Complete recovery of spinal cord compression following parathyroidectomy

ALKA GANESH*
M.D.

SUSY KURIAN†
M.D.

LILY JOHN
M.D.

Departments of *Medicine and †Pathology, Christian Medical College & Hospital, Vellore, S. India

Summary

A case is reported of spinal cord compression at T₂ level caused by a brown tumour of primary hyperparathyroidism involving the posterior arch of the vertebra. The spontaneous regression of cord compression following parathyroidectomy is documented clinically and radiologically.

Introduction

Hyperparathyroidism is suspected in patients who form recurrent stones, who have asymptomatic hypercalcaemia or, less commonly, who have single or multiple bone swellings. Hyperparathyroidism presenting as paraplegia due to involvement of the vertebral column by a bony swelling is rare but has been reported (Sui *et al.*, 1977; Shaw and Davies, 1968).

Case history

A 40-year-old man presented with progressive inability to walk for 2 months, and had been bed-ridden for 6 days before admission. He had severe pain radiating from the upper back down the inner part of the left arm corresponding to T₂ dermatome. Bony swellings appearing over the left shin and left clavicle over the last 2 years had been diagnosed elsewhere as osteoclastomas and had been treated by curettage and bone grafting.

Examination revealed grade 2 power in the lower limbs, and a sensory deficit for pain, temperature and touch, below T₄ cord level. The deep tendon reflexes in the lower limbs were exaggerated and the plantar responses were upgoing.

Investigations

Serum calcium concentrations on 3 occasions were elevated at 3.65 mmol/l, 3.50 mmol/l, 3.25 mmol/l; serum phosphorus 0.81 mmol/l; serum alkaline phosphatase 177 u./l; blood urea 3.65 mmol/l; serum creatinine 97.24 µmol/l. Chest X-ray (Fig. 1) showed expanding osteolytic lesions of the right glenoid cavity, lateral ends of both clavicles, right anterior end of third rib and left second rib. Plain X-ray of abdomen showed a right

renal calculus. Plain X-ray of the thoracic vertebrae (Fig. 2) showed a normal vertebral body of T₂. The pedicles were eroded and the end-on shadow of T₂ spinous process was missing. Lumbar myelogram showed a complete block to the flow of iophendylate injection at T₂ level.

FIG. 1. Chest X-ray showing bony lesion involving the glenoid cavity, and anterior end of the 3rd rib, and fracture of the right clavicle.

Course in hospital

A biopsy of the right clavicle produced irregular fragments of soft tissue and bone which were examined microscopically: there were increased numbers of osteoclastic giant cells along the margins of trabeculae of cancellous bone. Foci of haemorrhage and proliferating fibrous tissue containing large clusters of osteoclastic giant cells were also present. These features were considered to be

consistent with that of a 'brown tumour' of hyperparathyroidism. A diagnosis of primary hyperparathyroidism with spinal cord compression at T₂ level was made.

FIG. 2. Antero-posterior view of thoracic spine showing a normal T₂ vertebral body with poor delineation of pedicles and absent end-on spinous process (arrow).

A surgical exploration of the neck revealed an enlarged left inferior parathyroid, which was removed. The parathyroidectomy specimen consisted of a mass of soft pale brown tissue. Its cut surface showed it to be almost completely composed of a circumscribed pale brown nodule. Microscopic sections of this mass revealed compressed parathyroid tissue with a circumscribed tumour composed of sheets of polygonal cells with mildly pleomorphic dark staining nuclei and eosinophilic cytoplasm with many scattered thin walled vascular channels. There were focal areas of oxyphilic cell change. Secondary changes of hyalinization, haemorrhage, collections of inflammatory cells and haemosiderophages were seen at the periphery of the tumour. A diagnosis of parathyroid adenoma was made.

On the 5th postoperative day the patient remarked that his pain (T₂ root pain) had subsided completely. On examination it was found that sensory perception below T₄ level had reappeared. The para-paresis improved gradually from grade 2/5 power pre-operatively to grade 4/5 power on the 13th postoperative day, and he walked unaided on the 22nd day. Re-screening of the iophendylate injection on the 13th day showed contrast trickling past the T₂ site into the cervical region.

Discussion

This patient had unequivocal evidence of hyperparathyroidism presenting as multiple bony lesions and renal calculus, and repeatedly elevated serum calcium concentrations. In addition, he had clinical evidence of cord compression at T₂ vertebral level, substantiated by radiological demonstration of a complete block of the iophendylate column at this level.

The cause of the cord compression was not clear initially as the vertebral body of T₂ appeared normal. On careful scrutiny, however, it was seen that the normal end on shadow of the T₂ spinous process which should normally have been projected over the upper border of the T₃ vertebral body in the anteroposterior (AP) film was not present. Also, the pedicles of T₂ in the AP film were not seen, whereas those of T₁ and T₃ were sharply delineated. These features suggested that a lesion of the posterior arch of T₂ could be impinging on the spinal cord and compressing it from behind.

Since the patient's neurological deficit remained static throughout the period of investigation, it was decided to explore the neck before relieving the cord compression. After removal of the parathyroid adenoma, there was an unexpected dramatic relief of the root pain, and regression of the cord compression as shown by subjective and objective clinical improvement, and this was documented radiologically. This was evidence of rapid shrinkage of the bony lesion after parathyroidectomy. Brown tumours are known to regress after parathyroidectomy (Williams, 1974), whereas bone cysts, which appear identical to the former radiographically, do not. This is because bone cysts are fluid- or blood-filled cavities lined by fibrous tissue which do not possess osteoblastic activity.

A review of the literature showed 2 cases of brown tumours causing acute cord compression (Sui *et al.*, 1977; Shaw and Davies, 1968). Both cases had involvement of T₁₀ vertebral bodies, and the diagnosis was made at laminectomy. To the present authors' knowledge this is the first time that a brown tumour involving the posterior arch rather than the 'body' of a vertebra has been described. There have not been previous reports of complete regression of cord compression following parathyroidectomy.

References

- SHAW, M. & DAVIES, M. (1968) Primary hyperparathyroidism presenting as spinal cord compression. *British Medical Journal*, **4**, 230.
- SUI, K., SUNDARAM, M., SHULTZ, C. & KIRWAN, L. (1977) Primary hyperparathyroidism presenting as spinal cord compression. Report of a case. *Australian and New Zealand Journal of Surgery*, **47**, 668.
- WILLIAMS, R. (1974) *Text Book of Endocrinology* 5th Edn (Asian Edition), p. 737. W. B. Saunders Company, Philadelphia, London, Toronto; Igaku Shoin Ltd, Tokyo.