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## Humeral Head Osteonecrosis After Extracorporeal Shock-Wave Treatment for Rotator Cuff Tendinopathy

### A Case Report

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## Introduction

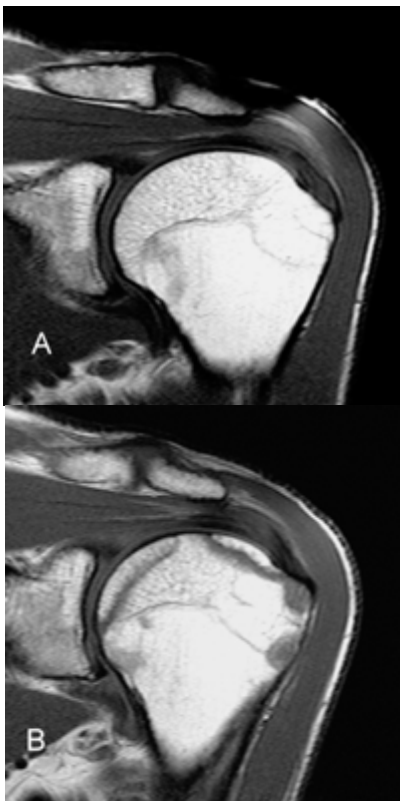
Extracorporeal shock-wave treatment has been used for calcific tendinitis of the shoulder for more than fifteen years<sup>1-3</sup>, and it is considered to be an effective and safe procedure<sup>4</sup>. Minor complications, such as pain, local soft-tissue swelling, cutaneous erosions, and erythema, have been reported in the literature<sup>4</sup>. We present a case of osteonecrosis of the humeral head that developed after high-dose extracorporeal shock-wave treatment. The patient was informed that data concerning the case would be submitted for publication.

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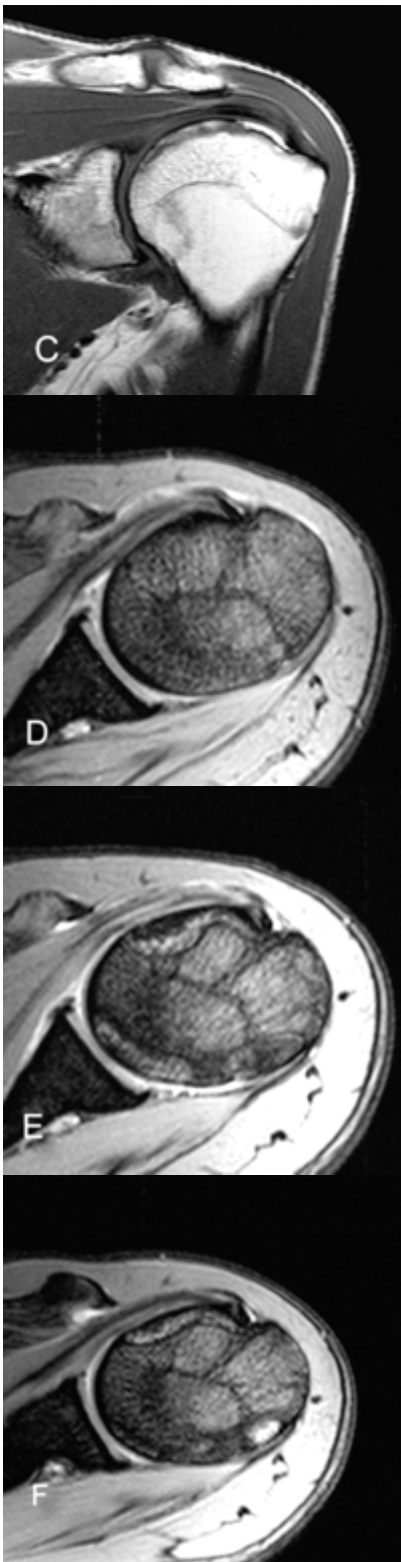
## ▶ Case Report

A forty-nine-year-old woman underwent serial magnetic resonance imaging examinations of the left shoulder over a period of ten months to evaluate pain, which had begun after she pulled a heavy object. The imaging was performed with a phased-array shoulder coil and a standard 1.5-T whole-body imager (Sonata; Siemens Medical Solutions, Erlangen, Germany). The protocol included coronal and axial T1-weighted imaging, coronal and sagittal turbo spin-echo T2-weighted imaging, coronal T1-weighted imaging with fat saturation, and axial T1-weighted imaging without fat saturation after intravenous injection of a gadolinium-based contrast agent (Omniscan; Amersham/GE Healthcare, Milwaukee, Wisconsin). The serial magnetic resonance examinations were performed before ([Figs. 1-A and 1-D](#)), three months after ([Figs. 1-B and 1-E](#)), and seven months after ([Figs. 1-C and 1-F](#)) extracorporeal shock-wave treatment. The first magnetic resonance examination revealed shoulder impingement with a partial tear of the supraspinatus tendon on the humeral side, subacromial and subdeltoid bursitis, and biceps tenosynovitis ([Figs. 1-A and 1-D](#)). There was no evidence of osteonecrosis of the humeral head. The patient then received one extracorporeal shock-wave treatment each week with a piezoelectric system (Piezoston 100; Richard Wolf, Knittlingen, Germany) for three consecutive weeks. The impulse rate was 3000 shocks per session with an energy of 0.78 mJ/mm<sup>2</sup>. The total energy given (2340 mJ/mm<sup>2</sup>) was within the range recommended by the manufacturer (300 to 5850 mJ/mm<sup>2</sup>).

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**Figs. 1-A, 1-B, 1-C, 1-D, 1-E, and 1-F** **Figs. 1-A through 1-F** A forty-nine-year-old woman had a painful left shoulder for ten months. Serial magnetic resonance examinations show the development of osteonecrosis after extracorporeal shock-wave treatment for the painful shoulder. Coronal T1-weighted images (Figs. 1-A, 1-B, and 1-C) and axial T2-weighted images with fat saturation (Figs. 1-D, 1-E, and 1-F) were obtained before (Figs. 1-A and 1-D), at three months after (Figs. 1-B and 1-E), and at seven months after (Figs. 1-C and 1-F) extracorporeal shock-wave treatment. Figures 1-A and 1-D reveal no definite abnormality in the humeral head. Figures 1-B and 1-E reveal a geographic lesion in the humeral head with abnormal signal intensity and abnormal contrast enhancement consistent with the diagnosis of osteonecrosis. Figures 1-C and 1-F reveal progression of the disease with a "crescent sign" in the humeral head.



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Three months after the extracorporeal shock-wave treatment, the follow-up magnetic resonance examination showed a newly developed area of osteonecrosis in the left humeral head ([Figs. 1-B and 1-E](#)) in addition to the previous findings. The magnetic resonance studies depicted geographic subchondral lesions of heterogeneous signal intensity on the T1-weighted image, high signal intensity on the T2-weighted image, and mild enhancement in the humeral head with preservation of its cortical outline. No specific treatment was given. Seven months after the extracorporeal shock-wave treatment, another follow-up magnetic resonance examination revealed a "crescent sign" in the humeral head ([Figs. 1-C and 1-F](#)). This sign was not present on the study performed at three months and was considered to demonstrate progression of the osteonecrosis.

The shoulder pain became intolerable, and the patient also noted progressive limitation of the range of motion of the shoulder. She therefore underwent surgical core decompression of the humeral head two months later. During this hospital stay, an investigation for known predisposing factors for osteonecrosis, including a coagulation profile and blood tests for autoimmune disease, revealed negative findings. Most importantly, the clinical history included no known predisposing factors such as injury, use of steroid medication, blood disorders such as sickle cell disease, excessive alcohol use, Gaucher disease, pancreatitis, radiation treatment, chemotherapy, decompression disease, connective tissue diseases, or dyslipoproteinemia.

## Discussion

Our review of the English-language literature revealed only one case, reported by Durst et al.<sup>5</sup>, of osteonecrosis of the humeral head after extracorporeal shock-wave treatment. In that patient, osteonecrosis was seen incidentally three years and four months after treatment, and the authors could not be sure when the disease process had started. In our patient, the osteonecrosis was diagnosed with magnetic resonance imaging three months after extracorporeal shock-wave treatment. While we cannot completely exclude the possibility of idiopathic osteonecrosis in our patient, the timing of the finding on magnetic resonance imaging in relation to the extracorporeal shock-wave treatment strongly supports our assumption that the shock-wave therapy was the cause.

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Extracorporeal shock-wave treatment is not yet a standard therapeutic technique in orthopaedics. The mechanism for the treatment effect on soft-tissue lesions is still unknown. Animal studies have indicated that stimulation of the expression of angiogenesis-related growth factor, which induces in-growth of neovascularization and increases cell proliferation, is a possible mechanism<sup>6</sup>. The effect of extracorporeal shock-wave treatment on tendinitis of the rotator cuff is controversial, despite some recently reported large series<sup>4,7-9</sup>. Compared with a placebo, high-energy extracorporeal shock-wave treatment was found to improve shoulder function and decrease the size of calcifications in the soft tissue. However, reduction in pain was not consistent in the previous studies. Extracorporeal shock-wave treatment is supposed to be a safe procedure, but adverse effects have occasionally been reported<sup>3</sup>.

The side effects of extracorporeal shock-wave treatment are dose-dependent. Chung and Wiley<sup>10</sup> characterized extracorporeal shock-wave treatment according to energy categories: low ( $<0.08 \text{ mJ/mm}^2$ ), medium ( $0.08$  to  $0.28 \text{ mJ/mm}^2$ ), and high ( $>0.6 \text{ mJ/mm}^2$ ). We are aware of only one report on the effect of high-energy extracorporeal shock-wave treatment on tendons<sup>11</sup>, and no standard protocol for extracorporeal shock-wave treatment has been established to date. An animal study revealed that a local energy density of  $0.3 \text{ mJ/mm}^2$  is the lower threshold for the occurrence of severe vascular damage<sup>12</sup>. Rompe et al.<sup>13</sup> recommended against high-energy extracorporeal shock-wave treatment ( $>0.6 \text{ mJ/mm}^2$ ) of tendon tissue after they investigated the effects of shock waves on the rabbit Achilles tendon. Peters et al.<sup>14</sup> reported hematoma formation in six patients after high-energy treatment and in two patients after low-energy treatment. In a report on 272 patients with tennis elbow, Haake et al.<sup>15</sup> found more side effects in the group treated with extracorporeal shock-wave treatment than in the placebo group. The most common side effects were transitory reddening of the skin, pain, and the development of small hematomas. Our patient was treated with a high energy level, but the dose remained within the range recommended by the manufacturer. We suggest that whenever high-energy extracorporeal shock-wave treatment is used for a shoulder disorder, the possibility of osteonecrosis developing should be considered.

Extracorporeal shock-wave treatment is currently considered the standard therapy for most renal and upper ureteral stones, and low, acceptable complication rates have been reported<sup>16,17</sup>. However, as the technique has become more widely available, serious complications as a result of injury to the kidney and the surrounding organs have been recognized. These complications have included direct injury to the organ itself and various vascular insults<sup>18-21</sup>.

These complications are thought to be the result of cell damage caused by cavitation produced by the shock

waves<sup>15</sup>, rupture of small blood vessels (with an inner diameter of <300 μm)<sup>22</sup>, complete detachment of endothelial cells in defined regions<sup>12</sup>, cell damage after ischemic-reperfusion injury, vasoconstriction, and free-radical production<sup>23</sup>. We theorized that the development of osteonecrosis of the humeral head after extracorporeal shock-wave treatment may share a common mechanism with the vascular damage seen after extracorporeal shock-wave treatment of renal stones. The arterial supply of the humeral head is known to derive mainly from the anterior humeral circumflex artery. According to a cadaver study by Duparc et al.<sup>24</sup>, the diameter of the anterior humeral circumflex artery ranges between 0.3 and 2 mm in adults. Some patients with a small artery may be more susceptible to the development of osteonecrosis after extracorporeal shock-wave treatment.

We documented a case of osteonecrosis of the humeral head that was detected three months after high-dose extracorporeal shock-wave treatment of rotator cuff tendinopathy. Extracorporeal shock-wave treatment is widely used in orthopaedics, and the possibility of such a severe complication should be considered. ■

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