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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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References

1. McConnell, A.A., Parfitt, V.J. & Walker, P.R. An unusual case of shock in a young woman. *Postgrad Med J* 1989, **65:** 120.
2. Oliff, A. The role of tumour necrosis factor (cachectin) in cachexia. *Cell* 1988, **54:** 141-142.
3. Mahony, S.M., Beck, S.A. & Tisale, M.J. Comparison of weight loss induced by recombinant tumour necrosis factor with that produced by a cachexia - inducing tumour. *Br J Cancer* 1988, **57:** 385-389.
4. Tracey, K.J., Lowry, S.F. & Cerami, A. Cachectin: a hormone that triggers acute shock and chronic cachexia. *J Infect Dis* 1988, **157:** 413-420.
5. Tracey, K.J., Beutler, B., Lowry, S.F. *et al.* Shock and tissue injury induced by recombinant human cachectin. *Science* 1986, **234:** 470-474.
6. Tracey, K.J., Lowry, S.F. & Cerami, A. Cachectin/TNF-α in septic shock and septic adult respiratory distress syndrome. *Ann Rev Respir Dis* 1988, **138:** 1377-1379.
7. Beutler, B. & Cerami, A. Cachectin, cachexia, and shock. *Ann Rev Med* 1988, **39:** 75-83.

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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References

1. Kirk, J.E. Premortal clinical biochemical changes. *Adv Clin Chem* 1968, **11:** 175-212.
2. Fabricius Hansen, I. *Investigations on Agonal Acidosis* P. Branner, Copenhagen, 1948. (Danish edition, P. Branner, Copenhagen, 1947.)

Salmonella endocarditis

Sir,

We read with interest the report by Echevarria and colleagues[1] 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.*

A 62 year old man presented with fever and diarrhoea. He had a history of partial gastrectomy for duodenal ulceration and had had anterior myocardial infarction 4 years previously. Stool and blood cultures grew *S. panama*. He was treated with intravenous ampicillin and chloramphenicol for 2 weeks. He relapsed one week after discharge and *S. panama* was again isolated from blood cultures. He was treated with oral ciprofloxacin for one week and again discharged. Three weeks later he was re-admitted with a transient right hemiparesis. He had fever, splinter haemorrhages, a dyskinetic apex beat and a variable systolic murmur. A two dimensional echocardiogram showed an apical left ventricular aneurysm containing thrombus. He was treated with intravenous ciprofloxacin 2.25 g daily, but was referred for cardiac surgery because of persistent fever, embolic phenomena and cardiac tamponade. An abscess was found at the base of the aneurysm. The thrombus was removed and the aneurysm was resected. He was discharged after a further 2 weeks of antibiotic therapy.

Infection of ventricular mural thrombus has rarely been reported.[1-3] Although there are reports of such infection with *Salmonella* species, we believe that one due to *S. panama* has not been described previously. In this case, the previous gastrectomy may have reduced the host defences against ingested salmonella.[4,5]

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References

1. Echevarria, S., Arjona, R., Alonso, J., Riancho, J.A., Revuetta, J.M. & Macias, J.G. False aneurysm formation after *Salmonella virchow* infection of a pre-existent ventricular aneurysm-survival after surgical resection. *Postgrad Med J* 1989, **65:** 168–170.
2. Schofield, P.M., Rahman, A.N., Ellis, M.E., Dunbar, E.M., Bray, C.L. & Brookes, N. Infection of cardiac mural thrombus associated with left ventricular aneurysm. *Eur Heart J* 1986, **7:** 1077–1082.
3. Catherwood, E., Mintz, G., Kotler, M., Kimbris, D., Lemmon, W. & Parry, W. Pseudoaneurysm of the left ventricle complicated by *Salmonella typhimurium*. *Am J Med* 1980, **68:** 782–786.
4. Waddell, W.R. & Kunz, L.J. Association of salmonella enteritis with operations on the stomach. *N Engl J Med* 1956, **255:** 555–559.
5. Hook, E.W. Salmonellosis: certain factors influencing the interation of salmonella and the human host. *Bull NY Acad Med* 1961, **37:** 499–512.

Listeria monocytogenes meningitis in previously healthy adults

Sir,

We read with interest the paper by Hearmon and Ghosh[1] but wish to discuss several points made therein.

Firstly, there is no convincing evidence that *Listeria monocytogenes* may be transmitted venereally. The isolates incriminated by Rappaport *et al.*[2] were subsequently identified as *Lactobacillus* spp. (Seeliger, 1988, oral communication), and the association between *Listeria monocytogenes* and recurrent abortion has yet to be conclusively demonstrated.[3] In addition, we wish to emphasize that most cases of neonatal listeriosis are of the so-called 'early onset' type, i.e. presenting within the first 5 days of life. This form of the disease, manifesting as septicaemia and multi-organ sepsis, often following a maternal flu-like illness, is frequently associated with chorio-amnionitis and almost certainly results from haematogenous spread from the mother rather than ascending infection from the vagina or cervix.[4]

We agree with Hearmon and Ghosh that listeriosis is not confined to the classical at-risk groups. Indeed in the outbreak in Canton de Vaud, in which soft cheese was the vehicle of transmission, 60% of the adult cases had no identifiable risk factor.[5]

The incidence of listeriosis in the UK is commoner in males in all age groups except in patients aged 75 years or older.[6] We disagree with Hearmon and Ghosh's hypothesis that this excess is due to partial immunity as a result of vaginal carriage in the female. Higher carriage rates in the vagina compared to those in the male urethra are likely to result from the proximity of the female genital tract to the anus. The prevalence of faecal carriage does not vary significantly between the sexes.[7]

We agree that listeria meningitis is difficult to diagnose but several clinical features may suggest the diagnosis.[8] The illness may have a subacute onset with fluctuating levels of consciousness and nuchal rigidity may be absent in up to 20% of adult patients. In addition, movement disorders such as ataxia, myoclonus and tremors are more common in listerial meningitis than in the classical pyogenic meningitides. It should be noted that hypoglycorrachia is not a consistent finding.[7]

Finally, we wish to emphasize that all apparently sporadic cases of listeriosis should be investigated promptly and vigorously. A detailed food history should be elicited and comprehensive sampling of all foods in the domestic refrigerator and 'high risk' foods from other locations, e.g. raw seafood from deep freezers, vegetables etc. should be undertaken without delay.

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References

1. Hearmon, C.J & Ghosh, S.K. *Listeria monocytogenes* meningitis in previously healthy adults. *Postgrad Med J* 1989, **65:** 74–78.
2. Rappaport, F., Rabinovitz, M., Toaff, R. & Krochik, N. Genital listeriosis as a cause of repeated abortion. *Lancet* 1960, **i:** 1273–1275.
3. Dick, J.P.R., Palframann, A.R. & Hamilton, D.V. Listeriosis and recurrent abortion in a renal transplant patient. *J Infect* 1988, **16:** 273–277.
4. Becroft, D.M.O., Farmer, K., Seddon, R.J. *et al.* Epidemic listeriosis in the newborn. *Br Med J* 1971, **3:** 747–751.
5. Bille, J. Epidemiology of human listeriosis in Europe. Presented at the *Comprehensive Conference on Listeria monocytogenes* 1988, Rohnert Park, California, USA.