Heterotopic bone formation following cerebral cysticercosis

A case report

R. F. SPENCER, V. GANPATH

Summary

A patient with severe spasticity after cerebral cysticercosis formed heterotopic new bone at several sites. It is suggested that this case provides some clues to the cause of this phenomenon in patients with neural injury.

Heterotopic bone formation may occur after a variety of disorders including spinal cord injury, poliomyelitis, chronic infection, and burns. Numerous reports exist of atypical bone formation around fractures in patients with head injuries. However, cerebral parasitic infestation has not been recorded in this context.

The cause of new bone formation in patients with neural injury is uncertain. Local as well as general factors may play a part. Recent evidence suggests that new bone formed around healing fractures in such patients may resemble 'heterotopic bone' more closely than 'fracture callus'.

Locally released morphogens in damaged muscle may play a part in allowing bone to form at unusual sites. In the case presented here, a nidus may have formed around muscle cysticerci initiating the process of new bone formation.

Case report

A 33-year-old man was admitted to hospital in a comatose condition after a short febrile illness. Upper motor neuron signs were demonstrated in all four limbs. Cerebral computed tomography (CT) showed an area of decreased density in the pontine region, and a calcific focus in the proximity of the pineal gland (Fig. 1). Routine serological tests for cysticercosis were positive. A course of treatment appropriate to the cerebral condition was started.

After recovery of mental capacity the patient was discharged from hospital. He was ambulant, but found difficulty performing basic functions necessary for independence owing to severe spasticity and joint contractures.

A programme of physiotherapy produced little improvement, and further cerebral CT was performed 2 years after initial presentation. This demonstrated further lesions, including a focus of calcification behind the posterior clinoid process (Fig. 2).

However, during the next year the patient's condition improved to the extent that he was again able to drive a car to work, but was forced to accept less skilled employment than his previous clerical position. He remained disabled, with fixed deformities of the shoulders, elbows, hips and knees.

Upper motor neuron signs persisted in all four limbs. His deformities at the time of review were most incapacitating in the left hip (fixed flexion deformity of 40° and fixed abduction deformity of 30°), left knee (fixed flexion deformity of 20°) and left shoulder (fixed abduction, flexion and internal rotation). The left hip and shoulder were ankylosed.

Radiography 6 years after the onset of the cerebral illness showed extensive peri-articular heterotopic bone formation around the left shoulder and left hip, and new bone formation posterior to the left knee (Fig. 3). It is not certain at what stage the new bone was first formed.

Discussion

Infection with *Taenia solium* is endemic in parts of South Africa, and cysticercosis has been implicated as a cause in a
significant number of cases of epilepsy in Durban. However, patients with the disease are rarely seen in an orthopaedic department unless muscle cysticerci are an incidental finding in radiography for other purposes.

Intracranial parasitic infestation may present as an acute encephalopathy, as in the patient described here. The residual neurological deficit is seldom thought to be an indication for orthopaedic assessment. However, full recovery of mental function must be anticipated, even if this seems unlikely.

In a review of head-injured patients who developed heterotopic ossification Garland et al. noted a correlation with spasticity. Ankylosis was found to be rare, and occurred in only 16% of joints. The abduction deformities found in our patient were not seen. Mendelson et al. reported an incidence of 20% of heterotopic ossification in head-injured patients. It is postulated that some sort of soft-tissue injury is an important initiator of new bone formation, possibly via locally released morphogens, which allow mesenchymal cells to form bone at extra-osseous sites. This process may be adjacent to a fracture, or a haematoma or (as in our case) an intramuscular parasite.

Unfortunately, in the case reported, no attempt at appropriate early treatment was made. A combined approach involving splintage, physiotherapy, nerve blocks, intramuscular alcohol injections and the use of pharmacological agents combined with well-timed surgical release procedures may have been of benefit. Once peri-articular bone has formed, surgical removal is hazardous and the results unpredictable. It should be postponed until 2 years after the onset of the process. Our patient refused such surgery and remains severely disabled.

We suggest that in patients with neural injury, particularly those with spasticity, a thorough examination for areas of soft-tissue damage elsewhere in the body should be performed so that pre-emptive measures may be initiated should heterotopic bone formation supervene.

REFERENCES