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A school of Red Herring

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Abstract

A 66-year-old man with chronic kidney disease presented with painless unilateral vision loss and bilateral optic disc swelling. Vision loss progressed in the affected eye and developed in the fellow eye. Evaluation for infectious, neoplastic, ischemic, and inflammatory disorders was unrevealing. High-dose corticosteroids appeared to stabilize his vision temporarily. Eventually he was diagnosed with papilledema in the setting of transverse venous sinus stenosis and dural arteriovenous fistula. His papilledema resolved, and his vision improved following stenting of the venous sinus stenosis and embolization of the fistula.

Keywords

Papilledema; transverse sinus stenosis; arteriovenous fistula; intracranial hypertension; stenting

Case Report

A 66-year-old Vietnamese man with a history of left cerebral stroke and IgA nephropathy was referred for evaluation of painless vision loss in both eyes. He had experienced slowly progressive vision loss in the left eye for six months. His ophthalmologist measured visual acuities of 20/30 in the right eye and 20/200 in the left eye. There was a left relative afferent papillary defect and bilateral optic disc elevation, more pronounced in the left eye. Static

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perimetry showed enlarged blind spots in both eyes and decreased sensitivity in the left eye. Magnetic resonance imaging (MRI) of the brain without contrast showed old left lacunar and internal capsule infarcts, but no space occupying lesions. Carotid duplex ultrasonography did not show arterial stenosis. Erythrocyte sedimentation rate (ESR) was normal. Over the next two months vision deteriorated in both eyes, and he was referred for consultation. He denied eye pain, headaches, fevers, weight loss, cough, and jaw claudication.

In addition to an ischemic stroke and IgA nephropathy, he had a history of hypertension and hypercholesterolemia. His medications included lisinopril, nifedipine, omeprazole, ranitidine, telmisartan, aspirin, and prednisone 60mg every other day for his nephropathy. Family history was non-contributory. He had last travelled to Vietnam two years prior and was a retired electrician without recent toxic exposures. He denied smoking, alcohol, and drug use.

His best-corrected visual acuity was 20/40 OD. He saw only hand motions OS. He correctly identified 11 out of 11 Ishihara plates with the right eye. Intraocular pressures were 16 mmHg in each eye by applanation tonometry. Anterior segment examination was normal. Dilated examination demonstrated 360 degrees of disk elevation in both eyes associated with peripapillary hemorrhages and vessel obscurations. A shunt vessel was visible on the right optic disc (Figure 1). The left disc was less swollen than the right and had early pallor consistent with axonal loss. The maculae were flat, and the retinal periphery was normal in both eyes. Goldmann perimetry showed an enlarged blind spot in the right eye and a very constricted field in the left eye with some inconstant responses in the paracentral temporal field (Figure 2).

What are the diagnostic considerations?

What would you do next?

Comments by Nicholas J. Volpe, M.D.—This is an older patient with bilateral disc elevation and non-simultaneous progressive vision loss. The differential diagnosis is broad. Ischemic etiologies are an important consideration given his vascular risk factors and cerebrovascular disease, although the chronicity of symptoms and the shunt or collateral vessel on the disc along with the relatively intact visual function OD would make ischemia exceedingly unlikely. Though he lacked systemic symptoms, giant cell arteritis is in the differential in someone this age with bilateral optic neuropathies, although the chronicity argues against this. Inflammatory, infectious, and neoplastic etiologies of bilateral optic neuropathies need to be considered even in the absence of relevant past medical history or systemic symptoms. These etiologies could either infiltrate the optic nerve or compress it intra-orbitally to cause optic nerve swelling. Given the presence of the collateral vessel, papilledema from elevated intracranial pressure (ICP) would be a leading possibility even though he does not have other symptoms of elevated ICP. In regards to his IgA nephropathy, this fairly common condition (1-4:1000) has rarely been associated with multiple myeloma³, which may cause elevated ICP from an intracranial plasmacytoma;^{8,15,21} however, he did not have an intracranial mass lesion on imaging. An association between IgA nephropathy and elevated ICP has only been reported in one abstract.^A Therefore his renal disease is

likely a red herring. His travel to Vietnam is also intriguing and raises the possibility of an atypical infection as a cause of his symptoms.

The chronicity of the disc swelling, the relatively intact visual function and field in the right eye, along with the disc appearance (with significant swelling and shunt vessel) suggest either elevated ICP, a constricting or infiltrating lesion, or possibly an infection. The normal MRI rules out a mass lesion causing elevated ICP; however, this does not exclude papilledema as there are multiple etiologies of elevated ICP that have a normal noncontrast brain MRI. The lack of contrast and fat saturated orbital imaging also limits its utility in excluding inflammatory, infiltrative and compressive optic nerve disease. Bilateral central retinal vein occlusions could have left the patient with swollen discs, shunts vessel, and poor vision. The normal ESR along with the slow progression over months argues against giant cell arteritis and non-arteritic ischemic etiologies.

At this point serological, imaging, and cerebrospinal fluid (CSF) work up are warranted to identify treatable etiologies of his optic neuropathies. Dedicated orbital MRI will include more sections to permit better optic nerve visualization. This should include fat saturated images that make it easier to visualize optic nerve abnormalities. Contrast would also be helpful to identify pathology, though this may be contraindicated because of his poor renal function.

Case report continued—Anti-neutrophilic antibody, angiotensin converting enzyme, lysozyme, Lyme, syphilis and Bartonella serologies, c-reactive protein, and vitamin B12 levels were all negative. He had a mild anemia and, as expected, his BUN and creatinine were elevated. Quantiferon TB-Gold test, a serological screening test for Mycobacterium tuberculosis, (TB), was positive. His sister, a nurse, reported prior negative testing for tuberculosis. Lumbar puncture (LP) was attempted at the bedside, but was unsuccessful. An MRI was scheduled. On follow up one week later, the right eye's visual field showed progressive constriction.

He was admitted to the hospital. MRI of brain and orbits without contrast showed a partially empty sella, increased T2 signal within the intraconal portion of bilateral optic nerves, and prominence of the optic nerve sheaths bilaterally. The major intracranial arterial and venous vascular flow voids were present. Computed tomography of the chest was normal.

An LP was successful, but no opening pressure was measured. The CSF had mildly elevated glucose with normal protein and cell count. Bacterial cultures and cryptococcal antigen were negative. TB polymerase chain reaction (PCR) was negative. Cytology was negative for malignant cells. There were no oligoclonal bands. Sputum samples did not show acid fast bacilli.

What would you do next?

Comments by Dr. Volpe continued—These results are intriguing. The positive quantiferon TB Gold raises the possibility of tuberculous optic neuropathy. Though the CSF did not show inflammation, and CSF testing was negative for TB, this was sampled at a site distant from the optic nerves. He has no other signs of TB involvement of the eye. MRI does

not show inflammation in the optic nerve or basilar meninges, though this must be interpreted with caution since gadolinium contrast was not administered. CT chest did not show pulmonary TB, which means that, if his optic neuropathies were from TB this is a focal process. In my experience, TB involvement of the optic nerve has a much more virulent and rapidly progressive course. I think input from infectious disease experts would be helpful at this juncture.

Prominence of the optic nerve sheaths and partially empty sella, both seen on MRI, are associated with elevated ICP. Unfortunately, ICP was not measured. If ICP is elevated, there is no CSF abnormality or intracranial mass to account for this. Repeat LP for measurement of opening pressure would also provide more CSF for study of possible mTB and neoplastic etiologies as a single sample is insufficient to rule these out.

Inflammatory etiologies have not been eliminated. Though the T2 changes within the optic nerves seen on MRI could simply reflect their injured state, they could also indicate active inflammation. We do not have the benefit of gadolinium contrast imaging to distinguish these possibilities. The various serologies, while helpful when positive, are inconclusive when negative.

It is worrisome that the patient is losing vision rapidly. Consideration could be given to empiric therapy with anti-TB medications and/or steroids.

Case report continued—Quantiferon TB-Gold was negative when repeated. The infectious diseases consultant believed that focal TB meningitis was unlikely given normal CSF constituents, no meningeal changes on MRI, and borderline quantiferon TB-Gold results. The risk of toxicity from anti-TB therapy was judged to outweigh the potential benefit. He received one gram of intravenous methylprednisolone daily for three days for possible inflammatory optic neuropathy with close in-patient monitoring.

An LP performed on the last day of his high-dose corticosteroid regimen measured an opening pressure of 5 cm of water. CSF TB culture showed no growth. Paraneoplastic evaluation included abdominal ultrasound, which showed a complex renal cyst, and serum testing for anti-CV-2 (CRMP-5), which later returned negative. A biopsy of the optic nerve sheath was considered for sampling of the peri-optic nerve fluid space and potentially relieving elevated optic nerve sheath pressure, but was deferred out of concern for procedure-associated morbidity. He was discharged on his baseline dose of prednisone 60mg every other day with unchanged vision.

What now?

Comments by Dr. Volpe continued—There hasn't been much progress towards narrowing the differential diagnosis. TB seems less likely in light of the infectious disease consultation, and the borderline quantiferon TB-Gold results are likely another red herring. Though elevated ICP seems less likely given the low opening pressure, this was his second LP in less than two weeks and was performed while he was on high dose steroids. Both the LP and the steroids might lower ICP, so the results could be an additional red herring in this challenging case. The renal cyst needs to be investigated as a possible malignancy causing a

paraneoplastic syndrome, although this would be a highly atypical presentation and disc appearance for a metastatic/infiltrative or paraneoplastic optic neuropathy. MRI orbits with contrast and magnetic resonance venography would be extremely valuable, and it would be worth investigating to see if this could be accomplished in the setting of his renal disease.

It is reassuring that his vision stabilized over the short term with IV steroid therapy, though it is not known if this is a treatment effect or coincident with his lower ICP. While it is expected that steroids would stabilize and/or cause regression of inflammatory etiologies, high dose IV steroids have also been used for severe vision loss in the setting of elevated ICP.²⁰

Case report continued—On follow up one week later, visual acuity had deteriorated to 20/80 OD and no light perception (NLP) OS. He was readmitted for further evaluation and empiric steroid therapy. After the first dose of IV steroids, his left eye's vision improved from NLP to 20/800. An LP showed opening pressure of 52 cm H₂O. CSF mTB PCR was negative, as was cytology. MRI of the abdomen without contrast did not show malignant features in the renal cyst. He was started on acetazolamide 500mg twice daily, but developed acidosis, which prompted reduction in dose.

What now?

Comments by Dr. Nicholas J. Volpe continued—With confirmation of elevated ICP we can conclude that the optic nerve pathology is papilledema. The use of steroids to treat elevated ICP is controversial, but they are thought to increase cerebrospinal fluid absorption at the level of the arachnoid granulations.¹² While a number of case series demonstrate successful visual outcome in patients with elevated ICP treated with steroids,^{14,19,22} there is no randomized, controlled trial to provide specific treatment guidelines. Steroids are generally not considered first-line therapy for elevated ICP because of their side effects and concerns for rebound ICP elevation when they are discontinued. They have been recommended when severe vision loss is present and when awaiting surgical intervention.^{19,22}

Further investigations are needed to determine the cause of the elevated ICP since he is in an atypical demographic for idiopathic intracranial hypertension (IIH). In Bruce et al's series of 407 patients with IIH, only 5% were 50 years or older at presentation, and these were less likely to present with headache, but more likely to have visual disturbances than their younger counterparts.⁴ Additional evaluation is warranted for venous sinus thrombosis, dural arteriovenous fistula, and lower spine disease (e.g. epidermoid tumor, vascular malformation), all of which can cause elevated ICP with a normal brain MRI.

Case report concluded—In consultation with the neuro-radiology and renal services, arrangements were made for imaging with contrast. For stage IV renal disease, the risk of nephrogenic systemic fibrosis was felt to be small and less than the risk from catheter placement and dialysis.^{5,6} Macrocytic gadolinium at the lowest possible dose was administered.

MR angiography and venography of the brain showed a high-grade stenosis of the right transverse sinus and poor flow in the left transverse sinus. There was no optic nerve enhancement. Catheter angiography demonstrated severe transverse venous sinus stenosis (Figure 3a) in association with a type 1 dural arteriovenous fistula involving the external carotid arteries bilaterally and the superior sagittal sinus. Catheter manometry revealed pressure drop from 55mm Hg to 15mm Hg across the stenosis. Phase contrast quantitative MR venography (MRV) showed a total venous outflow of 438 ml/min and a total arterial intracranial flow of 566 ml/min.

He underwent successful balloon angioplasty and stenting of the right transverse venous sinus stenosis (Figure 3) with normalization of the pressure gradient to 16 mm Hg. Venous flow improved from 114 mL/min to 727 mL/min in the right transverse sinus. Aspirin 325mg and clopidogrel 75mg were added to his regimen. His vision was stable, though he developed pulsatile tinnitus. He was maintained on acetazolamide and prednisone 60mg daily until staged catheter-based embolization of the AV fistula could be done. Vision in both eyes improved, and the papilledema resolved. At most recent follow up visual acuity was 20/60 right eye and counting fingers at 1 foot temporally in the left eye. Visual fields showed normal V4e in the right eye with stable blind spot, resolution of temporal I4e field loss, but new inferotemporal loss to I4e. In the left eye, the temporal island had expanded nasally (Figure 4). Some of the persistent vision deficit in the right eye was accounted for by cataract.

Comments by Ali Alaraj, MD—This case demonstrates the difficulty in the diagnosis of severe venous stenosis and dural arteriovenous fistula presenting with chronic increase in ICP. A role of venous stenosis/occlusion in the pathophysiology of IIH has long been postulated.²⁴ Venous stenosis, similar to venous thrombosis, may be a primary cause for elevated ICP by impeding venous drainage, elevating cerebral venous pressure, and decreasing CSF reabsorption. Venous stenosis can also be secondary to dural sinus compression from increased ICP. In this latter situation it contributes to a positive feedback loop to exacerbate elevated ICP. Venography performed immediately after ICP lowering by CSF drainage to assess for reversibility of the stenosis allows a distinction between primary and secondary stenoses. In this case the stenosis was diagnosed following a large volume LP, suggesting that it is a primary pathology.

Although non-invasive MRV imaging is the first line in establishing the diagnosis of venous stenosis/occlusion with a sensitivity and specificity of 93%,¹⁰ it is often difficult to determine if such a stenosis is hemodynamically significant. The role of direct retrograde venography and manometry is critical to differentiate hypoplastic venous sinuses and arachnoid granulation from hemodynamically significant stenosis.¹⁶ In this case manometry recorded a trans-stenotic gradient of 40mm Hg. The role of phase contrast quantitative MRV in non-invasive evaluation of venous stenosis has not been determined, but the results in this case hint at the effects of venous sinus stenosis on cerebral hemodynamics. In this case venous outflow was less than intracranial arterial flow, which suggests that blood was exiting the brain through different (non-measured) venous routes like the ophthalmic veins, pterygoid plexus, and trans-osseous routes. Following treatment, the difference between

arterial and venous flow decreased, and the total blood flow increased, suggesting not only a change in the drainage pattern but also in total blood flow.

The added arterial inflow from the external carotid artery through the fistula likely further exacerbated the venous hypertension in our patient. This agrees with prior reports of dural arteriovenous fistulas elevating ICP in patients with dural venous sinus stenosis.^{7,23} One postulated mechanism for elevated ICP is that increased dural sinus pressure from the fistula results in secondary decreased CSF absorption.^{17,18} A dural fistula supplied by the external carotid arteries is a common association of chronic venous sinus occlusion.²⁵ Ahmed et al postulated that reducing pressure by embolizing the fistula leads to reduction in the venous sinus stenosis.¹ This invites consideration of a possible causal relationship whereby altered hemodynamics from a stenosis lead to fistula formation or vice versa.

Decreasing the pressure difference across a stenosis may result in immediate drop in the ICP¹¹ and improvement clinically. There is also evidence for a clinical benefit from transverse sinus stenting.^{9,13} Though there has been controversy surrounding venous sinus stenting because of concerns about safety, we felt the risk benefit ratio of endovascular intervention to be favorable because of our patient's inability to tolerate medical therapy, the elevated trans-stenotic pressure gradient, and presence of stenosis following LP that suggested a primary etiology. These are consistent with criteria associated with success of endovascular angioplasty and stenting through normalization of the pressure gradient.² The stenting was performed prior to fistula embolization in order to relieve venous hypertension, a possible causative factor in fistula formation. Improvement in vision and papilledema during this interval suggests relief of the venous hypertension.

Discussion

Papilledema from elevated ICP is an important consideration in bilateral optic disc swelling even in the absence of headache. IIH is a diagnosis of exclusion in patients outside the typical demographic for this condition, i.e. young, overweight women, and evaluation for occult vascular, neoplastic and infectious etiologies of elevated intracranial pressure is necessary in order to appropriately target therapy.

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Figure 1. Color fundus photos of right and left optic nerves at time of initial neuroophthalmic examination. Right eye shows marked disk elevation with shunt vessels (arrows). Left eye shows diffuse disc elevation.

Figure 2.
Kinetic perimetry of left and right eyes at time of initial neuro-ophthalmic examination.

Figure 3.
Digital subtraction angiography with retrograde venous sinus contrast injection. A. left transverse venous sinus stenosis (arrow). B. Balloon dilation of left transverse venous sinus. C. Post-stenting showing no residual stenosis in the left transverse venous sinus.

Figure 4.
Kinetic perimetry of left and right eyes 1 month following angiographic treatment