

Case reports

Department of Nuclear Medicine, Chang Gung Memorial Hospital, School of Medicine, Chang Gung University, Taipei, Taiwan, Republic of China

P-F Kao
K-Y Tzen
K-J Lin
M-F Tsai
T-C Yen

Division of Immunology, Allergy and Rheumatology
J-Y Chen

Correspondence to:
Dr Pan-Fu Kao, Department of Nuclear Medicine, Chang Gung Memorial Hospital, 199 Tung Hwa North Road, Taipei, Taiwan, Republic of China.

Accepted for publication
4 February 1998

Rectus abdominis rhabdomyolysis after sit ups: unexpected detection by bone scan

Pan-Fu Kao, Kai-Yuan Tzen, Ji-Yih Chen, Kun-Ju Lin, Ming-Fong Tsai, Tzu-Chen Yen

Abstract

Uptake of technetium-99m methylene diphosphonate by the rectus abdominis muscle was unexpectedly found in a 29 year old man who had started to perform 30 to 40 sit ups a day for five days before the bone scan. After a week of rest, serum creatine kinase activity was still abnormal but muscle uptake of technetium-99m methylene diphosphonate had ceased. This specific muscle injury after short term sit ups as well as the resolution of the

phenomenon within a week are of interest.

(*Br J Sports Med* 1998;32:253–254)

Keywords: rectus abdominis; rhabdomyolysis; bone scan; technetium-99m; sit ups

Muscle uptake of technetium-99m (^{99m}Tc) bone tracers is seen in many conditions involving muscle damage, including electrical burns,¹ rhabdomyolysis,² and polymyositis.³ With respect to rhabdomyolysis after overexertion, specific muscle group injuries after different types of exercise have been reported: the pectoralis major after vigorous push ups,⁴ the quadriceps and hamstring muscles of marathon runners,⁵ and the teres major, triceps, biceps, and brachioradialis of weightlifters.⁶ This case report shows technetium-99m methylene diphosphonate (^{99m}Tc -MDP) uptake on rhabdomyolysis of the rectus abdominis muscle after sit ups and its resolution after one week of rest.

Case report

A 29 year old man was referred for a bone scan for lower back pain and to rule out sacroiliitis. The bone scan was performed three hours after intravenous injection of 925 MBq of ^{99m}Tc -MDP. Intense uptake of ^{99m}Tc -MDP was seen over a large area of the abdominal region (fig 1A). Single photon emission tomography (SPET) with ^{99m}Tc -MDP over the abdominal region showed uptake of radiotracer at the rectus abdominis muscle (fig 2). Questioning of the patient revealed that he had performed 30 to 40 sit ups a day for the preceding five days and that he felt pain in the anterior abdominal wall. The patient had not exercised regularly in the past. He started to do sit up exercises because he was told that this exercise might help his lower back pain. The bone scan results prompted us to check serum creatine kinase, lactate dehydrogenase, and myoglobin to rule out rhabdomyolysis. Serum creatine kinase activity had increased to 12 586 U/l (normal range 15–130 U/l) with 100% of the MM isoenzyme on the day after the bone scan. Serum total lactate dehydrogenase activity had increased to 149 U/l (normal range 47–140 U/l). However, serum myoglobin was undetectable (<50 µg/l).

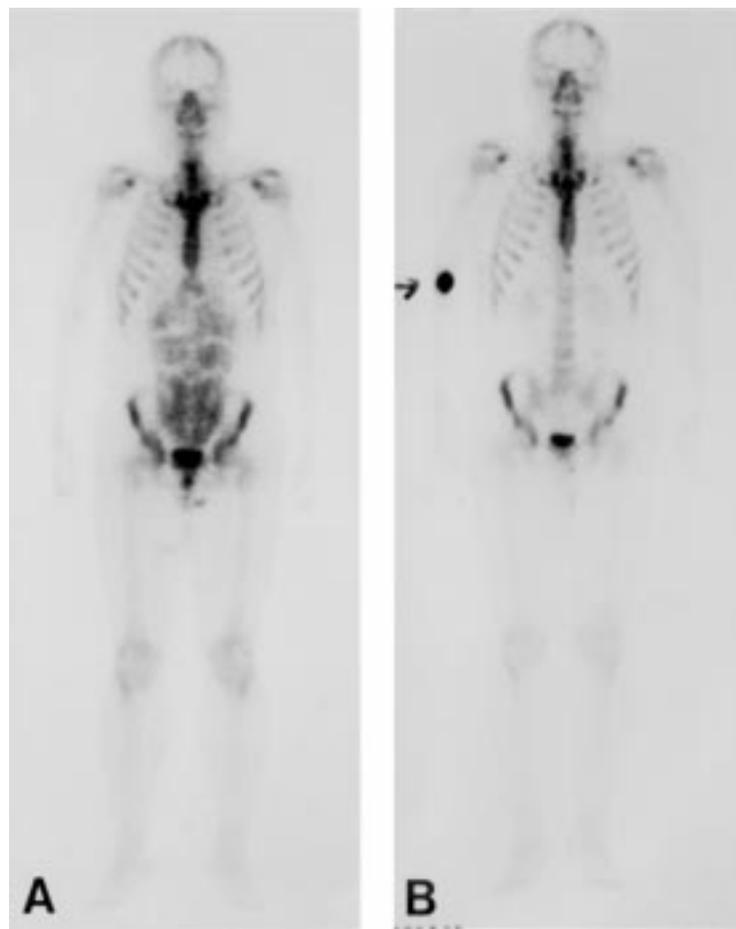


Figure 1 Anterior view of (A) the initial whole body technetium-99m methylene diphosphonate bone images and (B) the follow up bone scan one week later. The rectus abdominis muscle attaches above to the front of the xyphoid process and to the fifth to seventh costal cartilages and below to the pubic crest and symphysis. Signs of uptake of radiotracer had disappeared after one week.

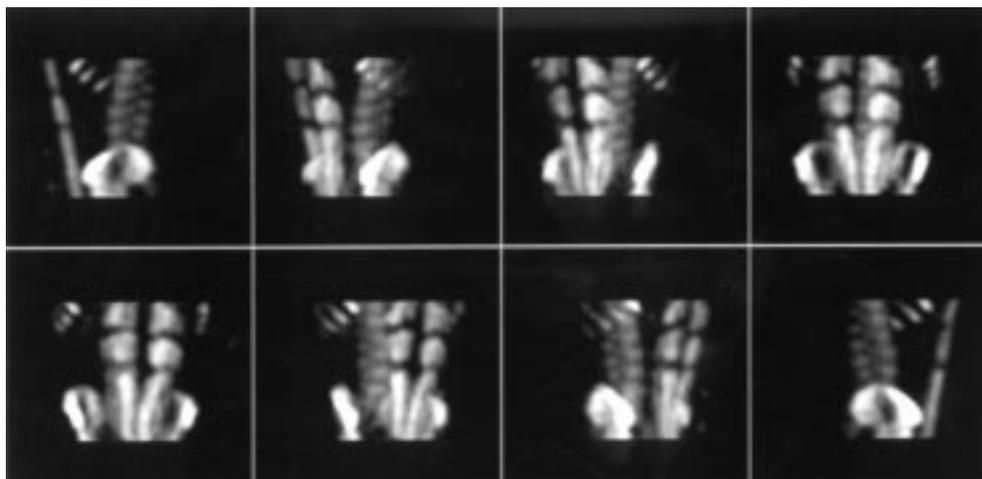


Figure 2 Abdominal single photon emission tomography with volume render display clearly showing radiotracer uptake in the rectus abdominis muscle with several photon-penetrating tendinous intersections transversed on the muscle. The lateral image shows a long and thin muscle uptake at the anterior portion of the abdomen.

After a week of rest, the anterior abdominal pain had disappeared and serum creatine kinase activity had decreased to 208 U/l (100% of MM isoenzyme) which was still above normal. Serum total lactate dehydrogenase activity had returned to normal (58 U/l). The follow up bone scan showed no uptake of ^{99m}Tc -MDP by the rectus abdominis muscle (fig 1B).

Discussion

The specific muscle that was exercised in this patient was localised by ^{99m}Tc -MDP uptake. As previously reported, overexertion by specific types of exercise may induce damage of specific muscle groups.⁴⁻⁶ Rectus abdominis muscle rhabdomyolysis after short term sit ups has not been reported in the literature. The chief function of the rectus abdominis muscle is flexion of the trunk against resistance and, in supine positions, lifting of the chest and, indirectly, of the head.⁷ Therefore overexertion through performing sit ups may cause damage to the rectus abdominis muscle.

In 1985, Frymoyer *et al*⁸ reported bone scan findings of seven cases of acute renal failure due to rhabdomyolysis from a variety of conditions. All of the initial scans in the study, performed between day three and 12, showed different intensity and extent of radiotracer uptake in the injured muscles. Follow up bone scans performed in three cases between days 22 and 31 showed marked but not complete resolution of the injury. In the present case, the ^{99m}Tc -MDP bone scan showed rhabdomyolysis of the rectus abdominis muscle, which was not initially suspected. Also, the resolution of the rhabdomyolysis after a week of rest was earlier than expected.

Siegel *et al*⁹ suggested the bone scan as a way of quantifying extent of muscle injury. Frymoyer *et al*⁸ supported the suggestion and reported that the higher the serum creatine kinase level the greater the radiotracer uptake in the injured muscle. We noticed that, in the above three cases, muscle uptake of ^{99m}Tc had not completely ceased between days 22 and 31, and serum creatine kinase levels were 4 to 10

times higher (49 000, 49 000, and 134 800 U/l) than that of our case.⁸ In a prospective study, Tiidus and Ianuzzo¹⁰ reported that increasing intensity and duration of muscular exercise resulted in increasing serum creatine kinase activity 24 hours after exercise. High intensity short duration exercise resulted in higher serum creatine kinase activity.¹⁰ Therefore we postulated that the more severe the muscle damage, the higher the serum creatine kinase level and the longer it takes for radiotracer uptake to cease as assessed by the follow up bone scan. Further work in more patients is required to confirm this hypothesis.

This case emphasises the potential dangers of the unaccustomed overexertion of even only 30 to 40 sit ups a day, and shows the speed of resolution of the problem after only a week of rest. Muscle damage in unfit subjects may be more common than is recognised. A bone scan is a useful tool in diagnosis and follow up of overexertion induced muscle damage. The deposition of radiotracer in damaged muscle is reversible, and the disappearance of the radiotracer can be related to healing. In addition, the muscle damage resolution on the bone scan was seen before serum creatine kinase activity returned to normal.

- 1 Kao PF, Tzen KY, Chang LY, *et al*. Tc-99m MDP scintigraphy in high-voltage electrical burn patients. *Nucl Med Commun* 1997;18:846-52.
- 2 Sagar W, Meckelburg RL, Chaikin HL. Bone scan in rhabdomyolysis. *Clin Nucl Med* 1980;5:321.
- 3 Spies SM, Swift TR, Brown M. Increased Tc-99m polyphosphate muscle uptake in a patient with polymyositis: case report. *J Nucl Med* 1975;16:1125.
- 4 Lentle BC, Percy JS, Rigal WM, *et al*. Localization of Tc-99m pyrophosphate in muscle after exercise. *J Nucl Med* 1978;19:2234.
- 5 Matin P, Lang G, Carretta R, *et al*. Scintigraphic evaluation of muscle damage following extreme exercise. *J Nucl Med* 1983;24:308-11.
- 6 Valk P. Muscle localization of Tc-99m MDP after exertion. *Clin Nucl Med* 1984;9:493-4.
- 7 Clemente CD. Fasciae and muscles of the abdomen. In: Clemente CD, ed. *Gray's anatomy*, 30th American ed. Philadelphia: Lea & Febiger, 1985:483-498.
- 8 Frymoyer PA, Giammarco R, Farrar FM, *et al*. Technetium Tc 99m medronate bone scanning in rhabdomyolysis. *Arch Intern Med* 1985;145:1991-5.
- 9 Siegel BA, Engel WK, Derrer EC. Tc-99m diphosphonate uptake in skeletal muscle: a quantitative index of acute damage. *Neurology* 1975;25:1055-8.
- 10 Tiidus PM, Ianuzzo CD. Effects of intensity and duration of muscular exercise on delayed soreness and serum enzyme activities. *Med Sci Sports Exerc* 1983;15:461-5.

Displaced avulsion of the ischial apophysis: a hamstring injury requiring internal fixation

Christopher T J Servant, Cledwyn B Jones

Abstract

A case is reported of an adolescent sprinter who was chronically disabled by pain after non-operative management for an acute hamstring injury. He had sustained an avulsion fracture of the ischial apophysis with displacement of 2.5 cm. Avulsion fractures of the ischial apophysis with displacement of 2 cm or more are unusual, but they frequently result in a symptomatic non-union, and early diagnosis, open reduction, and internal fixation is to be encouraged.

(*Br J Sports Med* 1998;32:255–257)

Keywords: hamstring; ischial apophysis; avulsion fracture; hip

The ischial apophysis (a secondary ossification centre forming the ischial tuberosity) may become avulsed from the innominate bone at any age between its appearance (at 13 to 15 years) and its fusion (at 16 to 25 years). The mechanism of injury is usually sudden contraction of the hamstrings or, less commonly, adductor magnus. The rest of the ischium is stabilised by the attachment of the strong sacrotuberous ligament to the sacrum. The patient is usually engaged in strenuous sporting activity in which there is a strong contraction of the hamstrings or an uncontrolled forceful hamstring stretch. Such activities include sprinting, long jumping, and hurdling.

We report the case of an adolescent sprinter who was chronically disabled by pain after non-operative management for an acute hamstring injury. We discuss the merits of early surgical fixation of significantly displaced avulsions of the ischial apophysis.

Case report

A 16 year old male athlete was sprinting in a school 100 m race when he pulled up suddenly with sharp pain in the proximal part of the right posterior thigh. He was unable to walk afterwards without considerable discomfort.

He attended his local casualty department the same evening, where it was thought that he had sustained a soft tissue injury to the hamstring muscles. He was discharged home with a pair of crutches to help mobilisation. Two weeks later there had been no improvement in his symptoms, and his general practitioner prescribed simple analgesics. Subsequently he received ten sessions of physiotherapy, including ultrasound therapy and stretching and strengthening exercises. After this, he was able to jog but he could not sprint or participate in running sports because of



Figure 1 Anteroposterior radiograph of the right hip, showing an avulsed ischial apophysis. The apophysis is displaced by 2.5 cm.

cramp-like pain in his posterior right thigh. He was unable to sit on the right ischial tuberosity.

Nine months after the injury, plain radiographs were obtained (fig 1), showing an avulsion of the right ischial apophysis at the origin of the hamstring muscles (fig 2) with 2.5 cm displacement of the apophysis. An orthopaedic opinion was urgently sought. On clinical examination at this stage he had mild tenderness over the right ischial tuberosity but no clinical weakness of the hamstring muscles.

In the end, 14 months after the injury, an open procedure was performed to reattach the avulsed ischial apophysis. Through a gluteal crease incision, with identification and protection of the sciatic nerve, the avulsed apophysis was exposed, cleaned of fibrous tissue, reduced, and fixed using three screws (fig 3). After two weeks of bed rest, the patient was mobilised; partial weight bearing was allowed for six weeks with the aid of crutches. Hip flexion was prohibited for six weeks and a knee extension splint was provided.

Clinical and radiological union of the ischial apophysis had occurred by review at three months, when gentle running was allowed. On review at six months, the patient had begun to experience a grating discomfort on walking and running. Clinical and radiological examination suggested that one of the three screws had loosened. One year after the initial operation all three screws were removed, under general

Institute of Sports
Medicine, Royal
United Hospital, Bath,
United Kingdom
C T J Servant
C B Jones

Correspondence to:
Mr C T J Servant,
Department of
Orthopaedics, Royal United
Hospital, Combe Park, Bath
BA1 3NG, United Kingdom.

Accepted for publication
13 February 1998

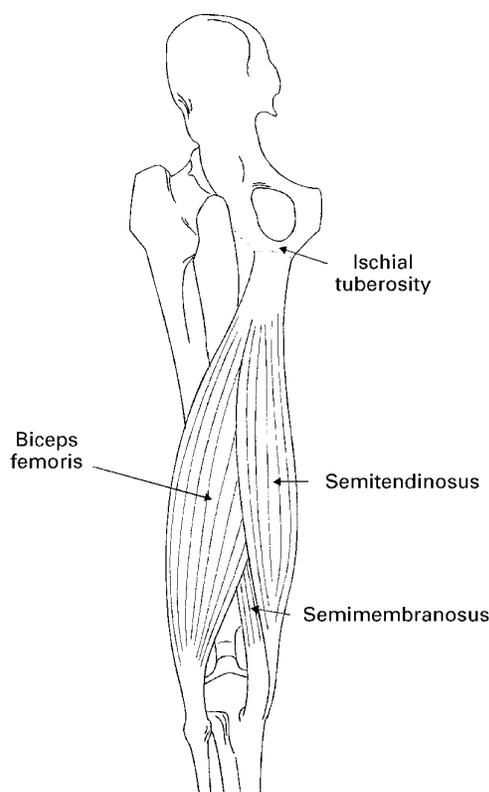


Figure 2 Posterior view of the left thigh showing the attachments of the hamstring muscles. The semitendinosus and the biceps femoris muscles attach to the ischial tuberosity as a common tendon. The semimembranosus tendon lies deep to these two and may either be separate or part of the common tendon.

anaesthesia, and it was confirmed that one screw was loose. He was mobilised with full weight bearing immediately after the operation.



Figure 3 Anteroposterior radiograph of the right hip, showing reduction and internal fixation of the ischial apophysis, using three cannulated partially threaded cancellous screws.

Subsequently there were no further problems. The patient soon returned to running and competitive sports, including rugby at university level. Three years after removal of the screws he had no limp, discomfort, or stiffness. Over this time he was able to sprint comfortably and he played rugby twice a week for his university.

Discussion

Hamstring injuries are routinely managed non-operatively.¹ However, caution should be exercised with proximal hamstring injuries, especially in adolescent children. Before the ischial apophysis closes in late adolescence, athletes are susceptible to an avulsion injury through the physis. This may be a bony avulsion or, more rarely, a cartilaginous avulsion occurring before the secondary ossification centre has appeared.

Most ischial avulsion fractures heal with a carefully directed progressive rehabilitation programme. Indeed many authors²⁻⁵ believe that early surgical excision or fixation of the avulsed fragment is not indicated, regardless of the amount of displacement, and it should only be considered if disability persists after a full conservative regimen has been carried out.

Although it is undoubtedly true that undisplaced or minimally displaced avulsion fractures can be expected to unite with few problems, accumulated evidence indicates that marked displacement frequently results in chronic disability.⁶⁻¹² The widely separated fragments are likely to heal with a fibrous non-union that often gives rise to buttock pain, an ischial mass, or significant weakness of knee flexion, resulting in a reduced level of athletic performance. Rarely, there may be sciatic nerve impingement.⁸ Our reported case confirms the observation that open reduction and internal fixation of avulsions with more than 2 cm displacement can produce good results.^{7 9 10 11 13 14} Normal function can be restored in both acute and chronic cases as a result of restoration of hamstring length by anatomical reduction and by elimination of pain on hamstring stretching.

Prolonged attempts at rehabilitation before open reduction and internal fixation will usually only delay return to previous levels of athletic performance.

We suggest the following management for a suspected hamstring injury in a child, particularly a proximal hamstring injury in an adolescent athlete.

A careful history of the injury usually includes sudden severe buttock or thigh pain during strenuous athletic activity, often with the sensation of a crack, snap, or pop. Examination shows local tenderness, and a gap may be palpable in the area of the ischial apophysis. Plain radiographs should be taken. An anteroposterior radiograph of the pelvis allows comparison with the opposite side.^{7 15} If the patient is young enough for the secondary ossification centre to have not yet appeared, an avulsion of the apophysis will not be visible radiographically, and magnetic resonance imaging (MRI) is recommended.¹⁵ MRI is also useful in showing

any soft tissue injury that may require surgical repair, such as rupture of the conjoined tendon of the hamstring muscles.¹⁵

If there is displacement of the ischial apophysis of 2 cm or more, open reduction and internal fixation with two or more screws should be carried out early. Cannulated screws allow accurate placement using a guidewire. In chronic cases of a symptomatic fibrous non-union, excision of the fibrous tissue and internal fixation produces a good functional result. Excision of the avulsed fragment should be reserved for those cases in which satisfactory reduction cannot be achieved.⁹

If there is no or minimal displacement of the avulsed apophysis, then conservative management should be pursued. Non-apophyseal injuries involving the musculotendinous junction or the muscle belly itself should also be treated non-operatively.¹ Complete rupture of the conjoined hamstring tendon is rare but is best treated by surgical repair.¹⁶

Non-operative and post-operative management consists of a well planned rehabilitation programme. This should concentrate on a short period of rest with relaxation of the hamstring muscle group, followed by protected weight bearing, and then a progressive regimen of hamstring stretching and eccentric strengthening.²

- 1 Agre JC. Hamstring injuries. Proposed aetiological factors, prevention and treatment. *Sports Med* 1985;2:21-33.
- 2 Metzmaker JN, Pappas AM. Avulsion fractures of the pelvis. *Am J Sports Med* 1985;13:349-58.
- 3 Canale ST. Fractures and dislocations in children. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*. 8th ed. St Louis: Mosby-Year Book, 1992:1127-8.
- 4 King J. Lower limb injuries in adolescence and childhood. In: Maffulli N, ed. *Sports medicine in childhood and adolescence*. London: Mosby-Wolfe, 1995:37-9.
- 5 Fernbach SK, Wilkinson RH. Avulsion injuries of the pelvis and proximal femur. *AJR Am J Roentgenol* 1981;137:581-4.
- 6 Schlonsky J, Olix ML. Functional disability following avulsion fracture of the ischial epiphysis: report of two cases. *J Bone Joint Surg [Am]* 1972;54:641-4.
- 7 Poulsen TK, Enggaard TP. Avulsion fracture of the ischial tuberosity. A rare lesion whose early diagnosis and correct treatment may prevent late sequelae. *Ugeskr Laeger* 1995;157:6140-1.
- 8 Miller A, Stedman GH, Beisaw NE, et al. Sciatica caused by an avulsion fracture of the ischial tuberosity. A case report. *J Bone Joint Surg [Am]* 1987;69:143-5.
- 9 Wooton JR, Cross MJ, Holt KWG. Avulsion of the ischial apophysis: the case for open reduction and internal fixation. *J Bone Joint Surg [Br]* 1990;72:625-7.
- 10 Kujala UM, Orava S. Ischial apophysis injuries in athletes. *Sports Med* 1993;16:290-4.
- 11 Martin TA, Pipkin G. Treatment of avulsion of the ischial tuberosity. *Clin Orthop* 1957;10:108-18.
- 12 Sundar M, Carty H. Avulsion fractures of the pelvis in children: a report of 32 fractures and their outcome. *Skeletal Radiol* 1994;23:85-90.
- 13 Howard FM, Piha RJ. Fractures of the apophyses in adolescent athletes. *JAMA* 1965;192:842-4.
- 14 Prunner RA, Johnston CE. Avulsion fracture of the ischial tuberosity. *Pediatric Orthopedics* 1990;13:357-8.
- 15 Brandser EA, El-Khoury GY, Kathol MH, et al. Hamstring injuries: radiographic, conventional tomographic, CT and MR imaging characteristics. *Radiology* 1995;197:257-62.
- 16 Orava S, Kujala UM. Rupture of the ischial origin of the hamstring muscles. *Am J Sports Med* 1995;23:702-5.

Iatrogenic acute hyponatraemia in a college athlete

Rob Herfel, C Keith Stone, Shaheed I Koury, Jim J Blake

Abstract

Hyponatraemia is one of the most common electrolyte abnormalities, leading to significant morbidity and mortality. In the most basic sense, hyponatraemia can be due to sodium loss or fluid excess. The extracellular fluid status is used to clinically divide hyponatraemia into three categories to help to determine both the cause and treatment required. Hyponatraemic patients can be categorised on the basis of their fluid status as hypovolaemic, euvolaemic, or hypervolaemic. Another distinction to make in evaluating hyponatraemia is whether the onset was acute or chronic in nature. The case presented here is iatrogenic acute hypervolaemic hyponatraemia in a college athlete. The patient presented in respiratory distress with an altered mental status after administration of hypotonic fluids for treatment of muscle cramps. Treatment included intubation, water restriction, and furosemide, to which he responded favourably. Hyponatraemia should be in the differential diagnosis for patients presenting after intravenous fluid administration.

(*Br J Sports Med* 1998;32:257-258)

Keywords: athlete; hyponatraemia; iatrogenic; acute

Hyponatraemia is one of the most common electrolyte abnormalities that lead to significant morbidity and mortality. It is classified as either acute or chronic as well as hypervolaemic, euvolaemic, or hypovolaemic in order to provide appropriate treatment. We present a case of iatrogenic acute hypervolaemic hyponatraemia in a college athlete.

Case report

A 22 year old black male college football player experienced leg cramps after practice and reported to the team doctor. He was diagnosed as having muscle cramps secondary to dehydration. Therefore 5 litres 0.45% normal saline with 5% dextrose was administered intravenously along with 3 litres of liquids by mouth over a five hour period. He later experienced shortness of breath and mental status changes and was brought to the University of Kentucky emergency department. Physical examination showed that he was in respiratory distress with confusion. Vital signs were: blood pressure 146/78 mm Hg; respiratory rate 48 breaths/minute; temperature 37°C; pulse 89 beats/

Department of
Emergency Medicine,
University of Kentucky
College of Medicine,
Lexington, KY 40536,
USA

R Herfel
C K Stone
S I Koury
J J Blake

Correspondence to:
Dr C K Stone.

Accepted for publication
26 February 1998

minute. Examination of the head, eyes, ears, nose, and throat showed normal pupils, no nystagmus, positive gag reflex, and no evidence of trauma. Cardiovascular examination showed a normal rate without murmurs or rubs. Auscultation of the lungs disclosed rales and crackles in all lung fields. Breath sounds, however, were equal. The abdomen was soft and non-tender with normal bowel sounds. Extremities showed no rashes or oedema. Neurological examination showed the patient to be confused and agitated, and intermittently he would not follow commands. He could move all four extremities and had no focal deficits. Chest x ray detected pulmonary oedema with a normal cardiac silhouette. Arterial blood gas tests showed a pH of 7.37, P_{CO₂} 4.79 kPa, P_{O₂} 8.38 kPa, and O₂ saturation 91%. Electrolytes were as follows: sodium 121 mmol/l, potassium 3.8 mmol/l, chloride 91 mmol/l, bicarbonate 21 mmol/l; blood/urea nitrogen was 3.93 mmol/l, creatinine 106.1 µmol/l, glucose 6.38 mmol/l, and creatine kinase 40 000 U/l. Initial urine myoglobin was negative. A computed tomography scan of the head was normal.

The patient was diagnosed as having hyponatraemia, pulmonary oedema, and probable rhabdomyolysis. He was admitted to the intensive care unit where he responded to treatment, which included intubation, fluid restriction, and furosemide. Myocardial injury was ruled out by serial electrocardiograms and the results of cardiac enzyme assays. An echocardiogram was normal. He was transferred to the floor after two days in the intensive care unit and was discharged on day three without complications.

Discussion

Hyponatraemia is due to sodium loss or fluid excess.¹ Hyponatraemic patients can be categorised on the basis of their fluid status as hypovolaemic, euvolaemic, or hypervolaemic.² The case presented is classified as hypervolaemic hyponatraemia. The underlying causes of hypervolaemic hyponatraemia include congestive heart failure, liver cirrhosis, nephrotic syndrome, renal insufficiency, and iatrogenic water intoxication. Iatrogenic water intoxication is a common cause of inpatient hyponatraemia,^{2,3} but is uncommon in the outpatient or prehospital setting.

Hyponatraemia has deleterious effects on the central nervous, cardiovascular, musculoskeletal, and renal systems. Owing to the effects on multiple organ systems, the signs and symptoms are multiple and non-specific. They include headache, nausea, vomiting, weakness, anorexia, and muscle cramps. More severe signs and symptoms such as rhabdomyolysis, disorientation, coma, seizures, diminished reflexes, pseudobulbar palsy, and focal neurological deficits are possible.^{1,4} Acute hyponatraemia is associated with a higher mortality than chronic hyponatraemia.

Hyponatraemia may be acute (onset 24–72 hours) or chronic.^{1,4–6} The severity and dura-

tion of onset both affect the clinical presentation. An acute disturbance causes symptoms at a higher serum sodium level compared with a chronic change. Acute versus chronic is important to discern as it affects the treatment and complications.

Treatment of hyponatraemia varies depending on the acuity, the severity of symptoms, the serum sodium level, and the patient's volume status. The goal in treating symptomatic hyponatraemia is to maintain tissue perfusion and decrease cerebral oedema while avoiding the complications of treatment. Chronic hyponatraemia is corrected more slowly than acute hyponatraemia to avoid the possibility of central pontine myelinolysis. Acute hyponatraemia can be corrected more rapidly with little chance of central nervous system complications. The rate of correction for acute hyponatraemia may be as high as 2 mmol/l per hour, while treatment of chronic hyponatraemia must limit the rise to 0.5 mmol/l per hour.²

Treatment of symptomatic hypervolaemic hyponatraemia involves water restriction, diuretic therapy, and administration of both normal and hypertonic saline. Water restriction is the first line of treatment, but cannot be used alone in patients with severe symptoms. Many authors state that hypertonic saline can be used when the patient exhibits severe symptoms or if the serum sodium level is less than 115–120 mmol/l. Hypertonic saline was not used in the case presented because the patient was otherwise healthy and responded well to diuresis.

Iatrogenic hypervolaemic hyponatraemia should be considered as a possible diagnosis in patients, especially athletes, presenting to the emergency department after the administration of intravenous fluids. The underlying cause of hypervolaemic hyponatraemia must be sought while initiating treatment. The severity of symptoms, duration of onset, fluid status, and the serum sodium level should be quickly determined to guide treatment. The treatment should consist of a combination of fluid restriction, diuretics, and either normal or hypertonic saline. Rapid identification and treatment of acute hyponatraemia can lead to decreased morbidity and mortality.

This work was presented at the Southern Medical Association's 91st Annual Assembly, at Charlotte, North Carolina, USA, on November 6–9, 1997.

- Mulloy AL, Caruana RJ. Hyponatremic emergencies. *Med Clin North Am* 1995;79:155–68.
- Wilson RF, Barton C. Fluid and electrolyte problems. In: Tintinalli JE, Ruiz E, Krome RL, eds. *Emergency medicine: a comprehensive study guide*, ed 4. New York: McGraw-Hill, 1996:118–40.
- Arief AL. Management of hyponatremia. *BMJ* 1993;307:305–8.
- Sterns RH. The management of hyponatremic emergencies. *Crit Care Clin* 1991;7:127–42.
- Hoyer J. Management of symptomatic hyponatremia: dependence on the duration of development. *J Intern Med* 1994;235:497–501.
- Oh MS, Carroll HJ. Disorders of sodium metabolism: hyponatremia and hyponatremia. *Crit Care Med* 1992;20:94–103.

Sensitivity of reticulocyte indices to iron therapy in an intensely training athlete

Michael J Ashenden, Geoffrey P Dobson, Allan G Hahn

Abstract

Iron deficiency anaemia, and its debilitating effect on performance, is an area of concern for many female athletes. Automated technologies that analyse individual reticulocytes may provide a sensitive measure of bone marrow response to iron supplementation. The reticulocyte characteristics of a female volleyball player with frank iron deficiency anaemia, and her subsequent response to oral iron therapy, are reported.

(*Br J Sports Med* 1998;32:259–260)

Keywords: iron deficiency anaemia; reticulocytes; female athletes; Technicon H*3

Department of Physiology and Applied Nutrition, Australian Institute of Sport, Canberra, Australia

M J Ashenden
A G Hahn

Department of Physiology and Pharmacology, James Cook University of North Queensland, Townsville, Australia
G P Dobson

Correspondence to: Michael J Ashenden, Department of Physiology and Applied Nutrition, Australian Institute of Sport, PO Box 176, Belconnen, ACT 2616, Australia.

Accepted for publication 13 February 1998

Female athletes have been regarded as being susceptible to iron deficiency anaemia based primarily on the low serum ferritin values often encountered in this population.¹ Reliance on low serum ferritin values as a single indication of iron deficiency is an unreliable diagnostic tool² since it is susceptible to several pathological and physiological conditions that may confound interpretation.³

An alternative to biochemical indices has been to diagnose iron deficiency anaemia on the basis of mature red blood cell parameters. However, iron deficiency has to be present for a long time before the indices of the mature cell population change sufficiently to be detectable by traditional haematology analysers.⁴ Many modern automated blood cell counters now permit similar red cell parameters to be measured directly in reticulocytes, which have only recently been released from the bone marrow and thus provide a more current indication of bone marrow status.⁵ One such analyser, the

Technicon H*3 (Bayer Diagnostics, Tarrytown, New York, USA), stains and identifies the remnant RNA within reticulocytes, and performs cell by cell analysis of 20 000 individual reticulocytes measuring the haemoglobin concentration (corpuscular haemoglobin concentration mean; CHCMr) and cell volume, from which is derived the haemoglobin content (corpuscular haemoglobin content; CHr) of the newly released cells.

The amount and concentration of haemoglobin within reticulocytes has been shown to differ between healthy subjects and iron deficient patients,^{5,6} and the amount of haemoglobin contained in each cell was found to increase in female patients with iron deficiency anaemia after oral iron therapy.⁴ It would be highly desirable if similar results were found in highly trained athletes, since early detection of iron deficient erythropoiesis can prevent the long term consequences of iron deficiency anaemia⁴ and so avoid any performance impairment. This technology has only recently been applied to athletic populations, and it is unclear whether reticulocyte indices are sensitive to iron deficiency in intensely training athletes.

Case report

An 18 year old female international level volleyball player provided a venous blood sample for routine monitoring at the beginning of her scholarship at the Australian Institute of Sport. Haemoglobin concentration was 11.4 g/dl and serum ferritin 10 µg/l (day 1). It was found on dietary counselling that the athlete had an aversion to consuming animal flesh. She was prescribed 350 mg/day Ferrogradumate (ferrous sulphate, equivalent to 105 mg elemental iron; Abbott, Sydney, NSW, Australia) and encouraged to consume two or three servings of red meat per week. Reticulocyte characteristics were monitored weekly using earlobe blood samples (days 7, 16, and 22). A progressive increase in CHCMr and CHr was clearly observed, which was noted within seven days of iron therapy (fig 1). A follow up venous sample taken about four weeks later showed that serum ferritin had increased substantially, together with an increase in haemoglobin concentration (day 36).

No further monitoring was undertaken until a subsequent routine blood sample was taken about two months after iron therapy had commenced (day 75). This showed a sharp decline in ferritin almost to values seen before iron supplementation (fig 1). There was no concomitant decrease in haemoglobin concentration. However, CHCMr and CHr had declined

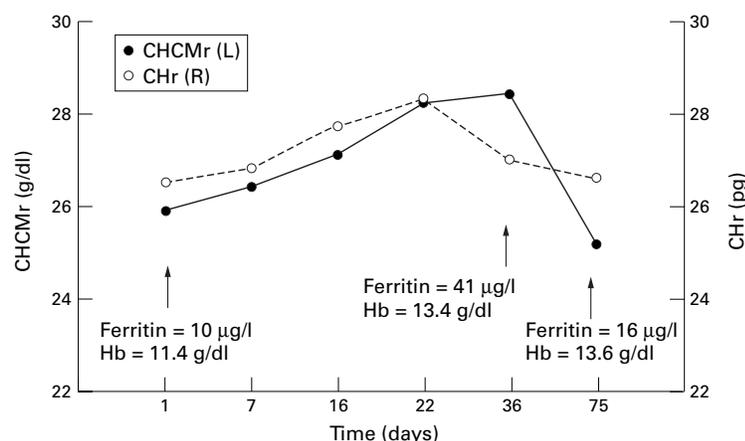


Figure 1 Concentration (CHCMr, left axis) and amount (CHr, right axis) of haemoglobin in reticulocytes of an iron deficient female volleyball player receiving oral iron supplementation. Hb, serum haemoglobin.

to values almost identical with initial values when the subject first presented with iron deficiency anaemia. An interview with the subject revealed that compliance with oral iron therapy had diminished compared with the initial weeks; iron tablets were taken on average only once per week. Unfortunately, a seven week overseas sporting tour prevented any further blood monitoring.

Discussion

Red blood cells have a finite lifespan, and about 1% of the red cell population is removed from the circulation every day.⁷ Virtually all of the iron contained in these senescent cells is recycled and available for subsequent haemoglobin production, which must match the rate of removal to maintain constant haemoglobin levels in the blood.⁷ If, through accelerated iron loss or inadequate dietary iron intake, body iron stores become depleted, haemoglobin production may be impaired. This will lead to a state of iron deficiency anaemia if left untreated.

This case confirms that reticulocyte characteristics in an intensely training athlete with iron deficiency anaemia respond to iron supplementation in a similar manner to those of sedentary subjects and could be utilised as an indication of a positive response to iron therapy. The increase in CHCMr and CHr occurred within seven days of supplementation, significantly reducing the one month interval required to detect a positive response in haemoglobin concentration.⁸

In addition, it provides a valuable illustration of the potential for reticulocyte indices to preempt a decline in haemoglobin concentration during the early stages of iron deficiency. As the

serum ferritin, CHCMr, and CHr values on day 75 were similar to those measured on day 1, it would appear that iron stores had again become depleted to such an extent that normal rates of haemoglobin production could not be maintained. This had not yet become evident from haemoglobin concentration, which showed no decline between days 36 and 75. On the basis of a decline in reticulocyte characteristics, an intervention strategy could be implemented before the early stages of iron depletion develop into iron deficiency anaemia.

Reticulocyte characteristics appear to be a valuable tool for detecting a positive response to iron therapy in intensely training athletes. This inexpensive test, which can be performed on skin prick blood samples,⁹ is also ideal for regular monitoring of athletes considered susceptible to developing iron deficiency anaemia.

- 1 Telford RD, Bunney CJ, Catchpole EA, *et al.* Plasma ferritin concentration and physical work capacity in athletes. *Int J Sport Nutr* 1992;2:335-42.
- 2 Weight LM. 'Sports anaemia' Does it exist? *Sports Med* 1993;16:1-4.
- 3 Borch-Johnsen B. Determination of iron status: brief review of physiological effects on iron measures. *Analyst* 1995;120: 891-3.
- 4 Brugnara C, Laufer MR, Friedman AJ, *et al.* Reticulocyte hemoglobin content (CHr): early indicator of iron deficiency and response to therapy. *Blood* 1994;83:3100-1.
- 5 Buttarello M, Bulian P, Venudo A. Laboratory evaluation of the Miles H³ automated reticulocyte counter. *Arch Pathol Lab Med* 1995;119:1141-8.
- 6 d'Onofrio G, Chirillo R, Zini G, *et al.* Simultaneous measurement of reticulocyte and red blood cell indices in healthy subjects and patients with microcytic and macrocytic anemia. *Blood* 1995;85:818-23.
- 7 Smith JA. Exercise, training and red blood cell turnover. *Sports Med* 1995;19:9-31.
- 8 Oski FA. Iron deficiency in infancy and childhood. *New Engl J Med* 1993;329:190-3.
- 9 Ashenden MJ, Parisotto R, Dobson GP, *et al.* Reticulocyte parameters can be obtained from capillary blood samples. *Aust J Med Sci* 1997;18:78-83.

Commentary

Iron deficiency anaemia is a common condition particularly in women and probably affects 10% of normal women at any one time. It may be more common among athletes especially those whose diet is low in iron and who use non-steroidal anti-inflammatory medicines, which can cause gastrointestinal tract blood loss through a combination of gastric irritation and reduced platelet function.

Diagnosis of iron deficiency anaemia in athletes can be complicated by co-existent sports anaemia which is in fact a dilutional pseudo-anaemia caused by increased plasma volume. Most of the new generation of automated blood cell counters can measure the haemoglobin content and concentration of reticulocytes (immature red cells). As these cells are the latest to be released from the bone marrow, they are the best indication of iron stores within the bone marrow; they will show a reduction in haemoglobin content and concentrations as iron deficiency occurs and an increase in these parameters in response to iron treatment. They are not affected by sports anaemia. Clinical experience with these measurements is accumulating, and they show great promise as early indicators of iron deficiency anaemia and response to treatment.

E J WATTS
Basildon, Essex