SHORT REPORT

# When physiology becomes pathology: the role of magnetic resonance imaging in evaluating bone marrow oedema in the humerus in elite tennis players with an upper limb pain syndrome

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Upper limb stresses are well recognised in tennis, and the normal physiological responses of the humerus to externally applied forces are well defined. Changes to both the microscopic and macroscopic bony architecture are often not apparent on plain radiographs in the early stages of a stress reaction. Bone scintigraphy is more sensitive, but not very specific to subtle changes, as is computer aided tomography. Magnetic resonance imaging (MRI) scans are now used to assess many musculoskeletal injuries, and may allow earlier recognition of changes. This study reports on eight high level tennis players (seven professionals, one highly ranked amateur), who all presented during the course of the 2002 Australian Open and its lead up events. All had an upper limb pain syndrome centred around the elbow. Each underwent an MRI scan. A group of asymptomatic players also had scans of the same area to serve as controls. All symptomatic patients had various levels of bone marrow oedema in their distal humerus, some with periosteal reactions, although most without. No scans revealed a cortical breach. The management of these reactions is problematic in players on an international circuit, as review is sporadic and not always coordinated. The timetabling of international tournaments means it is difficult for top players to obtain adequate rest to avoid injury. Awareness of this early overuse reaction needs to be raised to allow preventive and therapeutic options to be considered to reduce the incidence of this humeral stress response producing an upper limb pain syndrome.

The diagnosis of a bony stress injury is a clinical end point of an overuse phenomenon, which can be due to pure excess workload or as a response to altered biomechanics for any reason. Patients often present with symptoms long before there is evidence of a cortical breach on plain radiography.[1] Imaging with bone scintigraphy is more sensitive, but does not have the subtlety to reveal the true extent of the pathology. Computed tomography scans can show early cortical breaches before completed fractures,[2] but magnetic resonance imaging (MRI) can show intraosseous changes such as bone marrow oedema or periostitis, and localise them very accurately.[3] There have been previous reports of increased cortical thickness and density of the dominant humerus in tennis players.[4–7] The imaging boundary between a normal physiological stress reaction of bone and that seen in a pathological overuse syndrome is poorly defined, and when symptoms occur, the focus is on therapeutic intervention rather than prevention.

We report on the use of MRI in evaluating the severity of intraosseous oedema in the distal humerus in a group of symptomatic elite tennis players. A grading system is proposed that may assist doctors in recommending various periods of rest for players before their stress injury becomes a stress fracture.

## PATIENTS AND METHODS

We report a series of eight highly ranked tennis players who presented at the time of the 2002 Australian Open tennis tournament. There were four men and four women (age range 18–29, mean age 24) all of whom had symptoms of pain in their dominant humerus. One of the players was a junior on a satellite circuit. The elite players were all ranked in the top 100 in the world, with two top 10 players presenting. Each described the pain when serving, continuing after the match. The duration of symptoms varied between two and 16 weeks, with a mean of seven weeks.

Six of the eight players had a history of ipsilateral shoulder and/or elbow injuries, although none were having treatment for these at the time of the investigation.

Ten professional players who were being scanned for other injuries consented to a MRI scan of the humerus of their dominant arm to serve as controls. Each player confirmed that they had not had any symptoms in that arm over the previous six months.

All patients and controls had MRI scanning as part of their work up. These were obtained on a 1.5 T superconducting MR unit (Sigma Horizon; GE Medical Systems, Milwaukee, Wisconsin, USA). Direct axial and coronal localising images were obtained, then two specific sequences: (*a*) axial fast spin-echo (repetition time (milliseconds)/echo time (milliseconds) = 4000/30–45 (effective); 256 × 224 matrix; 4 mm section thickness with no intersection gap); (*b*) axial and coronal short tau inversion recovery (STIR) imaging (5000/45–60; 256 × 256; 3 mm with no gap).

All images were interpreted by a musculoskeletal radiologist and radiology fellow. A sports physician and an orthopaedic surgeon documented the clinical history and examined all patients. All details were correlated and checked by an orthopaedic fellow.

The scans were evaluated for morphology and signal intensity of the humeral cortex, periosteum, and bone marrow. Abnormal marrow signal was assessed for both length involved and the maximal cross sectional area of the humerus. The oedema was graded as mild (0–25%), moderate (25–75%), or severe (75–100%) on the maximal cross sectional area of abnormal marrow signal on axial scans.

Two of the subjects also had bone scintigraphy, and the results of these were graded in standard fashion.

**Table 1** Data on tennis players studied

| Player | Age | M/F | Duration of symptoms | Length (cm) | Oedema | Periosteal reaction | Bone scan uptake |
|---|---|---|---|---|---|---|---|
| 1 | 23 | M | 3 weeks | 10 | Mild | – | Moderate |
| 2 | 29 | M | 7 weeks | 15 | Moderate | – | – |
| 3 | 25 | F | 3 months | 12 | Severe | Mild anteromedial | – |
| 4 | 29 | M | 5 weeks | 7 | Moderate | – | – |
| 5 | 18 | F | 4 months | 9 | Severe | Mild anteromedial | – |
| 6 | 27 | M | 2 weeks | 10 | Mild | – | – |
| 7 | 29 | M | 4 weeks | 12 | Moderate | Moderate posteromedial | – |
| 8 | 19 | F | 2 months | 6 | Severe | – | Intense |

## RESULTS

Table 1 shows the results. Three patients were classified as having a severe bone marrow oedema reaction (fig 1), three a moderate reaction (figs 2 and 3), and two a mild reaction. Two patients with severe oedema also had periosteal changes, and one in the moderate group. No patients graded as having mild oedema had any identified periosteal reaction. Although no statistical analysis could be applied to the data because numbers were too small, there appeared to be a trend correlating duration of symptoms and severity of marrow changes on MRI scan.

Nine of ten controls had no bone marrow or periosteal changes on MRI. One had mild patchy uptake in the central one third of the humerus, but denied symptoms. This player had no history of shoulder pathology (table 2).

### Case examples

#### Case 1

A 27 year old male professional tennis player returned to competitive tennis after a shoulder injury and played in two grand slam finals before reporting distal arm pain for a period of 16 weeks. He could not complete multiple matches without experiencing pain. Investigations included plain radiographs showing hypertrophy of the cortical bone of the humeral shaft, a mildly increased uptake on technetium-99 bone scan, and severe intraosseous oedema in the distal humerus on STIR sequence MRI scans.

Novel treatment of short term, high dose oral corticosteroids was used to allow him to compete in an ITF final, but relative rest formed the mainstay of treatment. The player took a full six months away from tennis, and had full relief of his pain, with a documented decrease in the MRI changes at four months.

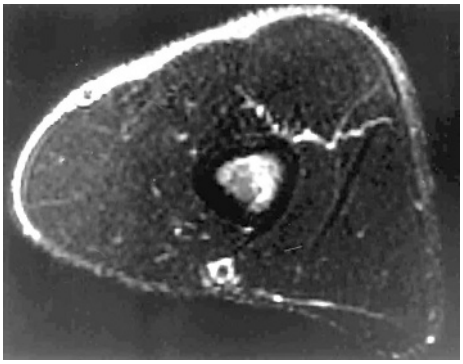

**Figure 2** Axial magnetic resonance imaging scan of the mid-humerus of a 29 year old tennis player with moderate changes. Patient consent has been obtained for publication of this figure.

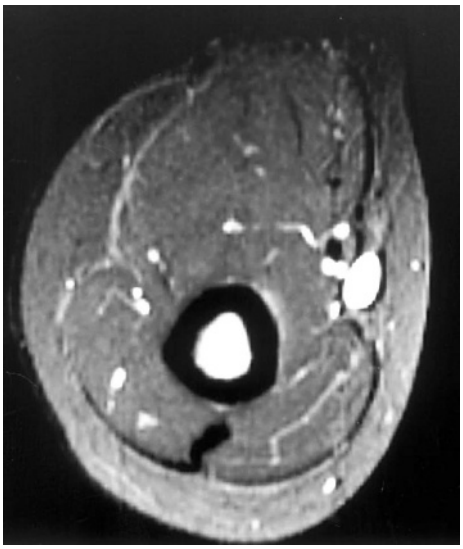

**Figure 1** Axial magnetic resonance imaging scan of the distal humerus of a 25 year old tennis payer with severe changes. Patient consent has been obtained for publication of this figure.

**Figure 3** Longitudinal magnetic resonance imaging scan of the humerus in the 29 year old tennis player with moderate changes. Patient consent has been obtained for publication of this figure.

**Table 2** Data on controls

| Player | Age | M/F | Side | Oedema | Periosteal reaction | Bone scan uptake |
|---|---|---|---|---|---|---|
| 1 | 34 | M | R | Nil | – | – |
| 2 | 21 | M | R | Nil | – | – |
| 3 | 21 | F | R | Nil | – | – |
| 4 | 22 | M | L | Nil | – | – |
| 5 | 26 | F | R | Nil | – | – |
| 6 | 21 | M | L | Nil | – | – |
| 7 | 32 | M | R | Patchy high signal | Nil (confined to midshaft medulla) | – |
| 8 | 28 | F | R | Nil | – | – |
| 9 | 27 | M | R | Nil | – | – |
| 10 | 28 | M | R | Nil | – | – |

The dominant side was scanned in each case.

### Case 2

An 18 year old junior semiprofessional satellite circuit player described four months of vague ''pressure-like'' pain around the elbow with play. She had severe changes on MRI scanning and a slight increase in uptake on nuclear bone scan. She rested for three months before rescanning showed resolution of the changes, and she returned to competitive tennis.

## DISCUSSION

The definition of a stress injury is reported variably, and is often not a concrete diagnosis. A fracture is the natural hard end point, but the bone can be detected reacting to stress across a continuum. The ''dreaded black line'' of the full thickness cortical fracture is seen in femoral necks of long distance runners, or the tibiae in dancers, as well as the classical march fractures in the feet of military personnel.[8 9] Bone scintigraphy alone may show an isolated increase in uptake, as seen in the navicular of running athletes or the scaphoids of gymnasts.[10–12] Finally, MRI marrow changes have been detected in the feet and ankles of asymptomatic runners after exercise, and in Australian Rules footballers with clinical osteitis pubis.[13 14] All this appears to demonstrate a continuum, from normal physiological stress responses to pathological processes caused by stresses placed on bone.

All our patients had pain in the limb under investigation, and all had bone marrow oedema changes on MRI scan. Although too small to analyse statistically, there appeared to be an association between the degree of marrow oedema and the presence of a periosteal reaction. It has been proposed that stress fractures occur as a result of the coalition of multiple microfractures within the trabecular bone.[15] If the repetitive sub-threshold stresses are removed from the area of abnormality, the normal responses of the bone can repair and remodel itself. This should allow resolution of symptoms.

Under normal circumstances, the humerus can tolerate the usual physiological loads without problem. However, if excess stress is exerted on the bone as the result of compensation by the patient for an ipsilateral injury or weakness, a stress response may be initiated. Six of the eight players had documented recent or concurrent shoulder injuries in the same upper limb as that under investigation. This may in part account for the overloading of the bone. To maintain power of the stroke, particularly when serving, or to impart a degree of spin on the ball, a player may overuse uninjured parts of the limb to compensate for lack of power generation more proximally. With the humerus, this usually involves excessive torsional stress, which is less well tolerated than compressive stress.

Early diagnosis of a concurrent pathology, with appropriate treatment and rehabilitation, may prevent an overuse injury occurring as a consequence. Coaches and therapists may be able to recognise a change in a player's technique early, possibly using video recordings, and advise the seeking of a medical opinion before serious damage occurs.

Quantification of MRI changes allows identification of these early bone stress reactions, and can detect subtle changes in both marrow and periosteal oedema.[3 16 17] Prevention of full blown stress fractures is critical if the athlete is to continue at his or her level of sport without requiring a significant period of time away because of injury.

The treatment for stress reaction injuries has classically been rest from the aggravating activity. Anecdotal treatment with short course oral corticosteroids, hyperbaric oxygen, and prostaglandin analogues have all been reported.[18] Application of external capacitive coupled electric fields has been used to enhance the healing process.[19] If a stress injury has progressed to a fracture, and its chronicity suggests a non-union, adequate stabilisation of the injury, with unloading of the area of injury, may be needed.[20]

## SUMMARY

We present a series of eight professional and semiprofessional tennis players with a previously unreported stress injury to their distal humerus. Some of the players had previously seen other medical and paramedical practitioners, who had given alternative diagnoses. Ten controls were also scanned, with one showing very slight changes. It is hoped that this paper will serve to highlight this new injury and increase awareness among practitioners who treat tennis players at this level presenting with somewhat vague symptoms around the elbow. An early and accurate diagnosis allows a suitable rehabilitation programme to be constructed with the player and coach to minimise missed tournaments and training and prevent further complications.

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## COMMENTARY

This is a landmark paper which describes a significant stress injury in the distal humerus of the dominant arm of professional and semiprofessional players. The fact that eight players were diagnosed within a single tournament and its lead up matches suggests that there may be a significant prevalence of such injuries among elite level tennis players. Future studies may shed more light on this. Six of the eight players had a history of recent or concurrent injury to the ipsilateral upper limb (shoulder or elbow). This suggests that the stress reaction may have resulted from over-compensation for suboptimal function in the upper limb. There is plenty of potential here for larger prospective studies to document the genesis of such injuries in players who return to play after shoulder/elbow injuries. This paper provides much food for thought for sports medicine practitioners treating upper limb injuries in elite tennis players.

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