

Bony skull neoplasms masquerading as giant cell arteritis

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CASE REPORT 1

A 59-year-old female smoker, with seropositive rheumatoid arthritis of 8 years' duration controlled on sulphasalazine, presented feeling unwell for 2 months with headache, giddiness and weight loss. Systemic examination was normal. She was tender over the scalp, especially over the temples. Her erythrocyte sedimentation rate (ESR) was 64 mm. A clinical diagnosis of giant cell arteritis (GCA) was made, and she was started on prednisolone 60 mg. Temporal artery biopsy was normal.

Further investigations revealed raised γ glutamyl transpeptidase (GGT) at 201 iu/litre (normal 7–33 iu/litre) and alkaline phosphatase at 186 iu (normal 30–120 iu). A chest X-ray showed a mass lesion in the left hemithorax, which, on biopsy, was found to be a small cell carcinoma. A computed tomography scan of her skull and a radioisotope bone scan were both consistent with widespread skull metastases.

CASE REPORT 2

A 60-year-old man presented with a 1-year history of aching discomfort of limb girdle muscles, visual disturbances in the form of flashing lights and headache with right-sided scalp tenderness. He also had urgency of micturition and nocturia. The ESR was 94 mm, and his GP diagnosed GCA and started the patient on prednisolone 40 mg/day. A dramatic symptomatic improvement was noted, and the ESR fell to 32 mm.

At the rheumatology clinic, the patient was found to have an enlarged prostate, elevated alkaline phosphatase at 1151 iu/litre and prostate specific antigen at 354 ug/litre (normal 0–4 ug/litre). A prostatic biopsy revealed a carcinoma, and a bone scan suggested multiple bony metastases in the skull, ribs, spine and right femur.

CASE REPORT 3

An 80-year-old woman was referred to the rheumatology clinic for pain in the left shoulder of 8 years' duration. Polymyalgia rheumatica had been diagnosed 8 years previously by her GP, because of generalized aches and pains and a raised ESR. She was taking prednisolone 15mg/day. Her left shoulder X-rays showed gross degeneration. When the prednisolone dose was reduced to 7.5 mg/day, she developed temporal pain and blurred vision. Pulsation of the temporal artery was just present on the left and absent on the right. ESR was 23 mm. GCA was diagnosed, and the prednisolone was increased to 15 mg/day, with initial improvement, but her symptoms fluctuated, and the ESR was consistently above 80 mm. Ten months later, she became generally unwell, and a chest X-ray revealed shadows suspicious of metastases. The ESR was 81 mm, the liver function tests were grossly deranged, carcinoembryonic antigen was 160 μ g/litre (normal <5 μ g/litre) and CA125 was 179 U/ml (normal <24 U/ml). A bone scan showed multiple skeletal deposits including skull metastases.

CASE REPORT 4

A 61-year-old man was seen in the medical assessment unit with a 4-week history of left temporal headache and scalp tenderness. His ESR was 120 mm, GGT was 90 iu/litre and alkaline phosphatase was 252 iu. GCA was diagnosed, and he was started on prednisolone 60 mg/day.

Symptoms improved initially, and the ESR fell to 35 mm, but these both relapsed as the steroids were reduced. After 6 months, a chest X-ray showed sclerotic bones suggestive of metastases. A bone scan showed extensive increased uptake throughout the axial skeleton, including skull, ribs, spine and pelvis. The prostate specific antigen was >1400 μ g/litre.

CASE REPORT 5

A 65-year-old woman presented to her GP with headaches and transient visual disturbances of the right eye for a few weeks. Three months later, the patient noticed sore lumps on her head, interpreted as thickened temporal arteries. Her ESR was 99 mm. The GP diagnosed GCA and started her on prednisolone 30 mg/day. The patient rapidly improved as did her headaches and head lumps. Two months later, when the prednisolone was reduced to 10 mg/day, the ESR went up to 89 mm. At rheumatology, a clinical diagnosis of GCA was thought likely, but blood tests revealed a paraproteinaemia of 7 g/litre (monoclonal immunoglobulin G Kappa), and a skeletal survey, including skull X-ray, did not reveal any lytic lesions. A bone marrow examination was consistent with multiple myeloma.

INTRODUCTION

Giant cell arteritis can be difficult to diagnose, and temporal artery biopsy is not always helpful. This paper reports five patients who were initially thought to have giant cell arteritis but proved to have another diagnosis. Four patients had bony skull metastases, while the fifth had multiple myeloma. These cases highlight the importance of carefully considering the differential diagnosis of giant cell arteritis before making a firm diagnosis.

CONCLUSION

These cases initially appeared to have giant cell arteritis, but each was shown to have an underlying neoplastic condition – in four cases bony skull metastases were present, while the fifth had multiple myeloma. Headache resulting from skull metastases, when associated with general malaise, weight loss and a raised erythrocyte sedimentation rate, can easily lead to a mistaken diagnosis of giant cell arteritis, and steroids may partially relieve the symptoms initially.

Skull metastases should always be considered when diagnosing giant cell arteritis, especially in the absence of a positive temporal artery biopsy. If there is a poor response to treatment or the symptoms are atypical, a lateral skull X-ray and/or bone scan should be considered.

We could find no references relevant to these cases in the literature. **HM**

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