CASE REPORT

Isolated Fourth Nerve Palsy in Tuberculous Meningitis

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ABSTRACT

Tuberculous meningitis is a type of subacute meningitis and like other intracranial processes can compromise ocular motor nerves, causing palsies. Trochlear nerve is an unusual isolated manifestation in this type of pathology. The authors report a 5-year-old boy presented in their clinic with a trochlear nerve palsy as unique neurological manifestation of tuberculous meningitis. Treatment with complete anti-tuberculous therapy and botulinum A toxin was needed to get the complete resolution of the nerve palsy. In tuberculous meningitis, the presence of high protein levels and higher number of cells in cerebrospinal fluid is associated with cranial nerve involvement. Usually cranial nerve palsies occur in combination. VI cranial nerve alone or a combined disorder of VI and III cranial nerves are the palsies most frequently presented. Much less common is an isolated disorder of IV cranial nerve in the course of meningitis. The length of full treatment with anti-tuberculous therapy in this case was of 1 year. The persistence of palsy 1 month after the beginning of the symptoms indicated the possibility of a botulinum toxin injection with complete resolution of the process without disability, ocular motility has remained normal on follow-up.

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Tuberculous meningitis is a type of subacute meningitis and like other intracranial processes can compromise ocular motor nerves, causing palsies due to inflammation from meningeal infiltration. Trochlear nerve palsy is an unusual isolated manifestation in this type of pathology, despite being a common cause of vertical diplopia.

Altıparmak and colleagues⁹ reported a case of an 18-year-old male patient with a IV nerve palsy as the only neurological manifestation of a tuberculous meningitis. We report a case of another isolated trochlear nerve palsy due to tuberculous meningitis that presented a favourable evolution without disability at the end of the treatment.

Case report

A 5-year-old male patient with no previous medical history was hospitalised into the paediatric department with intermittent high temperature, nausea, vomiting, and neck stiffness during the last 15 days. He lives in a rural area and has contact with different kinds of animals.

Laboratory tests in the emergency department exploration showed a leukocytosis with a level of C-reactive protein (CRP) of 8 mg/L (reference range: 0–10 mg/L), low level of sodium (Na⁺) (128 mmol/L), and respiratory alkalosis (pH = 7.52, PaCO₂ = 29 mm Hg, bicarbonate = 25.9 mEq/L). Thorax x-ray was unremarkable. Serological testing was negative. A lumbar puncture was done. In the cerebrospinal fluid (CSF) cells were elevated (leukocytes = 112 cells/μL, with 95% mononuclear cells), with elevated proteins as well (128 mg/dL) and decreased glucose (7 mg/dL). The level of plasma glucose compared was 126 mg/dL. The adenosine deaminase (ADA) test on CSF had a value on the limit of normality (5 U/L). The Ziehl-Neelsen stain used to identify acid-fast organisms, mainly mycobacteria, was negative, and the result of CSF for *Mycobacterium tuberculosis* culture was inconclusive as well. Cultures for bacteria and viruses on CFS were negative too. The skin test with tuberculin purified protein derivative (PPD) showed a positive reaction and interferon-γ was found on blood tests with levels of

Figure 1. MRI with intravenous contrast revealing an enhancement in basal cisterns in supra- and infratentorial compartments, especially on the right side.

0.65 U/mL. Cranial magnetic resonance imaging (MRI) with intravenous contrast revealed compatible findings with tuberculous meningitis. Enhancement was present in basal cisterns in supra- and infratentorial compartments, especially on the right side (Figure 1), with a perimesence-

phalic extension to the occipital protuberance and tentorium. Imaging also revealed two basal ganglia ischaemic lesions on the right (Figure 2). Focal enhancement was seen near the exit of the V cranial nerve and along the course of the III and IV cranial nerves (Figure 3).

Based on clinical signs and symptoms, laboratory tests, and the findings on the MRI, a diagnosis of possible tuberculous meningitis was made and anti-tuberculous therapy with rifampicin, isoniazid, pyrazinamide, and ethambutol was started. This treatment lasted 2 months, and isoniazid and rifampicin were held for 10 months more. The patient was also given methylprednisolone and cefotaxime intravenously in the first month.

During the hospitalisation, three weeks from the onset of symptoms, the patient was referred to the ophthalmology department with a chief complaint of torticollis head-tilt left. On examination, he presented esotropia and hypertropia (20 Dp) of the right eye (Figure 4). Park's three-step test or Bielschowsky head tilt test, used to evaluate a

Figure 2. MRI with intravenous contrast showing two probable ischaemic lesions. Diffusion weighted imaging showing two high intensity lesions likely to be ischaemic.

Figure 3. MRI with intravenous contrast showing a focal enhancement through the course of III cranial nerve (A), on the exit of the V cranial nerve (C), and revealing the affection of IV cranial nerve on its path around the brainstem (B).

Figure 4. Esotropia and hypertropia (20 Dp) of the right eye.

paretic extraocular muscle, particularly superior oblique muscle and trochlear nerve (IV cranial nerve) in acquired vertical double vision, was explored. Calculating the vertical deviation in primary gaze, left and right gaze, and right and left head tilt, the three-step test is able to eliminate each muscle's primary field of action, showing in our case a right superior oblique muscle under action, suggesting right trochlear nerve palsy. The patient did not express diplopia, but he noticed blurred vision and complained of torticollis; his best visual acuity was 20/20 in both eyes. In head tilt position, there was no suppression and stereopsis was conserved.

Treatment with alternate eye patch and eye exercises was advised. Due to the persistence of a disabling torticollis, 5 U of botulinum toxin A into

the inferior oblique muscle of his right eye and 5 U into the inferior rectus of his left eye were injected.

After 2 months, the torticollis showed a complete resolution and ocular movements were normal (Figure 5).

Discussion

Due to the long path the IV cranial nerve has through the cranium, trochlear nerve palsy is commonly seen after head trauma, compressive lesions, or microvascular ischaemia. Tuberculous meningitis is one of the most common causes of chronic or subacute meningitis. Many cases of tuberculous meningitis have complications such as focal weakness, hemiplegia, and from 20% to 50% of cases show consequences related to ocular motor nerve palsies,^{1,2} III and VI nerves being the most frequently affected depending on the series.³⁻⁵ Paresis of this last nerve has important prognostic implications, with a mortality of 50% if there is involvement of the same, and 60% if there is a complete ophthalmoplegia.^{4,5} Sharma et al. noted that the characteristic pathological feature of tuberculous meningitis is the presence of thick gelatinous exudates that are prominent in the basilar regions of the brain.⁵

Figure 5. Normal ocular movements 2 months after botulinum toxin A injection.

In tuberculous meningitis, the presence of high protein levels and an elevated number of cells in cerebrospinal fluid was more associated with cranial nerve involvement and subsequently with the severity of the disease.⁴⁻⁷

The VI cranial nerve can frequently be affected in isolation, possibly because of its long subarachnoid course, but usually cranial nerve palsies occur in combination. Involvement of the III nerve can occur due to basal meningitis, the long course of this nerve in the base of brain, vasculitis in the arteries supplying the midbrain region, a tuberculoma situated in the midbrain region, or because of an increase of intracranial pressure.⁷

Rama et al. presented 90 cases of ophthalmoplegia, narrating a solitary patient with an isolated IV nerve palsy in a case of brain stem tumour. Combined nerve lesions of III, IV, and VI nerves or III or VI cranial nerve alone palsies were more common. Neurotuberculosis was the aetiology of 19 cases of them, and it was the VI cranial nerve alone or a combined affection of VI and III cranial nerves the palsies most frequently presented.⁸ Much less common is an isolated affection of IV cranial nerve in the course of meningitis. In the present case the infratentorial enhancement seen on MRI with perimesencephalic extension and enhancement at the exit of V and along the path of III is compatible with direct involvement of the trochlear nerve.

The incidence of tuberculosis has been low in our area. However, in the last few years the number of cases is increasing, mainly due to immigration. The presented boy had spent his first childhood in another country with a relative affected by tuberculosis. He did not show impairment of consciousness as in the case reported by Altiparmak et al.⁹ during follow-up. Treatment with anti-tuberculous therapy was continued for one year. Within the first week of the diagnosis and treatment of meningitis and when a clinical recovery was obtained, the child showed a very evident head tilt torticollis with a high hypertropia. A month later, due to the persistence of the hypertropia and because it was a child with a risk of amblyopia, we decided on a treatment with botulinum toxin A injection. In the case presented by Altiparmak et al., the palsy resolved in 2 months without ocular-specific treatment. In ocular motor

disorders, follow-up assessment can be used to decide if treatment with toxin botulinum is needed. Acquired IV cranial nerve palsy is an uncommon clinical entity in childhood, and few cases are related to the process presented in the case. The persistence of palsy and consequent symptoms led to treatment with a botulinum toxin injection, resulting in complete resolution of the process without disabilities. Ocular motility has remained normal on follow-up.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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