LETTERS TO THE EDITOR

Dear Editor,

Role of endoscopic ultrasound in the management of pancreatic lesions

We would like to congratulate Professor Brian Jones on his recent article on the role of endoscopic ultrasound (EUS) in staging upper gastrointestinal cancers.[1] We completely agree that EUS has become an indispensable tool in the management of pancreatic cystic lesions. However, the role of EUS in the management of pancreatic adenocarcinoma is more controversial.

Modern multislicer computed tomography (CT) scans with maximal intensity projections (MIPS) and volume rendering can provide accurate assessment of the pancreatic head lesions and their relationship to surrounding structures. CT is better at detecting vascular invasion than EUS.[2] Complete loss of fat cuff around the superior mesenteric artery or portal vein/superior mesenteric vein occlusion would render the patient inoperable on the basis of locally advanced tumour. If a pancreatic tumour is touching or indenting portal or superior mesenteric vein without occluding it, most pancreatic surgeons would offer en bloc vascular resection or at least a trial of dissection. There is sufficient evidence that in this scenario portal vein/superior mesenteric vein invasion is mark of tumour location rather than any particular predisposition to vascular invasion. This has been shown by both histopathological[3] as well as survival[4] studies in pancreatoduodenectomy patients.

Endoscopic ultrasound and multislice CT have shown similar sensitivities and specificities with regard to detection of most pancreatic lesions, but when pancreatic lesions are less than 2 cm EUS has a more superior detection capability.[5] EUS is extremely useful at identifying lesions in patients with increased plasma Ca 19-9 levels and pancreatic duct dilatation, where a CT scan has failed to localize a lesion. Our own experience of high-volume EUS (>600/year) combined with multislice CT as part of our staging protocol has suggested that one method alone is not sufficient to indicate non-resectability. Our own practice is that if only one method indicates locally non-resectable tumour we will still proceed to a trial of dissection. Should they both indicate non-resectability then they are invariably correct. Lesions of the uncinate process and lymph node assessment offers further difficulties. The uncinate process is often difficult to assess endoscopically and can be better assessed by a CT scan. Similarly, lymph nodes may be enlarged by inflammatory factors rather than tumour involvement and great caution must be taken with interpretation of nodes seen on EUS unless a needle biopsy is taken.

Most patients with painless obstructive jaundice, increased tumour markers (normal serum immunoglobulin G4) and accompanying mass (on a multislicer triple phase CT scan with MIPS) have pancreatic adenocarcinoma and should be treated as such until proved otherwise. The role of fine-needle aspiration (FNA) in these patients has to be carefully considered, as a negative result would not change the surgical management. The need for FNA should be assessed on a patient-to-patient basis as FNA can potentially lead to pancreatitis, haemorrhage and sepsis. There also remains the risk of tumour seeding along the needle tract in potentially curable patients if the needle tract is not included in the resected specimen. However, FNA can be instrumental in diagnosing pancreatic lipoma, lymphoma, tuberculosis, sarcoidosis and inflammatory pseudo-tumours. FNA and obtaining diagnostic tissue remains vitally important if the patient is to be offered chemotherapy for locally advanced non-resectable tumours and offers potential in preoperative assessment of biomarkers of prognosis and response to therapy. In addition, failure to make a tissue diagnosis of cancer can lead to complications and death related to metal biliary stents in the long term for those patients who do not have cancer and it excludes them from participation in clinical trials of novel therapies. For these reasons, FNA biopsy diagnosis is almost more important in those patients that are not operative candidates than those that are. We agree with Professor Jones that in pancreatic cancer distant lymph node involvement is a poor prognostic marker. However, we disagree with the suggested algorithm (figure 8) that the patients with stage II disease (T3, N0, T1, N1, T2, N1, T3N1) should be palliated. Eighty per cent of the patients undergoing pancreatoduodenectomy would have positive lymph nodes.[6] Five-year survival rate in patients with positive lymph nodes can be between 10 and 20%.[6,7] Japanese studies have shown that long-term prognosis of these patients depends on the tier of lymph node involvement[7] and more recently this has been reaffirmed by studies analysing the ratio of number of positive lymph nodes to number of nodes examined.[8]

In most high-volume surgical centres (more than 20 cases per annum), the rate of mortality associated with pancreatoduodenectomy is less than 1%.[9] In this setting, patients with stage II disease should be offered pancreatoduodenectomy. We are all aware that surgery is not panacea for pancreatic cancer, but currently it remains the only option for cure in appropriately selected and staged patients. We feel that ultimately, salvation for these pancreatic cancer sufferers lies in research leading to prevention and early diagnosis.

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JASWINDER S. SAMRA,* D Phil, FRACS
ANDREW V. BIANKIN,†‡ PhD, FRACS
NEIL D. MERRETT,‡ FRACS
**Upper Gastrointestinal Surgical Unit, University of Sydney, Royal North Shore Hospital*, †*Garvan Institute, and* ‡*Bankstown Hospital, Sydney, New South Wales, Australia*

doi: 10.1111/j.1445-2197.2008.04447.x