PostScript

CORRESPONDENCE

Thoracic invasion in gastric carcinoma

In general, superior vena cava obstruction, an uncommon manifestation of lung cancer, is caused by carcinoma of the bronchus, and less commonly by lymphoma, metastatic disease, and intrathoracic tumours.[1] We describe a case of superior vena cava thrombosis caused by pleural metastases arising from a gastric adenocarcinoma.

A 70 year old man presented with a two month history of persistent epigastric pain, nausea, weakness, fatigue, anorexia, and progressive weight loss. He had smoked 30 cigarettes a day for many years. An endoscopic examination and an abdominal computed tomography scan demonstrated a gastric mass located in the upper third of the stomach, with multiple adenopathies on the head of the pancreas and the coeliac trunk. Histopathological study of the mass revealed a poorly differentiated carcinoma. The patient was sent to another hospital to evaluate palliative surgery. Four weeks later the patient was admitted because of progressive dyspnoea and coughing. Physical examination revealed cyanosis and jugular venous distention. The white blood cell count was 29.4×10^9 leucocytes/litre, with a left shift, and the erythrocyte sedimentation rate was 91 mm/hour. Other laboratory data were within the normal limit. A chest *x* ray showed a 10×10 cm diameter mass in the right upper lobe. A computed tomography scan of the chest (fig 1) confirmed the presence of a multilobulated mass with a sharp edge, which filled the right upper lobe without mediastinal mass or nodes. The centre of the mass was hypodense with thick fluid density. The mass invaded the superior vena cava causing intraluminal thrombosis. Fine needle aspiration showed haematic fluid with malignant cytology. These findings suggested the presence of pleural neoplasia with the appearance of haemothorax (tumorous necrosis) secondary to metastatic carcinoma. The patient died one week later.

Pleural metastases arising from a gastric carcinoma and causing a superior vena cava obstruction by invasion and thrombosis are extremely rare. Today, up to 97% of all cases of superior vena cava obstruction have a malignant aetiology, as a result of compression by tumour, mediastinal metastases, or intraluminal thrombus formation.[1] Less than 5% of cases of superior vena cava obstruction are caused by metastatic carcinoma, usually advanced breast, oesophageal, or pancreatic carcinomas.[2] However, gastrointestinal carcinoma, a common tumour, rarely causes superior vena cava obstruction.[3–5] In these cases, the usual mechanism is metastatic adenopathies in the mediastinum, which compress and block the superior vena cava. Up to 10% of tumours of the upper third of the stomach present mediastinal adenopathies. These metastatic adenopathies are more frequent in poorly differentiated carcinomas and those closer to the oesophagogastric junction.[2] Our case is probably the first case of gastric adenocarcinoma reported with invasion and thrombosis of the superior vena cava from pleural metastases and absent mediastinal metastases.

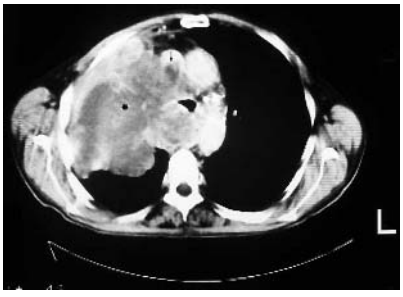

Figure 1 Computed tomography scan of the chest showing a mass filling the right upper lobe (asterisk), invading the superior vena cava and causing intraluminal thrombosis (arrow). No masses or nodes are seen in the mediastinum.

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BOOK REVIEW

Cells, Tissues, and Disease; 2nd edition

Edited by G Majno, I Joris. Oxford: Published by Oxford University Press, 2004, £120.00 (hardback), pp 1005. ISBN 0 19 514090 7

The words delight and textbook seldom appear in the same sentence, but their juxtaposition is more than warranted in the case of this superb book. It is a great improvement over the award winning first edition. As before, there is an introduction followed by 34 chapters (not counting the farewell, which is new); the size of the page has been marginally increased; the index has expanded to a more comprehensive 35 pages; and, most importantly, there are now (by my count) some 5149 references (from antiquity to 2003) and 1104 illustrations in total. The latter number includes vastly more than the 27 of the first edition in colour, and even those images that have been re-used seem to have been noticeably enhanced. The book addresses the subject matter of its subtitle (Principles of general pathology) in five logical, appropriate, and colour coded sections, namely: Cellular pathology, Inflammation, Immunopathology, Vascular disturbances, and Tumours. The authors have interwoven the history of medicine and pathology (so often given short shrift, but here replete with much fascinating illustrative material), the principles of the basic sciences, and the elements of clinical and pathological diagnosis with great skill. The pathology component (the one that I am most able to comment on) includes numerous informative diagrams and excellent macroscopic, light and electron microscopic photographs, in addition to much up to date material from the burgeoning field of molecular pathology (including mention of microarrays). Among the subjects receiving an especially lucid explanation are free radicals, heat shock proteins, apoptosis, thrombosis, and the biology of tumours.

Although a 34 author book would doubtless have proved even more encyclopaedic, the continuity of style and coherence of vision (of "pathology as physiology with obstacles" and of "the cell as the primal patient") in this dual authorship version more than compensate for the lack of numbers.

Emeritus professors Majno and Joris should be lauded for the monumental efforts entailed in producing a 1000 page text that is at once informative and entertaining; especially stimulating is the commentary on dogma incorporated in it from time to time. Their magnum opus should more than meet the requirements of the intended audience (students and teachers) in the fields of medicine and (human) biology—for whom it should, in my opinion, be required reading.

I lament the fact that nothing like this book was available when I was an undergraduate (medical) student; although heartened by the belief that the insights this book has provided have already improved my own lectures, I remain sobered by their comment that "faced with too much to learn each one of us must choose his or her maximum admissible level of ignorance"!

Lest this review appear too effusive, I feel obliged to criticise the second comma in the title, the choice of Pitot over Willis for an early definition of a neoplasm, and the fact that I could find no mention of Vesalius.

R M Bowen

CORRECTION

Sive JI, Baird P, Jeziorsk M, *et al*. Expression of chondrocyte markers by cells of normal and degenerate intervertebral discs. *Mol Pathol* 2002;**55**:91–7. The third author's name was misprinted as Jeziorsk M when it should have been Jeziorska M.

CALENDAR OF EVENTS

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP, The Cedars, 36 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: maggie.butler2@btopenworld.com

Breast Diagnostic Histopathology Update

22–23 September 2005, Hammersmith Hospital and Imperial College, London, UK

Further details: Wolfson Conference Centre, Hammersmith Hospital, Du Cane Road, London W12 ONN, UK. (Tel +44 (0)20 8383 3117/3227/3245; Fax +44 (0)20 8383 2428; e-mail wcc@ic.ac.uk)