Case report

OSTEONECROSIS OF THE HUMERAL HEAD AFTER EXTRACORPOREAL SHOCK-WAVE LITHOTRIPSY

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A 59-year-old woman with calcific tendinitis in her right shoulder underwent extracorporeal shock-wave lithotripsy. Three years and four months later she presented with osteonecrosis of the head of the right humerus. It is known that shock waves in patients with urological disorders can damage blood vessels. A possible reason for the development of osteonecrosis in this patient may have been damage to the blood supply of the head of the humerus.

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Extracorporeal shock-wave lithotripsy has become a common treatment for calcific tendinitis of the shoulder. No severe complications have been reported, and it is considered to be a safe procedure. We present a case of osteonecrosis of the head of the humerus after extracorporeal shock-wave lithotripsy. Possible pathomechanisms are discussed.

Case report

In June 1996, a 59-year-old woman presented with chronic pain in her right shoulder. Radiographs showed a deposit of calcium of 28 × 10 mm in the tendon of supraspinatus (Fig. 1). Since three subacromial injections of cortisone had not given symptomatic relief she underwent three sessions of extracorporeal shock-wave lithotripsy with 1600 to 1700 impulses at each session at a level of 12 to 13 kV over a period of one month. After treatment there was less pain and a full range of movement. Radiographs revealed a reduction in the size of the deposit of calcium by 79% to 12 to 13 kV over a period of one month. After treatment there was less pain and a full range of movement. Radiographs revealed a reduction in the size of the deposit of calcium by 79% to 12 mm, without any evidence of osteonecrosis of the humeral head (Fig. 2). Three years and four months later she presented again with chronic pain in the shoulder. Radiographs showed partial necrosis of the humeral head which was stage IV according to the classification of Neer (Fig. 3). MRI confirmed the radiological diagnosis of partial necrosis. There were no signs of osteonecrosis in the contralateral shoulder. Investigations for known predisposing factors for osteonecrosis were negative.

Discussion

To our knowledge no severe complications of extracorporeal shock-wave lithotripsy have been published in the orthopaedic literature. Minor problems such as pain in the shoulder, local soft-tissue swelling, cutaneous erosions, erythema, petechial haemorrhage and local subcutaneous haematomas have been described. Apart from a single report of changes in the MRI signal in the subcortical area of the greater tuberosity 12 weeks after treatment, no bony or cartilaginous changes have been demonstrated by radiography, MRI, sonography or arthroscopy. The longest radiological follow-up after shock-wave treatment to the shoulder is, however, only two years.

Gerber, Schneeberger and Vinh identified the ascending branch of the anterior humeral circumflex artery and its continuation, the arcuate artery, as being the main supply to the proximal humeral epiphysis. It was further shown that the closer this artery is injured to its point of entry into the bone the greater is the risk to the vascularity of the humeral head, because of a lack of distal anastomoses. The vessel enters the bone in the area of the lateral and superior aspects of the intertubercular groove. In our patient this point was only 10 mm from the deposit of calcium.

It is known that shock waves in patients with urological disorders have caused damage to blood vessels ranging from benign lesions of the endothelium to arterial occlusion with capillary extravasation, and even ruptures of the vessel wall. Destruction of the ascending branch of the anterior humeral circumflex artery could thus be responsible for the osteonecrosis in our patient. Also, the time interval between injury and the radiological appearance of osteonecrosis is comparable to that which may occur with fractures of the proximal humerus. Darder et al showed that it may take up to four years for radiological signs of osteonecrosis to develop in patients with four-part fractures of the proximal humerus. In our patient MRI showed that the osteonecrosis occurred in the area of perfusion of the anterior circumflex artery. The bone in the area of perfusion of the posterior circumflex artery was not affected (Fig. 4).

A possible alternative aetiology, although unlikely, may be the repeated subacromial infiltrations of cortisone. We were unable to find any evidence in the literature of osteonecrosis after subacromial infiltration of steroid, although there are some case reports of osteonecrosis after intra-articular injection.

We found no other predisposing factors for osteonecrosis in our patient. The development of idiopathic osteonecrosis may have
been simply coincidence and the reason for the development of osteonecrosis of the humeral head in this patient remains uncertain. With the increasing popularity of this form of treatment, experimental and long-term clinical follow-up studies are necessary to establish whether avascular osteonecrosis of the humeral head is in fact a rare, but severe, complication of extracorporeal shock-wave treatment. If shock waves are found to be harmful to the blood supply of the humeral epiphysis, care must be taken to avoid the intertubercular groove when targeting the shock wave on deposits of calcium.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References


