Posterior shoulder dislocation and humeral head necrosis in a recreational scuba diver with diabetes

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Gorman DF, Sadow MJ. Posterior shoulder dislocation and humeral head necrosis in a recreational scuba diver with diabetes. Undersea Biomed Res 1992; 19(6):457–461.—An insulin-dependent diabetic who was previously a recreational scuba diver suffered a posterior shoulder dislocation after a trivial injury and was subsequently found to have local necrosis of the humeral head. The only definite conclusion that can be reached is that this patient should not dive again.

diabetes mellitus

dysbaric osteonecrosis

aseptic bone necrosis

osteonecrosis

posterior shoulder dislocation

Aseptic necrosis of bone is seen in caisson workers, diving fishermen, and other occupational divers (1), but only rarely in recreational divers. Some authors include diabetes mellitus in a list of differential diagnoses of aseptic necrosis of bone (1), but this is also rare and appears confined to the hip. Posterior dislocation of the shoulder is usually consequent to a convulsion, electrocution, or some other significant trauma (2).

CASE HISTORY

A 34-yr-old male clerical officer (patient ER) came to the Royal Adelaide Hospital 2 wk after an injury to his left shoulder, complaining of persistent pain and limited mobility. The injury occurred when he pushed against a door jamb with his left arm, at which time he felt a sudden pain over the posterior aspect of his shoulder. Plain x-rays of this shoulder demonstrated a change in humeral head texture and a possible posterior dislocation. Both were confirmed by a computed tomography (CT) scan (Fig. 1), which also showed a large anterior impaction defect.

The patient had insulin-dependent diabetes mellitus (IDDM) diagnosed 7 yr previously. This was being monitored serologically and was well controlled. Before this diagnosis, he was an active recreational scuba diver, having completed about 100
dives. These dives were all conducted within the no-decompression depth-time limits published by the United States Navy (USN) (3) and to a maximum depth of 30 msw. Repetitive dives also conformed to USN regulations (3). He was not aware of the presence of the IDDM at the time of his initial diving medical examination, and insisted that he had never experienced any diving-related problems, including decompression illness (DCI).

As more than 50% of the humeral head was “radiologically intact,” shoulder reconstruction was performed using a modified McLaughlin procedure (4). Biopsies of the humeral head taken during the operation subsequently demonstrated well-established aseptic osteonecrosis with appositional new bone formation (Fig. 2A, B). The age of the necrosis was assessed from these biopsies to exceed 2 mo. (i.e., to easily predate the dislocation).

Six months later, after 4 wk of abduction splinting and subsequent mobilization, the patient had regained most shoulder movement and showed no signs of bone necrosis at any other site (Fig. 3). Other possible causes of aseptic bone necrosis (steroid therapy, tuberculosis, sickle cell anemia, chronic alcoholism, liver cirrhosis, hepatitis, pancreatitis, Gaucher's disease, rheumatoid arthritis, gout, ionizing radiation, syphilis, alcaptonuria, arteriosclerosis, hyperlipidemia) (1, 2) were excluded. There was no history of any previous trauma to the left shoulder.

DISCUSSION

This patient is an interesting exercise in the differential diagnosis of aseptic bone necrosis. The competing explanations here are trauma, IDDM, and diving (i.e., DCI). The trivial trauma reported cannot explain the bone necrosis, especially given the
Fig. 2. Patient ER. Microphotographs of bone biopsies from the left humeral head showing a necrotic spicule of lamellar bone (center) with osteoclastic tunneling resorption and advanced appositional woven new bone formation (peripherally). Top, low power (×100); bottom, high power (×200).
assessed age of the necrosis. Indeed, in the absence of preexisting necrosis, it is
difficult to rationalize the extent of the trauma and the subsequent posterior shoulder
dislocation (2). The relationship between IDDM and bone necrosis is far from certain,
despite its occasional listing as causal (1); there are no published large series of
diabetics showing any such relationship, and especially not in the shoulder.

Although the shoulder is a frequent site of dysbaric osteonecrosis (1, 5–11), diving
by itself is not a good explanation for this patient. In the absence of overt DCI
(1, 9–11), dysbaric osteonecrosis is uncommon in either recreational divers or in
commercial divers who have not exceeded 30 msw. Fishermen divers who dive to
less than 30 msw do develop dysbaric osteonecrosis (1, 5, 7, 8, 10), but these divers
frequently dive in excess of published no-decompression time limits and often develop
DCI. Similarly, caisson workers who have not been compressed beyond 4 bar atm
abs and still develop dysbaric osteonecrosis (1, 6, 10) differ from our patient in that
their work-shifts in the caisson usually vary between 4 and 8 h. Despite a staged or
slow decompression, some of these caisson workers also experience DCI. Indeed,
DCI is a major risk factor for bone necrosis, and in active North Sea commercial
divers who have been diving for less than 10 yr and have not had DCI, the risk of
dysbaric osteonecrosis approaches zero (9). In this context, our patient’s experience
of 100 dives to 30 msw or less, all within the USN no-decompression depth-time
limits, is negligible. These comments on prevalence are relevant to x-ray-apparent
bone disease, as in our case, and not to more sensitive (perhaps over-sensitive)
techniques such as bone scintigraphy, magnetic resonance imaging (MRI), and ultra-
sonography (1, 12–15).

It is possible that this bone necrosis and subsequent joint dislocation are due to
a combination of initially unrecognized IDDM and diving. The possible nature of
that combination cannot be elucidated much further given the paucity of data available
both for IDDM and bone necrosis and for IDDM and DCI, and given that the etiology and pathogenesis of dysbaric osteonecrosis is still controversial (1, 10). Nevertheless, IDDM theoretically could predispose to DCI (16) and hence presumably to dysbaric osteonecrosis (1, 10); dysbaric osteonecrosis can arise from even a single hyperbaric air exposure (17) and can be progressive, even in the absence of any further diving/ decompressions (18).

The only definite conclusion that can be reached is that this patient should not dive again, not the least for the problems intrinsic to IDDM (16). Despite current good progress, the patient must also remain under medical surveillance.

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REFERENCES