

Letters to the Editor

The role of malignancy in lactic acidosis and shock

Sir,

In their paper on an unusual case of shock in a young woman, McConnell *et al.*¹ described the development of lactic acidosis in a shocked patient who had previously had a right Patey mastectomy for invasive ductal adenocarcinoma. We do not agree with the authors' opinion that the lactic acidosis was of type B, precipitated by significant liver impairment, and that the shock was due to the type B lactic acidosis.

Although the patient presented with elevated levels of bilirubin, transaminases, alkaline phosphatase and lactate dehydrogenase, only the lactate dehydrogenase was markedly raised, and clotting parameters and albumin level were normal, indicating that the liver dysfunction was not so severe as to impair lactate metabolism, and to produce so high a blood lactic acid level (16 mmol/l). Moreover the patient was shocked at presentation and in this situation it is more reasonable to assume that the lactic acidosis was mainly of type A due to the shock state and inadequate tissue oxygenation, but not the opposite as the authors thought.

As regards the cause of the shock, the patient had advanced metastatic cancer, and it is well recognized that in malignancies the macrophages release immune hormone (cachectin) also known as tumour necrosis factor^{2–4} which is responsible for a variety of serious disorders including shock, lactic acidosis and adult respiratory distress syndrome, where death occurs by abrupt respiratory muscle failure and apnoea.^{5–7}

In the reported case there was no evidence of infection and the patient died with shock accompanied with respiratory failure and apnoea due, most probably, to shock lung syndrome. We believe that cachectin was the mediator.

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This letter has been shown to Drs McConnell, Parfitt and Walker who reply as follows:

Thank you for the opportunity to reply to Drs Odeh and Bassan. The vast majority of patients who die with cachexia due to cancer do not show the usual features of lactic acidosis. It has been reported that one of the biochemical changes of the premortal state is an increase in lactate levels,¹ but Kirk ascribes this to hypoxia, based on the work of Fabricius Hansen.² The patient that we described was not hypoxic on admission, despite being shocked, and she became clinically hypoxic and required ventilatory support only 2 hours before death, that is, the lactic acidosis was established before any hypoxia occurred.

In the other sources which we quoted which described patients with solid tumours who developed lactic acidosis, all the patients had hepatic metastases. As we mentioned in our letter it is probable that the tumour tissue may have contributed to the lactate load, but we still consider that the critical factor was the liver's capacity to deal with the normal daily production of lactate. Such a decrease in hepatic function may occur without necessarily resulting in a low serum albumin or impairment of the routinely measured clotting parameters, especially when it is remembered that the entire course of the acute illness lasted only about 10 hours. Post-mortem revealed very extensive replacement of the liver by metastatic tumour tissue.

The differences in interpretation of this case are probably an indication of the amount still to be discovered about the pathogenesis of lactic acidosis.

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Salmonella endocarditis

Sir,

We read with interest the report by Echevarria and colleagues¹ of a case in which *Salmonella virchow* infected a left ventricular aneurysm. We wish to report a similar case in which the infective organism was *S. panama*.