pylori, may cause chronic active gastritis. Like C pylori, the number of organisms can probably be suppressed but complete eradication will probably be difficult. Was case 2 endoscoped and assessed symptomatically at a later date: the causative organism may well have been detectable. Dr Logan and his colleagues may be right that "Gastrospirillum hominis" causes non-ulcer dyspepsia, but larger studies are needed and I think it is still premature to draw conclusions from one single patient history.  

Lack of significant effect of therapeutic propranolol on measurable platelet function in healthy subjects

Pamphilon and colleagues have shown that propranolol, a non-selective β blocker, does not significantly inhibit platelet function when therapeutic doses are administered to healthy subjects. They suggest that a different response might be obtained in patients with cardiovascular disease and hyperactive platelets. Some additional comments may be of interest. 

The authors discuss the possibility that β blockers exert their action in situations where circulating catecholamine concentrations are very high. They assessed the effect of acute hypoglycaemia (during insulin stress tests) on arterial and venous platelet function in healthy volunteers. The platelet aggregometry was normal in these subjects. The authors also state that it is possible that the type of β blocker used may influence the results. In conclusion, they suggest that further studies are needed to clarify the role of β blockers in the management of patients with cardiovascular disease.


Dr Pamphilon et al comment:
The comments of Barradas and Mikhailidis are of interest and point to the relevant mechanism whereby β blockers may exert a beneficial effect in patients with myocardial infarction and ischaemic heart disease—that is, modulation of platelet hyperaggregability in susceptible subjects. The observation that propranolol but not metoprolol enhances platelet function and aggregation in vitro (and indeed in vivo) and the effect of propranolol on the platelet volume in either group of subjects using the Coulter S Plus 4 rather than the Coulter C256 channeliser.


Leukaemic phase of mantle zone (intermediate) lymphoma: its characterisation in 11 cases

In his study of lymphoma classifications a distinguished editor of the Journal of Clinical Pathology reflected the confusion and cynicism of the average pathologist at the perceived hair-splitting of the pathologists in their attempts to categorise non-Hodgkin's lymphomas. The confusion over the classification of low grade non-Hodgkin's lymphomas is well illustrated by the report on the leukaemic phase of mantle zone (intermediate) lymphoma by Pombo de Oliveira et al. The category of intermediate cell lymphoma was first proposed by Berard et al. It was noted that the intermediate lymphoma contains some lymphocytes resembling those of well differentiated lymphocytic tumours and some atypical cells more closely resembling the poorly differentiated lymphocytes of nodular or follicular lymphomas. It is now widely accepted that lymphocytic lymphoma of intermediate differentiation (INT) and mantle zone lymphoma (MZL) are the same tumour, perhaps at different stages of evolution, and Jaffe et al have suggested that MZL should be the preferred term.

The category of centrocytic lymphoma was introduced into the Kiel classification to describe a diffuse lymphoma of small, slightly irregular lymphoid cells that were originally thought to be of follicle centre derivation (hence the name centrocyte). Subsequent studies have shown that centrocytic lymphoma is a distinct entity, clinically, morphologically, and phenotypically distinct from follicle centre cell lymphoma. It is unfortunate and confusing that the term centrocytic lymphoma is still applied to this tumour even though the term has been shown not to be of follicle centre origin. Many authors have suggested that INT/MZL is the same entity as centrocytic lymphoma. In a review of INT/MZL, Jaffe et al concluded that this tumour, "clinically, morphologically, and immunophenotypically appears virtually identical to centrocytic lymphoma of the Kiel classification." Pombo de Oliveira et al argued slightly from that position and conclude that, "centrocytic lymphoma and INT are closely related morphologically and phenotypically but may not be entirely interchangeable pathological entities." The authors further state that, "perhaps the application of molecular, genetic, or cytogenetic markers may allow such cases to be appropriately classified in future". In fact, cytogenetic studies suggest a relationship between INT and lymphocytic lymphoma. It is my opinion that the recognition of INT based on the finding of a mixture of cells with round and cleaved nuclei is flawed. It has been recognised for years that quite apart from biological variability, fixation and processing artefacts can induce considerable variation within a single neoplasm. Lymphocytic lymphomas and centrocytic lymphomas have cells that may be identical histologically. It can, however, be difficult to separate these neoplasms on the morphology of the lymphoid cells alone, particularly in suboptimally fixed tissues. There are, however, many other features that assist in the differentiation of these neoplasms, perhaps the most reliable being the identification of parainmunoblasts which are always present in variable numbers in lymphocytic lymphomas and never found in centrocytic lymphomas. On the basis of this criterion, figs 7 and 8 of the paper by Pombo de Oliveira et al show a centrocytic lymphoma and figs 10 and 11 a lymphocytic lymphoma. Clearly, the issue of INT will not be resolved until a more reliable means of identification, other than variability of nuclear shape, is used for cases selected for study.

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