens capsule
	Disc drusen	Elevated disc is not hyperaemic, no oedema of the peripapillary RNFL, autofluorescence with B-scan or red free filter
Unilateral disc oedema	Non-arteritic ischaemic optic neuropathy	Diagnosis of exclusion with normal ESR/CRP, typically associated with hypotensive event, APD present, pale or hyperaemic disc, fellow eye often demonstrates 'crowded disc' appearance, visual field loss
	Arteritic ischaemic optic neuropathy	Mean age 70 years, associated temporal tenderness, headache, jaw claudication, polymyalgia rheumatica; visual acuity less than 6/60, pallid oedema, fellow eye with normal disc cupping, elevated ESR/CRP, positive temporal artery biopsy, visual field loss
	Vascular a) Diabetic papillopathy b) Hypertensive retinopathy c) Retinal vein occlusion	a) Presence of diabetes, hyperaemic disc oedema with venous engorgement, unilateral 60 per cent and bilateral findings 40 per cent, absence of significant optic nerve dysfunction with no to minimal APD or visual field defect b) HTN retinopathy in the presence of markedly elevated blood pressure with associated vasoconstriction of retinal arterioles, flame-shaped haemorrhages and exudates c) Hyperaemic disc swelling with venous engorgement, intraretinal haemorrhages in distribution of affected venule.
	Inflammatory Sarcoid, Lyme, multiple sclerosis *Bartonella*, tuberculosis, systemic lupus erythematosus, Leber's hereditary optic neuropathy	Acute unilateral visual loss, APD present, dyschromatopsia, possible associated anterior chamber reaction or vitritis, vessel sheathing, may have lumpy white appearance with granulomatous aetiology from sarcoid, pain on eye movement and other neurologic symptoms with MS, stellate maculopathy with *Bartonella*, productive cough with TB
	Infiltrative/compressive Syphilis, herpes virus, varicella zoster, toxoplasmosis, toxocariasis, neoplastic infiltration such as leukaemia, neoplastic compression such as lymphoma, meningioma and glioma, thyroid orbitopathy	Infiltrative presents with acute unilateral visual loss, variable dyschromatopsia and visual field loss, APD present Compressive presents as a progressive visual loss with associated signs of proptosis, vessel engorgement and optociliary shunt vessels
Bilateral disc oedema (may rarely have unilateral disc oedema at presentation)	Acute papilloedema	Transient visual obscuration, may be visually asymptomatic in some patients, enlarged blind spot on visual field testing, typically minimal to no APD, minimal to no dyschromatopsia, predominately bilateral disc oedema, obliterated cupping, absent SVP, associated symptoms of headache, nausea, and vomiting

RNFL: retinal nerve fibre layer, ESR: erythrocyte sedimentation rate, CRP: C-reactive protein, APD: afferent pupil defect, HTN: hypertension, SVP: spontaneous venous pulse

Table 1. Visual and non-visual symptoms of increased intracranial pressure[1–4]

CASE HISTORY

A 77-year-old Caucasian man was sent to the eye clinic for evaluation of blurred vision. He had been an inpatient, who was initially admitted for evaluation of acute delirium, persistent low back pain and right ear pain with nausea and vomiting for two weeks. His medical history included hypertension, vertigo, gastric reflux disorder, depression, opioid dependence, alcohol and tobacco abuse, dyslipidaemia and severe degenerative spine disease with scoliosis. On admission, computerised tomography (CT) of the head showed no evidence of acute intracranial process but suggested right mastoid air cell effusion which was confirmed with a temporal bone scan sequence. He was given vancomycin and moxifloxicin for suspected otitis media on admission and an otolaryngological consultation was requested. The delirium was attributed to hyponatraemia, hypomagnesia and hypokalaemia, which abated after correction of these electrolyte imbalances during his hospitalisation. The hypokalaemia was corrected with supplementation; however, the hyponatraemia persisted with low fractional sodium excretion despite volume replacement and the resolution of nausea and vomiting. The sodium did respond to fluid restriction and the discontinuation of hydrochlorothiazide. Antidiuretic hyponaturaemia was likely due to medication-induced and pain-related syndrome of inappropriate antidiuretic hormone (SIADH). He remained afebrile throughout his hospital stay but his complete blood count initially presented leukocytosis (19.11 K/mm^3) with

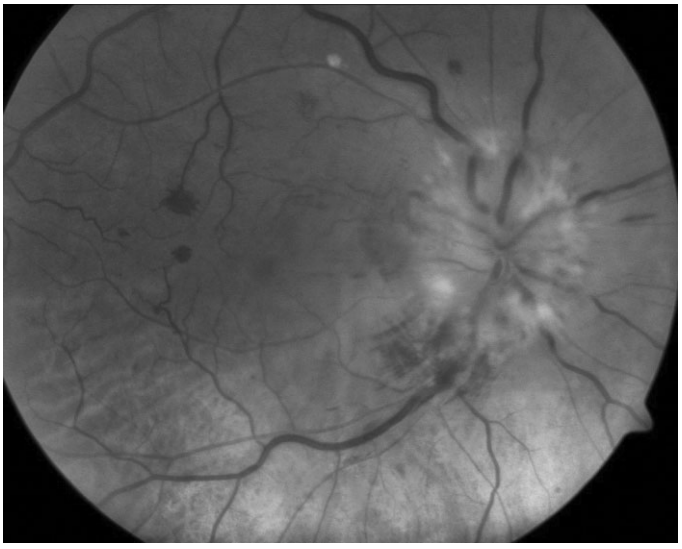

Figure 1. Patient's right eye, showing disc congestion and peripapillary haemorrhages.

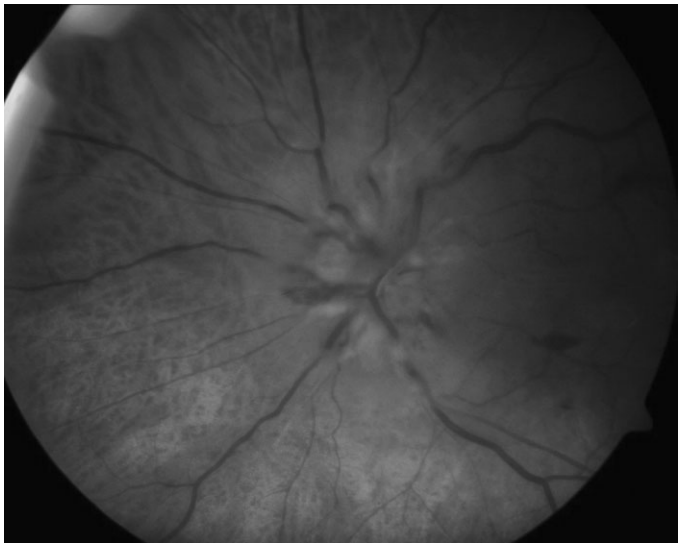

Figure 2. Patient's left eye with obliterated cupping and obscured margins.

a white blood cell count of 18.04 K/mm^3 with identifiable segments at 70 per cent and bands at 13 per cent. Multiple blood and urine cultures yielded no growth. Chest X-ray was normal. His C-reactive protein was undetectable and Westergren erythrocyte sedimentation rate was 2.0 mm per hour.

The otolaryngologist evaluated the patient and found no initial clinical indication of acute mastoiditis, although bilateral longstanding severe sensorineural hearing loss was noted. He was also seen by infectious disease services, which stopped his antibiotics after lumbar magnetic resonance imaging (MRI) confirmed the absence of an occult abscess. The leukocytosis continued to improve, even after discontinuing the antibiotics. At that time, the leukocytosis was considered to be induced by stress because of the recent death of his spouse. Within one week, his pain had resolved. He was then transferred to a long-term care unit for rehabilitation. At this time, he visited the eye clinic for evaluation of blurred vision, which was initially noticed two weeks earlier. The subjective history was difficult to elicit due to the patient's severe hearing deficit. He did deny ocular pain but complained of a diffuse headache, which he was unable to further classify regarding location, intensity, frequency or duration. His ocular history was unremarkable.

Visual acuities were 6/30 in the right eye and 6/24 in the left eye, with no improvement with a pinhole. The attempted subjective refraction was restricted by the patient's hearing disability, but no improvement in acuity could be obtained. Pupils were reactive with a physiological anisocoria but no afferent defect was noted. Extraocular motilities revealed an abduction deficit in the right eye consistent with a right sixth cranial nerve palsy. The patient denied pain on eye movements. Confrontation visual fields were full to finger counting, for both eyes. Anterior segment examination showed mild meibomian gland stasis with 1+ diffuse conjunctival hyperaemia in both eyes. Intraocular pressures were 16 mmHg in both eyes by Goldmann tonometry.

Dilated fundus examination of both eyes revealed posterior vitreous detachments. There were multiple peripapillary haemorrhages associated with bilateral optic disc oedema, which presented with elevated topography, obliterated cupping and obscured margins. Subtle retinal microfolds had formed temporal to the disc (Figures 1 and 2). The patient was diagnosed with bilateral disc oedema and a right sixth cranial nerve palsy, which warranted an immediate head CT scan, which was performed due to its immediate accessibility over MRI. The findings of sixth nerve palsy and bilateral disc oedema led to a provisional diagnosis of papilloedema. Neurologic consultation was requested with recommendation for lumbar puncture, pending confirmation of the absence of an intracranial mass on imaging.

Neurological evaluation found the patient alert with no facial asymmetry, no pronator drift or gross motor weakness in extremities and no inco-ordination. The neurologist's assessment indicated high suspicion for right temporal lobe abscess secondary to mastoiditis on the right, in addition to evidence of persistent leukocytosis, right sixth cranial nerve palsy and bilateral disc oedema. Subsequently, neurology confirmed that there was no space-occupying lesion or definite cerebral abscess on CT scan, and therefore proceeded with a lumbar puncture, which showed an elevated opening cerebral spinal pressure of 55 cm of water. In addition to increased ICP, the cerebrospinal fluid was evaluated and presented a clear colourless fluid with minimally elevated protein at 47.7 mg/dl, nucleated cells at 11 cells/μl with predominant lymphocytes at 68 per cent, the venereal disease research laboratory (VDRL) test was non-reactive, glucose was normal and herpes simplex virus polymerase chain reaction negative. The patient was started on intravenous mannitol and maintained at a position greater than 30 degrees due to increased ICP with headache. He was empirically prescribed acyclovir due to lymphocytosis and intravenous ceftriaxone due to prior evidence of mastoid effusion.

His leukocytosis was monitored until the cerebrospinal fluid VDRL remained nonreactive, mycological culture showed no growth in four weeks and the bacteriology

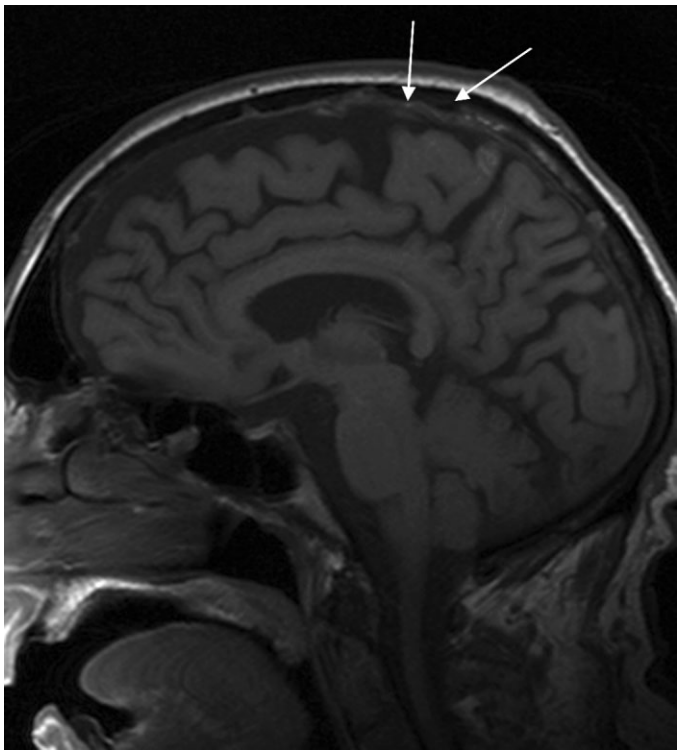

Figure 3. Sagittal T1-weighted image demonstrating complete occlusion of superior sagittal sinus. Hypointense lesion with abrupt discontinuity of smooth contour, diagnostic of thrombosis.

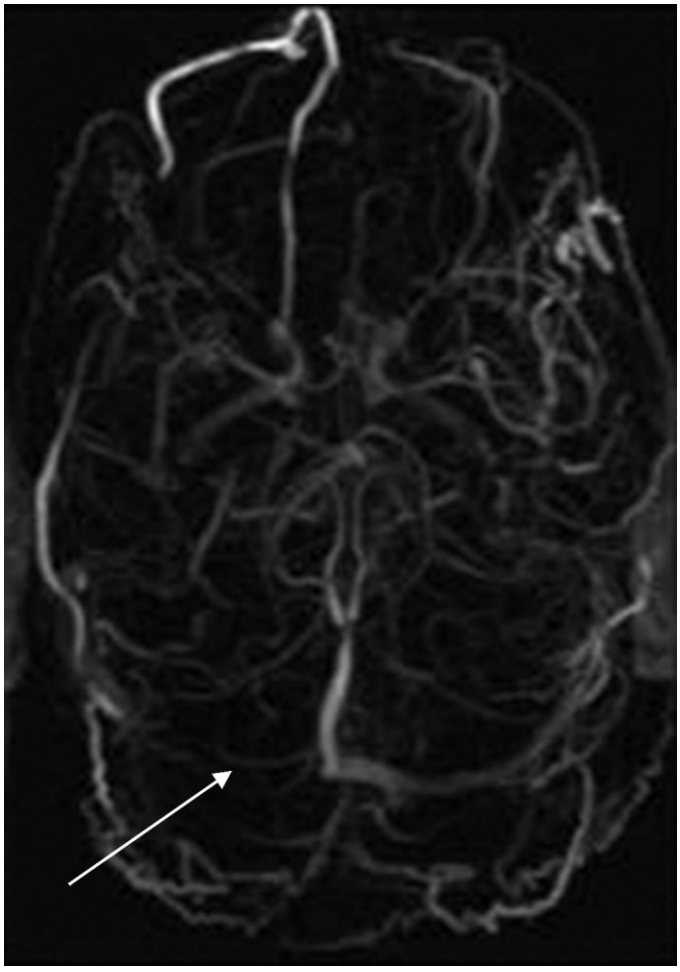

Figure 4. Magnetic resonance venographic image demonstrating occlusion of right transverse sinus.

showed no organisms on Gram stain, negative India ink preparation and no growth on culture. Additionally, cryptococcal antigen, Epstein–Barr virus titre and blood cultures were negative. These cytological results indicated that there was no identifiable growth of organisms within the cerebrospinal fluid or serum, and thus no evidence of primary meningitis. He had been scheduled immediately for MRI and magnetic resonance venography and the infectious disease services agreed with the empirical treatment of the mastoiditis, as being a possible precipitant to either an intracranial abscess or thrombus.

The contrast-enhanced MRI and magnetic resonance venography showed fluid opacification of several bilateral mastoid air cells and no abnormal leptomeningeal or parenchymal enhancement. Most notably, a lack of venous high flow signal indicative of dural venous sinus thrombosis was evident on magnetic resonance venography (Figures 3 and 4). The imaging results were consistent with a complete thrombosis of the superior sagittal sinus and right transverse sinus, which explained the patient's increased ICP. He was immediately started on enoxaparin 1.0 mg/kg twice daily, intravenous heparin and continued on the same antibacterial regimen, namely, ceftriaxone. The antiviral was discontinued, as the cause of his septic thrombosis was thought to be a bacterial mastoiditis.

The patient was continued on intravenous heparin and initial improvement was seen. The patient developed heparin-induced thrombocytopenia and was switched to a low molecular weight heparin. Due to increased impairment of vision noted by the university neurologist, the anticoagulant was discontinued and an immediate lumbo-peritoneal shunt procedure was performed. Unfortunately, within days after the shunt procedure and after the reintroduction of the anticoagulant, the patient developed a cerebellar haemorrhage and passed away.

DISCUSSION

Typically, papilloedema presents with bilateral disc oedema; however, there are published reports of unilateral optic disc oedema under the influence of increased ICP. There are two known reasons for the presentation to be unilateral, namely asymmetric or unilateral optic atrophy and anatomical asymmetry that prevents one nerve from swelling.[7,8] As papilloedema can present unilaterally in patients without optic

Symptoms of increased intracranial pressure

Visual symptoms	Non-visual symptoms
- Transient visual obscuration	- Headache, typically worse in the morning, worse in recumbence or Valsalva manoeuvre
- Enlarged blind spot	
- Diplopia secondary to cranial nerve VI palsy	- Nausea, vomiting, pulsatile tinnitus
- Loss of central vision with chronic cases	- Cerebrospinal fluid rhinorrhoea

Table 2. Disc oedema differentials and clinical characteristics[1,2,7,9–12]

Conditions associated with papilloedema necessary testing	
- Tumours	- MRI with and without contrast
- Arteriovenous (AV) malformation	- MRA or cerebral angiography
- Intracranial haemorrhage	- Non-contrast CT scan
- Infections (meningitits, encephalitis)	- Lumbar puncture with CSF evaluation, MRI
- Inflammatory (sarcoidosis)	- MRI, lumbar puncture with CSF evaluation, serology
- Neoplastic infiltration (lymphoma, leukaemia etc)	- MRI with and without contrast, serology
- Obstruction of venous drainage	- MRI with and without contrast, MRV, CT venography

MRI: magnetic resonance imaging, MRA: magnetic resonance angiography, CT: computed tomography, CSF: cerebrospinal fluid, MRV: magnetic resonance venography

Table 3. Conditions leading to increased intracranial pressure and necessary clinical testing[1,9,10,15,17,19,25–27,29]

atrophy, it must be considered among the differentials for all cases of either unilateral or bilateral optic nerve oedema (Table 2).

Many types of pathology can cause optic disc swelling under the influence of raised intracranial pressure. Intracranial masses, such as cerebral tumours, may cause an increase in pressure through several different mechanisms, including increase of intracranial tissue, diffuse cerebral oedema and blockage of cerebrospinal fluid with direct compression of the drainage system. In evaluation of these, MRI with gadolinium contrast is the preferred imaging test because it has a higher sensitivity to detect pathologic causes of raised cerebral spinal fluid pressure, including neoplastic pathology. A diagnosis of papilloedema can legitimately be conferred when lumbar puncture reveals elevated increased intracranial pressure, with opening pressure greater than 25 cmH_2O.[1,9] Before a lumber puncture can be performed, neuroimaging of the brain is mandated to rule out a space occupying lesion with the potential to cause brainstem herniation during the lumbar puncture procedure.[1]

In addition, any pathology that restricts cerebrospinal fluid absorption into the venous sinuses may also cause increased intracranial pressure and papilloedema. This pathologic mechanism may be seen in cerebral arteriovenous malformations, cerebral venous sinus thrombosis, intracranial haematomas, cerebral neoplasms and cerebral infections. Cerebral arterio-venous malformations cause shunting of arterial blood directly into the venous system, raising the venous pressure. Thus, the arteriovenous malformation does not need to rupture to cause the shift in intracranial pressure.[1] Patent arterio-venous malformations may be diagnosed either by magnetic resonance or cerebral angiography. Additionally, the increased ICP resulting from the arterio-venous malformation may be caused by the inadequate resorption of cerebrospinal fluid secondary to the shunted arterial flow within the venous channels. This same mechanism is encountered for patients who develop a thrombus in a cerebral venous sinus, obstructing venous flow, which results in raised venous pressure. Patients with acute and occasionally chronic subdural haematomas may also develop papilloedema. The optic disc oedema may develop as soon as hours after the haemorrhage or may take weeks of increased ICP.

Infectious causes can manifest increased ICP due to blocked venous resorption. Patients with meningitis or encephalitis may develop mild papilloedema, occurring in only 2.5 per cent of 2,178 cases published by Hanna and colleagues.[10] Considering infectious causes, a lumbar puncture should be performed to analyse both the opening pressure and the contents of the cerebrospinal fluid. Typically, the cerebrospinal fluid is analysed for protein, glucose and cell count. Cytologic findings of leukocytosis may indicate a possibility of meningitis, cancer or atypical infections, including cryptococcus, syphilis or fungal infections. These intracranial infections may be identified by aberrant cytology and detection of antigens in the cerebrospinal fluid, as well as radiographic signs of contrast enhancement (Table 3).

Commonly, a cause for raised ICP cannot be found and this condition is classified as pseudotumour cerebri (PTC) or idiopathic intracranial hypertension (IIH), which is a diagnosis of exclusion, characterised by raised ICP in the absence of a space-occupying lesion or other identifiable pathology. Lumbar puncture reveals normal cerebrospinal fluid contents. IIH can affect an age range of infants to young adults but rarely affects patients over the age of 45.[1] Theories exist for the pathogenesis of idiopathic intracranial hypertension supporting a predilection in obese women of childbearing age and an association with certain medications, such as tetracycline, nalidixic acid, corticosteroids or vitamin A intake.[1,2] One possible cause of idiopathic intracranial hypertension is related to a decrease in cerebrospinal fluid absorption secondary to cerebral parenchymal oedema as a result of an increase in total water content.[1,11]

The diagnosis in this case was elusive prior to the eye examination, which revealed critical findings. Elevated ICP was the leading differential for this case of disc oedema due to the clinical presentation of headache with bilateral optic nerve swelling and concurrent sixth cranial nerve palsy, normal pupillary function with the absence of an afferent defect and full confrontational fields. The briskly reacting pupils and absence of an afferent pupil defect provided evidence against the differentials of

asymmetric or unilateral findings, such as anterior ischaemic optic neuropathy, optic neuritis and infiltrative papillitis. The clinical picture of disc oedema had no associated inflammatory vitreal or anterior chamber reaction and no associated retinitis, suggesting the lack of a direct infectious cause. His blood pressure was 115/71 without significant hypertensive retinopathy, ruling out hypertension as a cause. Computerised tomography was available immediately and was performed because we suspected an infection and wanted to obtain a lumbar puncture as soon as possible. The absence of a space-occupying lesion permitted clearance for the lumbar puncture. Because of the previous concern for mastoiditis, a brain abscess, meningitis and venous thrombosis were also considered significant differentials, thus the MRI, the cerebral venography and evaluation of the cerebrospinal fluid were crucial radiologic procedures. Mastoiditis can cause cerebral abscesses and Kao and colleagues[12] performed a clinical analysis on 53 patients with a diagnosed brain abscess and found the most common predisposing condition was mastoiditis, seen in 19 per cent of their cases. Our patient subsequently underwent MRI evaluation and no abscess was identified; however, magnetic resonance venography confirmed thrombosis in the superior sagittal and right transverse sinuses.

Cerebral venous sinus thrombosis

Obstruction of the intracranial venous drainage system secondary to a thrombus is a vital differential for patients with increased ICP. Both aseptic and septic causes should be considered. The superior sagittal sinus is typically the venous channel affected with aseptic thrombosis in both children and adult patients. Most of these patients have hypercoagulopathy from a haematologic disorder, including deficiency in protein S or C, factor V Leiden mutation, thrombocythaemia, anti-anticardiolipin antibodies, antithrombin antibody and thrombocytopenic pupura.[1] Thromobotic events may correlate with nutritional vitamin B6 and B12 deficiencies leading to elevated homocysteine levels, which are more commonly seen in people with thrombosis. A genetic variant, known as methylenetetrahydrofolate reductase (MTHFR gene mutation) may also lead to hyperhomocysteine levels by the impairment of folate processing.[13,14]

Venous sinus thrombosis may be associated with pregnancy, renal disease, medications, such as oral contraceptives or inflammatory causes, such as Behçet's disease, sarcoidosis and systemic lupus erythematosus. The risk of developing a cerebral venous thrombosis is also increased after a patient experiences head trauma with a higher risk following lumbar puncture, jugular catheter insertion, surgery and drug use. The most common risk factors for patients over the age of 65 are acquired thrombophilia and malignancy.[15] Malignancy is a vital consideration as a risk factor for thrombosis. Patients diagnosed with cancer are in a hypercoagulable state. In fact, up to 11 per cent of patients with cancer are clinically diagnosed with thromboembolism.[16] The most common malignancies associated with thrombosis are in the lung, colon and breast.[17] Not only can the malignancy alone cause a hypercoagulable state for the patient but the tumour can externally compress the vascular wall leading to turbulent blood flow. Septic thrombosis may occur but the incidence has dramatically decreased since the development of effective antimicrobial agents. Infection may spread from venous sources proximal to the adjacent cerebral venous sinuses. For example, otitis media has a propensity to cause septic thrombosis of the transverse sinus, while paranasal sinusitis has a tendency to cause cavernous sinus thrombosis secondary to anatomical proximity. Papilloedema commonly occurs early with cases of venous thrombosis resulting from mastoiditis but develops later when the septic thrombosis affects the cavernous sinus.[1] In fact, venous sinus thrombosis in six to 12 per cent of the adult cohort may be caused by an infection.[18]

When a thrombotic event occurs in the cerebral venous sinus channels, there is a secondary increase in cerebrospinal fluid pressure because of decreased absorption via the arachnoid granules located along the intracranial venous sinuses. Thrombi most commonly affect the superior sagittal and lateral sinuses with an increased incidence of intracranial hypertension, when the thrombus is located in the superior sagittal sinus.[19] This increased incidence is most likely due to the majority of the cerebral cortex venous drainage through the superior sagittal sinus.[1] As a compensation mechanism for cerebral venous thrombosis, a collateral pathway may be initiated with observable dilation of the cerebral veins. Ultimately, the increased venous pressure has the potential to cause decreased capillary perfusion, leading to both vasogenic and cytotoxic oedema.[20]

Clinically, patients with cerebral venous sinus thrombosis will present with classic symptoms or signs of raised ICP. There are three neurologic syndromes that raise concern for cerebral venous thrombosis: isolated intracranial hypertension syndrome, focal syndrome and encephalopathy. Isolated intracranial hypertension syndrome is classified by headache, papilloedema and visual problems related to increased ICP. Headaches are the most frequent symptom of cerebral venous thrombosis and typically are localised with no relationship to the sinus affected. The onset is typically gradual and may resemble a migraine with aura.[1,3,21] Focal syndrome is identified by signs and symptoms related to the anatomical relationship and physiological function of the thrombosed sinus. Isolating the venous sinus affected by the thrombosis may be possible clinically by the patient's presenting signs and symptoms. A cavernous sinus thrombosis will present with mostly ocular manifestations, including orbital pain, proptosis, chemosis and diplopia due to ocular motor palsies.[22] If the sagittal sinus is the occluded channel, the patient will suffer from motor deficits, commonly bilateral, and typically experience seizures. Isolated headaches are the frequent symptom, if the lateral sinus is involved and aphasia is a presentation particularly if the left lateral sinus is affected;[23] Patients are more prone to experience a pulsating tinnitus if the jugular vein or lateral sinus is involved.[24] However, pulsating tinnitus is common in any cause of increased intracranial pressure, including idiopathic intracranial hypertension. Encephalopathy related to cerebral venous thrombosis will present as decreased cognitive function, including possible delirium and lethargy. Initial symptoms may also include motor weakness (37 per cent) and seizures (39 per cent).[18] Elderly patients are more likely to suffer with mental and vigilance issues rather than headaches.

Upon presentation of signs and symptoms of cerebral venous thrombosis, a diagnosis of cerebral thrombosis is typically established with neuroimaging, of which characteristic signs may present in either MRI or CT studies. Sensitivity for detection is at its highest when performing combined brain MRI and magnetic resonance venography. Cerebral CT venography is an additional

option for evaluation of the venous system. Ozsvath and colleagues[25] performed a study on 24 patients to compare the validity of CT versus magnetic resonance projection venography. Cerebral CT venography was superior to magnetic resonance venography, when isolating cerebral veins and dural sinuses; however, both were equivalent in the actual diagnosis of cerebral venous thrombosis. However, there is a risk of radiation exposure associated with CT.

When visualising cerebral venous thromboses on MRI, the signal intensity depends on the age of the thrombus related to the haemoglobin degradation of the clot. The MRI signal is dependent on proton density and interactions. When different chemical products arise from haemoglobin degradation, the result is signal changes in T1 and T2 weighted images.[26] If imaging is performed within the first five days of thrombus formation, there is an isointense signal on T1-weighted images and a hypointense signal on T2-weighted images. When greater than five days, the thrombosed area will become hyperintense on both T1 and T2 images. The variation of signal characteristics increases when the age of the thrombus is greater than one month.[27] On CT, the most common sign identified is known as the empty delta sign, which is a contrast-enhanced radiologic image that demonstrates a hyperdense triangle with central hypodensity within the affected sinus.[28]

Neurologic imaging continues to be the gold standard for diagnosis of cerebral venous thrombosis with no single confirmatory serologic testing available. An elevated D-dimer result may indicate signs of thrombosis; however, normal levels do not eliminate the diagnosis, nor is it specific for cerebral thrombi. Lumbar puncture is an additive test to evaluate for evidence of septic causes of cerebral venous thrombosis, such as meningitis, which is found in 25 per cent of patients with cerebral venous thrombosis.[29] In general, cytologic cerebral spinal fluid abnormalities on lumbar puncture are revealed in 30 to 50 per cent of patients with cerebral venous thrombosis, including increased red blood cell count, increased proteins and lymphocytic pleocytes.[29]

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