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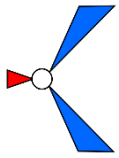

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later, the aspect of the oesophageal wall was completely normal. Now, almost sic years after presentation, this patient is alive and well with no signs of recurrence. Discussion Oesophageal involvement by lymphoma is, in most cases, secondary. Affected mediastinal lymph nodes may either cause narrowing of **the lumen** due to external compression or the oesophageal wall may be invaded directly when tumours spread beyond their anatomical boundaries. Primary involvement, defined as lymphoma developing in the oesophageal wall itself, is rare, especially in its isolated form when it is the only focus of the disease. Including our two patients, only eight cases of isolated primary oesophageal involvement by lymphoma have been published. Tables I and II summarise their main characteristics. As shown, recurrence of Hodgkin's disease, 10 or more years after initial treatment for this condition, was diagnosed in most cases. The nodular sclerosing form was the dominant histological sybtype, although this was not specified in all patients. Isolated primary non-Hodgkin's lymphoma of the oesophagus seems to be exceptionally rare; in both reported cases, **oesophageal lesions** were the first manifestation of the disease. Presenting symptoms were not specific as neither patient initially complained of dysphagia. An atypical presentation may contribute to a diagnostic delay, although this also occurred in patients with more specific symptoms. Diagnostic difficulties in those cases were related to confusing radiological and endoscopic appearances or failure to obtain histological confirmation of lymphoma. A picture at barium swallow typical of malignancy, similar to that described in our case 2 patient, was only reported by Berman et al. Other authors were confronted with a much more puzzling radiological picture, especially in early stages of the disease: a nodular aspect or irregular narrowing of the oesophagus due to submucosal tumours was mentioned most often and interpreted as either peptic stricture, radiation stricture, or external compression. Such interpretations seemed justified as all initial biopsies, with one exception, were negative, indicating either no abnormalities or merely chronic inflammation. Eventually the correct diagnosis was made in seven patients after repeated endoscopic biopsies (case 1 and 2, Hambly and Blundell), open biopsy or examination or surgical specimens. Negative endoscopic biopsies can, to some extent, be explained by the specific growth pattern of the tumour; primary lymphoma arises in submucosal lymphoid patches, hence not within easy reach of biopsy forceps. Accessibility may improve when the tumour grows towards the **oesophageal lumen**, but matters become more complicated when growth is primarily directed in the opposite direction, towards the surface. Fistula formation - for example to the trachea - may then be the first sign of the true nature of the disease. Radiological and endoscopic appearances are mainly dependent on

the presence of intraluminal lesions

. Submucosal swelling may be localised, circumferential, or nultifocal, resembling benign conditions like fibrotic strictures, leiomyoma, achalasia, or varices. Polyps or **papillomatous lesions** may develop as a result of pronounced local proliferation. **Such lesions** are by no means unique and similar patterns may be found when other

	parts of **the gastrointestinal tract** are affected by lymphoma. Although difficult, an early diagnosis of primary oesophageal lymphoma seems to be vital as the response to treatment is often good. Table 2 shows that this is well documented for Hodgkin's disease. Four of the six patients survived five years; two of these four patients were still alive beyond that period, including our case 2 patient, the only long term survivor treated exclusively with combination chemotherapy. Of the three patients that died, the cause of death was not mentioned in one case, a second patient died of oesophageal recurrence, the third succumbed to systemic lymphoma five years after irradiation of a single oesophageal manifestation; local treatment had been completely successful, as indicated at necropsy, which failed to reveal any signs of malignancy in the oesophagus. With the few data presently available, it is difficult to assess whether isolated primary involvement by non-Hodgkin's lymphoma would be equally responsive to treatment; our case 1 patient died of complications before any relevant treatment could be undertaken, whereas follow up in the case reported by Berman et al was too short; only successful local excision of the tumour was reported although 'radiotherapy and chemotherapy were recommended to the patient'. long term survivors (six to nine years) after irradiation or chemotherapy have been described in patients in whom primary oesophageal non-Hodgkin's lymphoma was part of more widespread disease. We have little reason to assume that the response of solitary lesions would be fundamentally different. Given the rarity of isolated primary oesophageal involvement by lymphoma, one may question its clinical significance. Nevertheless, it is our opinion that clinicians should be familiar with its clinical picture as much is at stake; the diagnosis is easily missed, which may have far reaching consequences in terms of prognosis. Dysphagia or, occasionally, more atypical complaints related to the upper gastrointestinal tract developing in young patients with a history of lymphoma warrants suspicion, even in the absence of specific symptoms, enlarged lymph nodes, or a long disease free interval. Histological proof may be difficult to obtain but should be vigorously pursued. Lacking sufficient data, specific advice about treatment can not be given; both local and systemic approaches have resulted in long term survival. Our findings suggest that MOPP combination chemotherapy may be successfully applied. It presents a useful alternative for patients in whom a local approach is contraindicated - for example