Heterotopic ossification is characterised by the periarticular deposition of ectopic bone. It typically occurs after trauma, neurogenic injury, or congenital causes. Idiopathic heterotopic ossification has been rarely reported. A patient who developed idiopathic heterotopic ossification in the intensive care unit without any known predisposing conditions is presented.

Idiopathic heterotopic ossification in the intensive care setting

J E Lane, R J Dean, G D Foulkes, P W Chandler

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CASE REPORT

A 38 year old African-American woman was admitted to hospital with complaints of headaches, sweats, chills, productive cough with dyspnoea, and fever. Her past medical history included hypertension and an uncomplicated childbirth. She denied the use of tobacco and alcohol, and had no allergies. Her medications included ranitidine, sertraline, and atenolol.

Physical examination revealed an acutely ill young woman who was alert and oriented. Vital signs included the following: blood pressure 98/70 mm Hg; pulse 166 beats/min; respiration 28 breaths/min; temperature 39.1°C. Pulmonary examination revealed bilateral crackles throughout all lung fields. Cardiovascular examination demonstrated resting tachycardia without murmurs or bruits. Her abdomen was mildly protuberant with active bowel sounds. Tenderness and hepatomegaly were noted over the hepatic capsular area. Neurological examination was normal.

Laboratory evaluation disclosed the following (reference range in parentheses): total creatine phosphokinase 1531 U/l (26–140 U/l); prothrombin time 12.9 sec (10.9–13.1 sec); partial prothrombin time 36 sec (25–35 sec); serum lactate dehydrogenase 882 U/l (208–378 U/l). Haematological analysis revealed a white blood count of 8300 × 10⁹/l (4.5–11.0 × 10⁹/l) with 92% blasts, a platelet count of 87 × 10⁹/l (150–450 × 10⁹/l), and a haemoglobin of 96 g/l (120–160 g/l). Serum potassium was decreased to 2.7 mmol/l (3.5–5.1 mmol/l). Arterial blood gas analysis included the following: pH 7.52; carbon dioxide pressure 4.0 kPa; oxygen pressure 5.9 kPa (room air). A chest radiograph revealed changes consistent with bilateral interstitial pneumonia.

The patient was transferred to the intensive care unit for progressive hypoaemic respiratory failure and placed on mechanical ventilation. She required prolonged ventilatory support secondary to acute respiratory distress syndrome and bilateral pneumonitis. She continued to remain febrile and neutropenic. Transoesophageal echocardiography demonstrated a broad based sessile vegetation on the mitral valve suspicious of bacterial endocarditis. A diagnosis of leukaemia was made from the peripheral smear. She was treated with a number of antimicrobial agents, including vancomycin, amphotericin, and ciprofloxacin, for presumed endocarditis.

Her platelet count continued to decrease and she developed joint stiffness in her elbows and knees. She developed decreased range of motion and pain with subsequent diffuse muscle wasting of the upper extremities. Heterotopic ossification of the periarticular regions at knee and elbow joints (figs 1 and 2). Alkaline phosphatase reached a peak of 903 U/l (42–98 U/l).

DISCUSSION

Idiopathic heterotopic ossification has been rarely reported. Heterotopic ossification is typically categorised as due to

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trauma, neurogenic injury, or myositis ossificans progressiva. This patient developed fulminant heterotopic ossification without any of these inciting causes.

While the pathophysiology of heterotopic ossification is unknown, the underlying defect involves extraneous fibroblastic differentiation into osteoblasts, typically occurring as a result of an initial inflammatory lesion. A systemic factor is likely to be involved in the pathogenesis of heterotopic ossification. Cope discussed potential pathophysiological inducers of heterotopic ossification, including bone morphogenetic protein, electrophysical lines, and abnormal intrinsic magnetic or electrical forces. Bone morphogenetic protein can function to induce undifferentiated mesenchymal cells to form osteoblasts. Recently there have been several reports of heterotopic ossification after neuromuscular blockade. While our patient was not chemically paralysed, she was immobile for an extended period. The lack of any known inciting cause in our patient complicates the understanding of pathophysiological cause. In our case, we suspect that multiorgan dysfunction syndrome, chronic immobilisation, and hypoxaemia were involved in triggering a systemic factor.

The diagnosis of heterotopic ossification is a clinical challenge that is often delayed secondary to patient immobilisation, complications with ventilatory support, approximating range of motion in critically ill patients, inconclusive laboratory analysis, and delayed radiographic findings. Diffuse idiopathic skeletal hyperostosis (DISH) is a common disorder that must be considered within the differential diagnosis of such presentation. DISH is manifested by back pain and spinal stiffness and is most common in the thoracic spine. It is important for physicians to be aware of heterotopic ossification, especially for those not accustomed to its non-specific and delayed clinical presentation. It is helpful for the clinician to recognise this to differentiate heterotopic ossification from other processes that can occur in patients with prolonged ventilation such as critical care polyneuropathy and diffuse idiopathic skeletal hyperostosis.

**Summary points**

- Heterotopic ossification is characterised by periarticular deposition of ectopic bone.
- Heterotopic ossification typically occurs after trauma, neurogenic injury, or congenital causes.
- The pathophysiology is unknown.
- Common signs and symptoms include decreased range of motion, pain, swelling, and erythema.
- Laboratory findings are non-specific and may not aid in early diagnosis.

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**Figure 1** Sheet-like heterotopic ossification of the knee.

**Figure 2** Heterotopic ossification of the elbow joint.
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