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Letter to the Editor

An unusual case of shock in a young woman

Sir,

Lactic acidosis in patients with leukaemia or lymphoma is well known but in association with solid tumours it is rare and has seldom been described in association with breast cancer.[1-3] We report a case in a young woman with metastatic breast cancer, presenting with shock which rapidly progressed to death.

A 31 year old woman was admitted having been mildly unwell for 4 days with subsequent progressive dyspnoea for 2 days. There was no recent febrile illness, no other cardiac or respiratory symptoms or history, apart from a long history of moderate smoking. Six months previously a breast lump had been noted, investigation showing an invasive ductal adenocarcinoma. Bone scan, chest X-ray and liver function tests were all normal. A right Patey mastectomy was performed in August 1987. Post-operatively radiotherapy was given.

On examination she was well-nourished, shocked, apyrexial, tachypnoeic and breathless but not cyanosed. Her pulse rate was 120 per minute and blood pressure was unrecordable. The liver was 4 finger breadths enlarged. A central venous pressure line was inserted and as the venous pressure was very low, gelofusine was infused raising her blood pressure to 95/65 mmHg with a simultaneous filling pressure of 12 cm H_20. Investigations revealed a metabolic acidosis with an increased anion gap, high serum lactate (16 mmol/l), normal Pao_2, glucose, renal function, clotting parameters, full blood count, chest X-ray and electrocardiogram. No salicylate or paracetamol was detected in the serum. On further questioning she denied any overdose, recent excessive alcohol intake and had not used a tampon for several weeks. There were no stigmata of toxic shock syndrome. The differential diagnosis was between septic shock (including toxic shock syndrome) and a type B lactic acidosis. Treatment was with broad-spectrum antibiotics, Parentrovite, colloids, inotropic support and controlled bicarbonate therapy. Gynaecological investigation did not suggest uterine sepsis or perforation. After an initial improvement, she deteriorated rapidly, became hypoxaemic, and despite assisted ventilation, died approximately 8 hours after admission.

Blood cultures were negative. Her liver function tests were abnormal: raised bilirubin, transaminases and alkaline phosphatase, markedly raised lactate dehydrogenase, but normal albumin. At post-mortem, metastatic breast cancer involved several para-aortic lymph nodes, two lower thoracic vertebrae and the liver, with very extensive replacement of the latter. The lungs were congested but otherwise the respiratory and cardiovascular systems were normal. Multiple sections through the uterus showed the coil *in situ* and no evidence of infection.

Recent review articles[4,5] have emphasized the crucial role of lactate disposal mechanisms, particularly hepatic, in maintaining lactate homeostasis, and this is reflected in the fact that whilst a healthy 70 kg man produces 1200–1500 mmol of lactate per day, the normal serum lactate is around 1 mmol/l. Lactate and equivalent amounts of H^+ are utilized in the Krebs cycle and gluconeogenesis in the liver (60%) and kidneys (30%).[6] Many patients with type B lactic acidosis have underlying liver impairment, although increased lactate production may be a precipitating cause. In all the recorded cases associated with solid tumours, there have been hepatic metastases with significant liver impairment, indicating that impaired hepatic clearance is a major factor, although tumour cell production of lactate may contribute. In this patient it is thought that the shock was due to the type B lactic acidosis itself subsequent to hepatic impairment, since there was no evidence of any other cause of shock.

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