BILATERAL AVASCULAR NECROSIS OF THE CAPITATE
A CASE REPORT AND A REVIEW OF THE LITERATURE

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A case of bilateral avascular necrosis of the capitate is presented. A review of the literature has identified a clear-cut clinical syndrome. The aetiology and pathology of this syndrome is discussed and a new method of treatment is proposed.

Avascular necrosis of the capitate is a rare condition. It is well recognised as a sequel to severe trauma, as in the naviculocapitate syndrome. There have been only four cases reported in the world literature in which severe trauma has not played a major aetiological role (Jönsson 1942; Newman and Watt 1980; Lowry and Cord 1981; Kimmel and O'Brien 1982). We present a case of bilateral avascular necrosis of the capitate with no history of trauma and discuss the possible aetiology.

On examination, neither wrist was swollen, but both were tender over the hamate/capitate area. Both wrists had full pronation, supination, and radial and ulnar deviation. Dorsiflexion was 80° on the left and 75° on the right. Palmarflexion was 80° on the left and 70° on the right. There was no crepitus in either wrist. Radiographs showed avascular necrosis in both capitate bones; there were degenerative changes in the carpal joints and cysts in the other carpal bones (Figs 1 and 2).

CASE REPORT

A right-handed bus conductor aged 18 was seen at another hospital, complaining of pain and stiffness in his right wrist. There was no history of trauma to the wrist. His hobby was weight-lifting. Examination revealed a wrist that had minimal diminution of movement in all directions. Radiographs showed avascular necrosis of the capitate. As he was still able to work no treatment was prescribed.

He presented again at the age of 20 with increasing loss of power in his left wrist, associated with stiffness and tingling. Clinical examination and radiographs were similar to those of the other wrist. Treatment with a non-steroidal anti-inflammatory drug gave symptomatic relief.

Aged 25, he fell off his motorcycle and injured his right wrist. No fractures were identified radiographically, but he was placed in a plaster cast for a week. After physiotherapy, the grip in his right hand became worse and he was unable to use a screwdriver. Vibration caused an increase in his pain and this prevented him from pursuing his new hobby of motorcycle scrambling. It was at this stage that he was referred to our unit.

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Because of the severity of the symptoms in the right wrist, surgical treatment was recommended. This consisted of excision of the head of the capitate and replacement with a silicone prosthesis—an elastomer universal small joint spacer. Figures 3, 4 and 5 show the stages of the operation.

He was reviewed two years after operation. He had returned to work as a lorry mechanic, though with some discomfort in the wrist. He had, however, virtually a full range of movement in the wrist and the radiograph showed the prosthetic spacer still in position (Fig. 6).
established avascular necrosis. The head of the capitate has no collateral blood supply, being vascularised from the middle third of the bone. Any lesion that causes disruption of this supply is a potential cause of avascular necrosis. The naviculocapitate syndrome is a prime example of such an occurrence. Lowry and Cord (1981) suggest that a vascular anomaly may be responsible and that trauma not severe enough to cause a fracture is an exacerbating factor.

**Histology.** Histological examination showed that the excised capitate had normal hyaline cartilage at one end and degenerate cartilage at the other. There was a large area of established bone necrosis with appositional new bone formation around the necrotic trabeculae (Fig. 7). The appearances were those of revascularised healing bone necrosis.

**DISCUSSION**

We could find reports of only four cases of idiopathic avascular necrosis of the capitate in the literature. We add a fifth, the first to be reported with bilateral disease. All five patients have features in common: all presented in their late teens or early twenties; the dominant (right) hand was predominantly affected and the head of the capitate was the area most severely involved; in the bilateral case, the dominant hand was the more severe. The suggested aetiology of this condition differs in all the cases reported. Three major aetiological factors will be discussed: the blood supply, the mobility of the wrist, and trauma.

**Blood supply.** The blood supply of the capitate is well documented (Barber 1972). The vessels enter the bone where the ligaments are attached on the dorsal and volar surfaces. The head of the capitate has no collateral blood supply, being vascularised from the middle third of the bone. Any lesion that causes disruption of this supply is a potential cause of avascular necrosis. The naviculocapitate syndrome is a prime example of such an occurrence. Lowry and Cord (1981) suggest that a vascular anomaly may be responsible and that trauma not severe enough to cause a fracture is an exacerbating factor.

**Hypermobility or instability.** Both Newman and Watt (1980) and Kimmel and O'Brien (1982) demonstrated hypermobility or instability in both wrists of their patients. They felt that this was a significant factor in the causation of the disease. Logically, if the vessels enter with the ligaments and these are lax, then relatively minor trauma, which stretches or kinks the ligaments, might well cause damage to the vessels. Moreover, ligamentous injuries to the wrist causing instability are more common than is generally realised (Gilula and Weeks 1978).

**Trauma.** There is a clear history of trauma in two of the reported cases (Newman and Watt 1980; Lowry and Cord 1981), though in only one was it regarded as a major causative factor. Newman and Watt have suggested that the condition may be a variant of the naviculocapitate syndrome. They hypothesise that an undisplaced fracture of the capitate, very difficult to visualise on radiographs, is the underlying pathology. In combination with an unstable wrist joint, the conditions are favourable for avascular necrosis of the head of the capitate to occur. Repeated minor dorsiflexion injuries may cause stress fractures of the capitate and this also could be a causative factor.
In our case, minor trauma with repeated weight-lifting may have been a cause. We were unable to demonstrate any carpal instability as both wrists were affected and stiff by the time he presented to us. It would appear that this condition is very rare because a combination of factors is needed to produce it; these include an anomalous blood supply, an inbuilt carpal instability, and trauma at an appropriate age.

Our case is also of interest because only two of the previous patients have undergone any surgical treatment for their avascular necrosis. Procedures performed have included fusion of the intercarpal joints, and excision of the bone with a palmaris longus tendon graft inserted as a spacer. Both these procedures gave acceptable results.

Conclusion. Avascular necrosis of the capitate is rare, but should be considered in all young people who have had a dorsiflexion injury to the wrist which is not severe enough to cause a recognisable fracture, and in whom the symptoms do not settle rapidly. Initial treatment should be conservative and expectant. We suggest, however, that if the symptoms persist then a replacement arthroplasty should be considered.

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REFERENCES