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Letters and comments

Contributors to this section are asked to make their comments brief and to the point. Letters should comply with the Notice printed on the inside back cover. Tables and figures should only be included if absolutely essential and no more than five references should be given. The Editor reserves the right to shorten letters and to subedit contributions to ensure clarity.

Response to paper by A James Eccersley et al.

Referral guidelines for colorectal cancer – do they work?

Ann R Coll Surg Engl 2003; **85**: 107–10

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We also recognised the importance of investigating the impact of the fast track system for suspected colorectal cancer. In addition, we felt it necessary to assess the pattern of colorectal cancer referrals made to our department.

Over 12 months, we received 185 fast track referrals – similar in number to your published data; however, only 15% of these patients were diagnosed with colorectal cancer. Our department during this same 12-month period managed 95 cases of colorectal cancer of which 68% were GP referrals; only 47% of these were fast track referrals the remaining 53% had been referred as routine. In-house referrals from other specialties including gynaecology, gastroenterology and care of the elderly accounted for 22%. The remaining 5% had presented as emergencies Only one-third of colorectal cancers appear to be coming through the fast track system with a further one-third being referred as routine and a fifth going to non-surgical specialties. This would indicate that the majority of cancers are not fitting the guidelines for urgent referral leading to a delay in presentation due to inappropriate referral as non-surgical or routine. In an attempt to meet the 2-week deadline, are routine referrals now facing an increased waiting time for first appointment?[1,2] With 30% of cancers being referred in this way, is this 2-week rule actually affecting prognosis adversely?

Rational for the introduction of the 2-week rule was that early diagnosis improved survival. One explanation for the high mortality in the UK was the more advanced disease seen in the patients at presentation as compared to other countries. At present, the fast track system does not appear to be fulfilling its intended role.

GP support and public awareness certainly need to be addressed. We may also gain more through introduction of an effective screening programme and improving the management of patients already diagnosed with the disease.[3]

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Letter to the Editor

When surgeons became SARS patients

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Two cardiothoracic surgeons (IYPW and SW) contracted severe acute respiratory syndrome (SARS) at the Prince of Wales Hospital, Hong Kong, in early March 2003.[1,2] Both surgeons developed fever and watery diarrhoea after contact with the index SARS patient. Although there was no respiratory symptom initially, their oxygen saturation dropped significantly within the

next few days, with increased respiratory distress and persistent high fever. Chest roentgenography and computed tomography showed features of consolidation similar to bronchiolitis obliterans organising pneumonia. Both surgeons were transferred to an intensive care unit (ICU). Blood tests showed elevated creatinine kinase, lactate dehydrogenase and alanine aminotransferase. Elevation of these enzymes was shown to be associated with higher rate of ICU admission and death.[1] Intravenous ribavirin and methylprednisolone were given as described.[1] Their respiratory functions slowly improved and they were eventually discharged from ICU. Although still dependent on nasal oxygen supplement, active rehabilitation was started on the isolation ward and they were discharged home 21 and 27 days after the onset of illness, respectively.

Comment

The index patient with SARS was admitted to ward 8A of Prince of Wales Hospital in early March. Within the next week, 18 healthcare workers and 16 medical students developed fever, chills and malaise.[1] The use of a nebulised bronchodilator for this index patient, with the increase in droplet load, was believed to be the route by which the disease spread.[2] It has been reported that all 156 patients with the diagnosis of SARS in late March were traceable to this index case.[1]

It has always been difficult for surgeons and physicians to accept the sick role. Most doctors are reluctant to abandon the role of doctor for patient.[3] The care-providers also experienced difficulties when treating a doctor-patient as negative counter-transference reactions and feelings of inferior professionalism are possible obstacles to effective treatment.[4] Doctor-patients should have trust in their physicians in order to achieve maximal therapeutic outcome. We observed the high spirits and morale of our intensive care and medical colleagues in facing this novel and lethal disease. Many of our colleagues volunteered to work in the SARS wards and they have to stay within the hospital compound for more than a month without going home; their selflessness was widely appreciated.

Our surgical practice has to be modified as we are now facing a new and highly contagious disease. The clinical presentations of SARS could be diverse and non-specific especially in old patients.[1] A high degree of awareness and adequate self-protection should be adopted while attending new patients. Strict infection-control measures have to be followed while performing endotracheal intubation, tracheostomy and bronchoscopy as they are now classified as high-risk procedures during the SARS epidemic. Some patients developed spontaneous pneumothorax during the phase of pulmonary destruction and required tube thoracostomy.[5] Hopefully, with earlier diagnosis and treatment, the risk of spreading the disease can be minimised and a better therapeutic outcome will be achieved.

In short, SARS is a new disease entity with a novel virus being identified to be the culprit.[6] The natural history of the disease is not well documented and the period of virus shedding after recovery remains undetermined.[2] The daily practice of individual doctors and, probably more important, the whole healthcare system have to be improved in order to cope with the outbreak of this new disease. The social and economical impact is expected to be significant in the long term.

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Response to paper by C Kouriefs & NA Watkin

What to do if it gets 'bigger'

Ann R Coll Surg Engl 2003; **85**: 126–8

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We were interested to read this review of intra-operative penile tumescence. Those frustrated by this clinical problem may be interested to know of our own approach to this situation, which was published

contiguously in another journal.[1] Although not a common occurrence, intra-operative penile tumescence during endoscopic surgery is a troublesome challenge to the urologist. We feel that the safest and simplest solution is to utilise freely available dental lignocaine and adrenaline. This is administered as an intracavernosal injection of lignocaine 2% and epinephrine 1:80,000 (0.0125 mg/ml) using a standard dental syringe and cartridge. This simple, safe, inexpensive and convenient procedure reliably induces detumescence using a handy prepackaged system that is readily available for immediate use. It avoids the potential overdose from calculating complex dilutions as well as time delay and is easily used during both spinal and general anaesthetics. A second dose can be given safely and the senior author has used this technique for more than 5 years on numerous occasions and has termed it 'a urological cold shower'.

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Letter to the Editor

Erosive adenomatosis of the nipple – a report of three cases

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We report three cases of erosive adenomatosis of the nipple in women of different age groups. Erosive adenomatosis of the nipple (EAN) is a rare, benign neoplasm of breast lactiferous ducts. Peak incidence is in the fifth decade in women, although it has been described rarely in children.[1] The condition was first described in 1955 by Jones[2] who termed the condition 'florid papillomatosis of the nipple'. Although uncommon, it is an important condition to recognise to avoid a misdiagnosis of malignancy and the consequent unnecessary surgery.

The first case was a 65-year-old woman who presented with a short 2–3 week history of tender nodularity in the left breast associated with some nipple erythema. Initial examination revealed a small 0.5 cm nodule near the left nipple with mild surrounding erythema. Breast examination bilaterally was otherwise normal and clinic ultrasonography and mammography unremarkable. A clinical diagnosis of a benign skin nodule was made and the patient was re-assessed 6 weeks later when a residual 0.5 cm left nipple ulcer was noted. A diagnostic wedge biopsy was then performed and demonstrated an erosive adenoma of the nipple. There was no invasive carcinoma present. The patient was managed by wide excision alone including nipple sacrifice. Follow-up at 1 year revealed no evidence of local recurrence. The second case was a 36-year-old woman who presented with a 4-month history of a rash involving the right nipple, associated with some breast discomfort. Clinical examination demonstrated an erythematous area involving the nipple with an area of tender nodularity in the inferior breast. Breast examination was unremarkable. Initial investigations included clinic ultrasonography and bilateral mammography and were both normal. A clinical diagnosis of an eczematous skin nodule was made but incisional biopsy followed by local excision with preservation of the nipple ducts demonstrated an erosive adenoma of the nipple that was completely excised (Fig. 1). The third case was a 37-year-old patient who presented with a 6-week history of bleeding from the right nipple which had been cracked for several years following breast feeding her first child. A nipple smear for cytology was taken which showed inflammatory cells only. A wedge biopsy of the nipple was taken under local anaesthetic which revealed erosive adenomatosis with no evidence of Paget's disease. The patient was offered

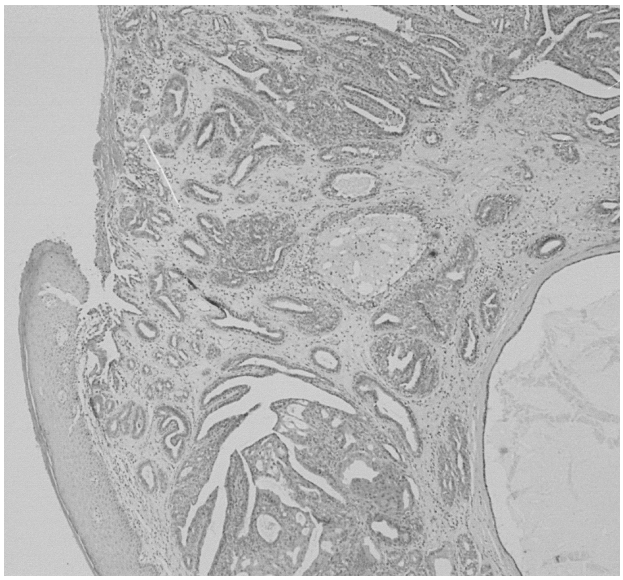

Figure 1 Erosive adenomatosis of the nipple (x200).

nipple excision as a curative procedure and the specimen analysis confirmed an erosive adenoma with complete excision.

These three cases highlight the clinical diagnostic difficulty in EAN. Clinically, it may be mistaken for Paget's disease and histologically it can be mistaken for carcinoma. EAN generally presents clinically with a unilateral erythematous crusting lesion with hardening of the nipple. Ulceration may or may not be present. The condition most commonly affects women but has been reported in men.[3] The mean age of diagnosis of EAN is 45 years.[4] Clinical assessment should include clinical examination and appropriate imaging depending on the age of the patient to exclude an underlying infiltrating carcinoma. Punch or wedge biopsy can be used although excisional biopsy avoids the need for multiple procedures. Histological diagnosis can be difficult as there can be a variety of histological appearances. All forms of this lesion are thought to compose of two apparent cell types – epithelial luminal cells and basal myo-epithelial cells. A number of immunohistochemical reagents have been employed to aid in diagnosis including carcinoembyronic antigen (CEA), vimentin (VIM), glial fibrillary acidic protein (GFAP) and muscle specific actin (MSA).[5] Although immunohistological evaluation can aid in differential diagnosis between EAN and invasive carcinoma of the breast, it may not be capable of distinguishing between EAN and other benign proliferative mammary lesions. Clinical management of these lesions should be by complete excision. Since it is a benign condition, follow-up and surveillance should be within the NHS breast screening programme guidelines.

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Response to paper by JWF Catto & DJ Alexander

Pancreatic debridement in a district general hospital – viable or vulnerable?

Ann R Coll Surg Engl 2002; **84**: 309–13.

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Whilst I agree with Catto and Alexander that good results may be achieved in a given subspecialty field in a district general hospital (DGH) when comprehensive management is provided by an interested, trained and skilled surgeon and quality support from anaesthetic, radiological and nursing services, the reader was given a rather restricted snapshot of the management of severe acute pancreatitis in a DGH and, therefore, remains sceptical of the authors' conclusions.

The authors' described a radical approach to pancreatic debridement in 12 patients that included splenectomy (9 patients) and bowel resection (5 patients) with a favourable mortality rate of 25%. It is not clear, however, what the outcome (mortality rate) of other patients with severe acute necrotising pancreatitis who did not undergo surgery has been during the same period, as it is possible that the surgeon's bias towards patient selection has influenced results. In their literature review, the authors referred to some published series with mortality rate of 6–10% from acute pancreatitis but did not provide their own results. In their conclusion, the authors described a '30-day' mortality (though most current series refer to 'in-hospital' mortality) of 2.5% for resection of carcinoma and chronic pancreatitis over a 4-year period, but provided data on pancreatic debridement over a selected 2-year period only.

Furthermore, the authors failed to provide convincing arguments to support their 'radical' approach to surgery. Their approach did not seem to shorten hospital stay or reduce cost of hospital treatment compared with the other discussed series. It was carried out at the expense of a relatively high rate of bowel resection, which may reflect collateral mesenteric vascular injury from aggressive surgery. Though not clinically evident from this small series, I am rather concerned about the liberal addition of splenectomy with its recognised immune depletive effects,[1] particularly in patients with severe acute necrotising pancreatitis who have established deficiency of cellular immunity[2,3] in the face of serious intra-abdominal sepsis. In addition, the authors referred to the previously cited argument against extensive pancreatic

debridement of higher rates of diabetes, but failed to provide data on postsurgical endocrine deficiency from their own series.

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Response on behalf of the authors by

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Regrettably, our original paper was lost during the editing process, having originally been submitted in July 1999, although we were delighted with its eventual publication. We have presented our 5-year experience at the Newcastle Meeting of the Association of Upper GI Surgeons (September 2000). This detailed a 12% 30-day mortality (hospital mortality, 23%) for pancreatic necrosectomy (17 patients) and a 2.2% 30-day mortality (hospital mortality, 6.8%) for pancreatic resection (44 patients).

Our paper is a highly selected series. It includes all patients with infected necrosis in whom the decision has been made to formally debride and this included patients with complicated acute pancreatitis referred internally from other firms. Our own mortality rate for acute pancreatitis is low, but meaningless, as we do not claim to have information on all patients with acute pancreatitis in our own institution. The actual incidence of pancreatitis is notoriously difficult to pin down and I would be interested to know if Mr Ammori has confident data of his own institution.[1] It is our policy to drain, percutaneously, any fluid collection in patients who are deteriorating and reserve surgical debridement for patients with solid infected necrosis. Our surgical approach is deliberately aggressive. We recognise that we are taking out a normal spleen in middle-to-late aged patients to effect radical clearance of the pancreatic bed and surrounding inflamed area. We are impressed that by doing so most patients require one operation and they survive. All three deaths in the paper were in patients with established multi-organ failure prior to surgery, hence the importance of recognising the deteriorating patient and intervening before multi-organ failure supervenes. Recurrent or persisting sepsis after intervention for pancreatic infection is likely to be the most common cause of treatment failure.[2] It is noteworthy that patients can continue to drain infected fluid via the drain sites often for a number of months post procedure.

With regards to the risk of post-splenectomy sepsis, I refer Mr Ammori to the Working Party of the British Committee for Standards in Haematology.[3] The subsequent risk of post-splenectomy sepsis in this low risk group of patients is likely to be very small and we agree that overwhelming post-splenectomy infection should be preventable if simple precautions are taken. Although patients with acute pancreatitis have been shown to have reduced cellular immunity and it was kind of Mr Ammori to reference one of my own papers, its clinical significance is unknown. Pancreatic sepsis has high morbidity/mortality and we have shown that the combination of radical and early surgery can achieve good results. Colectomy was performed in all three patients with established multi-organ failure who subsequently died. The apparent ischaemia may have been secondary to generalised poor perfusion in compromised patients; however, the approach has been to not close the abdomen on potentially ischaemic gut as postoperative assessment in these patients is difficult enough. There was no experience of direct trauma to colic vessels and by operating on fitter patients, before deterioration, the need for colectomy has reduced. One patient in our series is diabetic and there are three who are maintained by supplementation with pancreatic enzymes.

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