The level of prolongation of the prothrombin time at which a contemplated percutaneous biopsy should be abandoned in favour of a transjugular or plugged procedure has likewise not been defined. It would be helpful if future audits or reviews aimed to clarify this point. A prospective study of the efficacy and duration of action of fresh frozen plasma is underway at the Queen Elizabeth Hospital.

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Increased CA 125 in tuberculous peritonitis

EDITOR,—O’Riordan et al reported the case of a patient with tuberculous peritonitis, ascites, and pleural effusion also who had a remarkably increased concentration of serum CA 125 (Gut 1995; 36: 303–5). Because of the increase in this tumour marker an ovarian cancer was suspected and a laparotomy was performed with a negative result for cancer. The authors consider the increased values of CA 125 the most interesting aspect of the case, and review other published reports on increased CA 125 values in patients with tuberculous ascites.

I believe, however, that the only interesting and noticeable aspect of the case would have been if the patient had had a normal value of CA 125. This tumour marker, commonly used in the diagnosis and follow up of ovarian cancer, increases in a variety of processes including pleura, pericardium, and peritoneum including endometriosis, peritonitis, pelvic inflammatory disease, and surgical trauma.1,2 The increase in CA 125 is not unexpected because this antigen has been detected on mesothelial cells in pleura, pericardium, and peritoneum, particularly in areas of inflammation.

Benign inflammatory effusions, particularly ascites, are associated with increased serum concentrations of CA 125 with values up to 100 times the upper normal limit in some cases.3,4 CA 125 has proved to be an excellent marker of non-malignant conditions involving liver diseases (sensitivity 98-4%, specificity 95-9%, efficiency 96-9%).6 This marker is also very sensitive to minimal amounts of ascites and correlates very well with the amount of ascitic fluid.7 Thus, the specificity of CA 125 for ovarian cancer is very low in the presence of ascites of whatever origin.

In addition, the return of CA 125 to normal after anti-tuberculous treatment in the patient of O’Riordan et al is not unexpected. CA 125 decreases to normal values when the ascites is removed in cirrhotic patients and increases again when ascites recur.9

The authors conclude that 'tuberculosis needs to be considered in the differential diagnosis of ascites with increased tumour markers'. It is well known, however, that CA 125 is an unspecific marker of ascites of whatever aetiology. Tuberculosis is a disease that only uncommonly produces peritoneal effusions, represents only one of the many aetiologies of ascites. Finally, I believe that a laparotomy should not have been performed in this case because both computed tomography and cytological study of ascitic fluid were negative for ovarian cancer and the very high concentration of CA 125 could have been easily explained by the existence of both ascites and pleural fluid.

Reply

EDITOR,—Dr Collazos’ points concerning CA 125 are valid. He is, however, an expert in the area and has published widely on CA 125. We felt this case to be of interest to a general audience because they might not have been familiar with the wide range of different cancers that can increase CA 125. The concentrations of CA 125 are high in many conditions as outlined in Dr Collazos letter. We mentioned all of these and more in our discussion. Dr Collazos does not mention how he would have made the diagnosis. He might have legitimately requested laparoscopy in place of laparotomy. He suggests neither. We believe he overstates the value of negative computed tomography and non-diagnostic ascitic fluid cytology in excluding intra-abdominal tumour, however. Such measures can only reduce the relative risk that a tumour is present.

One additional point of some interest is that despite our considerable experience with immunocytochemistry and CA 125 in cytological preparations, we were unable to identify positive staining in the mesothelial cell population in the ascitic fluid specimen in retrospect. Had the immuno-panel been performed during the case this too would have urged us towards more invasive investigations.

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Adenomas and a family history of colorectal cancer

EDITOR,—The data presented by Gaglia et al (Gut 1995; 36: 385–90) imply that adenomas in hereditary non-polyposis colorectal cancer (HNPPC) occur with increased frequency, show a predilection for the proximal bowel and are more likely to be invasive compared with sporadic colorectal cancer. It is important to note that these data are at complete variance with our own.1 We have suggested that the high risk of colorectal cancer in HNPPC is not explained by the increased initiation of

Letter

Letters

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Reply

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