38. BILATERAL INTERNUCLEAR OPHTHALMOPLEGIA AND THIRD NERVE PALSY IN GIANT CELL ARTERITIS

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Introduction: We present a case of a gentleman with atypical headache symptoms clinically diagnosed as giant cell arteritis (GCA) and initiated on high-dose oral steroids. He subsequently developed progressive neurological deficit including bilateral internuclear ophthalmoplegia (INO), as well as third cranial nerve involvement despite above treatment. He received IV methylprednisolone and demonstrated clinical response, temporal artery biopsy confirmed histological evidence of GCA. The nature of his presentation was atypical of cranial giant cell arteritis. Few reported cases describe INO in the context of GCA, with bilateral manifestation being rarer, especially with additional third cranial nerve involvement.

Case description: A 66-year-old gentleman presented with a 7-day history of bilateral temporal headaches. He noted prominence of both temporal arteries with mild tenderness during this period. He denied any visual changes, PMR symptoms, and jaw claudication or weight loss. He had been experiencing generalised fatigue and myalgia for the preceding 4 months. He was noted to have raised inflammatory markers (CRP 194, ESR 74) and due to the non-specific nature of headache, concern was for possible meningitis. CT head scan was unremarkable and lumbar puncture and CT-CAP did not demonstrate any abnormality including evidence of infection.

He was assessed by the rheumatology team and a clinical diagnosis of GCA was made. He was initiated on prednisolone 60mg daily, and...
A 73-year-old patient with a background history of hypertension and mild asthma presented with a three-week history of ocular pain, headache and photosensitivity after a fall. CT head and lumbar puncture revealed bilateral parafalcine frontal lobe changes which were felt to be suggestive of active cerebral vasculitis. Cerebral vasculitis can be suspected on brain imaging if the logical deficit cannot be explained easily by territorial distribution of the supplying vessel. MRI imaging did not identify any focal pathology and as mentioned previously, following treatment, his clinical findings fully resolved in a gradual manner over a period of weeks.

In regards to the management of his case, following exclusion of infection and in the absence of visual findings on presentation, he was started on prednisolone 60mg daily (40-60mg dose suggested within current BSR guidelines). He described improvement of his headache and CRP was seen to improve initially. Despite this treatment, he developed features of INO and third nerve palsy as described. Implementation of IV methylprednisolone therapy prevented further progression of his symptoms and subsequent resolution of raised CRP. However, restarting prednisolone at 60mg after this appeared to demonstrate incomplete control of his condition, thus it was increased to 80mg. Decision to introduce methotrexate at an early stage was made in anticipation of likely difficulties in weaning prednisolone.

He remains under close follow-up to monitor prednisolone weaning. He currently has not had any recurrence of his symptoms.

Key learning points: GCA can present in an atypical manner and should remain a differential in cases of unexplained headache with associated inflammation. A combination of INO and third nerve palsy is an atypical manifestation of this condition. Due to the nature of his presentation, our gentleman was seen by various specialties including the acute-medical team, rheumatologists and neurologists. Fortunately, a probable diagnosis of GCA was made early and appropriate treatment was initiated. However, as this case demonstrates, response to treatment can vary and such adjustments were made to accommodate for this, potentially preventing long term disability.

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