

Bilateral Avascular Necrosis of the Trapezoid

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We present a 5-year follow-up of a patient with bilateral necrosis of the trapezoid that improved clinically and radiographically with nonoperative treatment. (*J Hand Surg* 2011;36A:1678–1680. Copyright © 2011 by the American Society for Surgery of the Hand. All rights reserved.)

Key words Avascular necrosis, trapezoid bone, wrist vascularity.

AVASCULAR NECROSIS OF a carpal characteristically presents with wrist pain, stiffness, and weak grip. The lunate is affected most commonly (Kienbock disease), followed by the scaphoid (Preiser disease), capitate, pisiform, and trapezium.^{1–5} We found only one report describing avascular necrosis involving the trapezoid in the English language literature.⁶ We describe a patient with bilateral avascular necrosis of the trapezoid that responded to nonoperative measures.

CASE REPORT

A 53-year-old, right-handed baggage handler presented with a 6-year history of slowly progressive bilateral dorsal wrist pain that was associated with weakness and impaired key pinch. There was no history of trauma, alcoholism, or steroid intake and no systemic comorbidity such as diabetes or peripheral vascular disease. The pain impaired the patient's ability to perform normal daily activities and carry baggage at work. On examination, there was tenderness localized to the anatomical snuffbox but no associated swelling or effusion. There was no gross joint instability and thumb and wrist movements were normal bilaterally. Grip strength was 14 kg on the right and 20 kg on the left.

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Plain radiographs demonstrated increased trapezoid density bilaterally with subchondral sclerosis (Fig. 1). Magnetic resonance imaging (MRI) demonstrated wide areas of signal change including low signal intensity on T1-weighted images and increased intensity on T2-weighted images in both trapezoids, consistent with avascular necrosis (Fig. 2). The entire left trapezoid was affected, whereas the right was only partially affected. Blood tests revealed no markers for autoimmune disease and no biochemical abnormalities. Technetium-99m bone scintigraphy showed moderate bilateral uptake of both trapezoids.

The patient received nonsurgical treatment that included analgesics and wrist splinting without thumb immobilization, followed by physiotherapy to improve wrist movement and strength. He noted a near complete resolution of symptoms and signs within 9 months. On clinical and radiological assessment at 5 years, the patient was completely pain free with no requirement for regular analgesics. Wrist movement was full bilaterally, and grip strength had increased to 34 kg on the right and 26 kg on the left. Magnetic resonance imaging demonstrated evidence of partial revascularization of the trapezoids and no bone fragmentation (Fig. 2). The subchondral sclerosis persisted on follow-up x-rays but with less intensity. The only residual symptom was occasional left-sided wrist pain associated with heavy lifting. During the treatment period, the patient changed from baggage handler to administrator. However, according to the patient, the job change was unrelated to wrist pain.

DISCUSSION

Osteonecrosis of the carpals is rare. Risk factors for avascular necrosis include a history of trauma, alcohol intake, and steroid medication.^{7,8} In this case, there were no identifiable predisposing factors and no single



FIGURE 1: **A** Right wrist and **B** left wrist posteroanterior radiographs demonstrating sclerosis of both trapezoids.

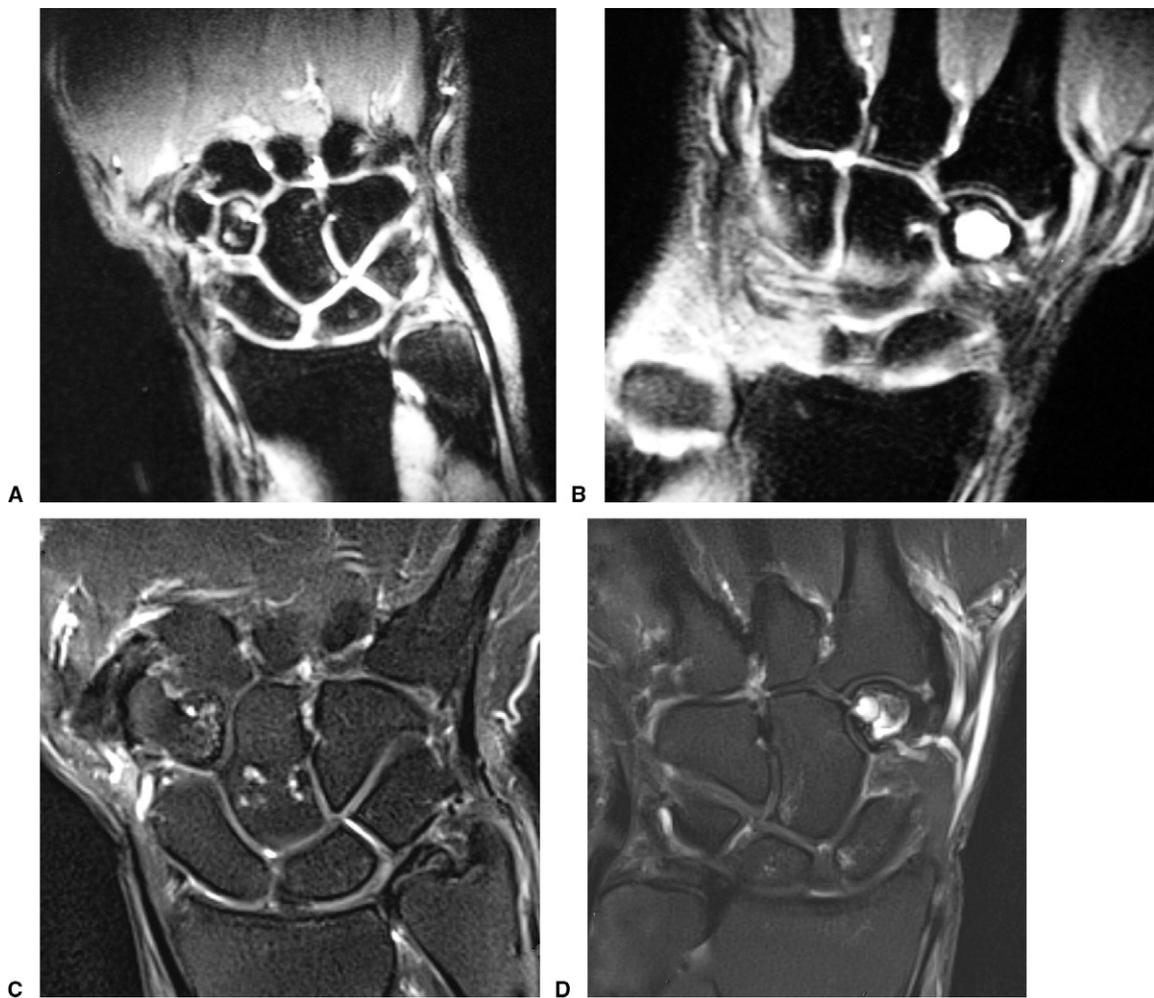


FIGURE 2: **A** Right wrist and **B** left wrist T2-weighted coronal MRI showing avascular necrosis of both trapezoids. **C** Right wrist and **D** left wrist T2-weighted Fat saturation coronal MRI showing partial revascularization 5 years after presentation.

precipitating event, although as a baggage handler the patient may have experienced repetitive microtrauma. The bilateral occurrence raised the possibility of a systemic cause, although none was identified.^{9–12}

Magnetic resonance imaging is the best imaging technique to diagnose avascular necrosis. It is more sensitive than radiographs and bone scintigraphy.^{13–19} Necrotic bone generally produces a low or absent signal

on T1-weighted images because of the absence of normal bone marrow elements, whereas signal uptake is increased on T2-weighted images (bone marrow edema). The intravenous injection of gadolinium improves the MRI specificity of avascular necrosis diagnosis.^{6,13–19} Computed tomography images can help identify secondary changes such as subtle depressions, collapse, or fragmentation. Sometimes osteoarthritis, subchondral cysts, and transient osteoporosis may mimic osteonecrosis on plain films or MRI.

A study by Gelberman and Gross²⁰ analyzed carpal vascular anatomy in cadaveric specimens and identified 3 specific patterns, each with a different avascular necrosis risk profile. The trapezoid and the hamate were classified into group 2, a low-risk group characterized by absence of internal anastomoses. Avascular necrosis of the hamate is also rare.²¹ The trapezoid benefits from a varied extraosseous blood supply including branches from the dorsal radiocarpal, intercarpal, and basal metacarpal arches as well as from the radial recurrent artery. Therefore, the trapezoid does not depend on a single vessel but is nourished by a rich network of dorsal (70%) and palmar vessels (30%), which may explain why it is affected rarely by avascular necrosis.¹⁸

The natural history of osteonecrosis is unpredictable. Bone revascularization may occur or the condition may progress to sclerosis and bone fragmentation.^{22,23} Treatment includes nonoperative measures as well as surgical intervention including arthrodesis, osteotomy, and excision arthroplasty.^{22,24–26} Some authors have advocated the use of vascularized bone grafts to encourage bone revascularization.⁶ In addition to having a protective effect, the rich blood supply of the trapezoid may also make it more likely to recover.^{18,20}

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