Olsker’s disease is not a telangiectasia but a tumour

Str.—I have read with great interest the instructive article by Zentler-Munro et al. about an association of vascular abnormalities and hepatic fibrosis in Olsker’s disease. Above all, I was impressed by their photographs. In Figure 3, there are dilated vascular spaces practically devoid of erythrocytes and still other ones full of red blood cells. I covered the latter by white stain and looked with astonishment how few hepatocytes remained from original hepatic tissue. To all appearances, a tissue of hepatic lobules was almost destroyed by vessels surrounded by lymphocytes, 7-8 poietic mesenchymal cells (Figs 2, 3, 4, 5, 6, 7, 8) reminded me immediately of similar vessels that I had observed when I studied cutaneous vascular tumours. At that time, I described hyperplastic capillaries composed of epitheloid undifferentiated endothelial cells and situated in the vicinity of a capillary haemangioma, a haemangiopericytoma, and an angioleiomyoma. The capillaries were so similar to sweat glands that a careful morphological analysis was needed in order to distinguish these two structures. These hyperplastic capillaries could give rise: (a) to long narrow strips of smooth muscle tissue by an in situ differentiation of their cells into smooth muscle cells; (b) to pathological muscular vessels by a short distance centrifugal migration and a differentiation of their cells into vascular wall muscular cells; and (c) to ectatic capillaries surrounded by fibroblast-like cells by a migration of their cells into the extravascular space and their differentiation there. 1

A similarity between the vessels described by Nödl1 and the vessels I have observed suggests that Nödl described in reality tumoral lesions. I looked, therefore, for hyperplastic capillaries in his article. I found them in Figs 3, 4, 6c, 6d, 7, 8. Nödl does not mention them because he considers them to be sweat glands. I believe that at least some of these structures are hyperplastic capillaries: (1) a number, a location, and a morphology of pathological muscular vessels indicate that they are not formed by remodelling pre-existing normal vessels; (2) some of them are collapsed and undergoing involution suggesting their high turnover; (3) there are structures clearly recognisable as mesenchymal but similar to sweat gland tubules (Figs 3, 4, 6d — in the middle 7, a large tubule on the right side, 8 — on the right side); (4) referring to my previous observation, endothelial cells in the ‘sweat glands’ often degenerate, most probably because of hypoxia, and their nuclei form clusters leaving empty space in their vicinity (Fig 7 — in the upper right corner); (5) endothelial cell nuclei often possess an elongated form. When they are oriented longitudinally hyperplastic capillaries manifest histopathological patterns incompatible with genuine sweat glands (Fig 7 — in the upper right corner). Finally, there is a remnant of an original hyperplastic capillary composed of several layers of undifferentiated epitheloid endothelial cells (Figs 9, 10). Because there is no other pathological process present, the remnant is not a reactive hyperplasia but a manifestation of tumoral growth.

If Olsker’s disease is a vascular tumour antiangiogenisis3 may be tried in its therapy. This proposition is supported by a successful treatment of this disease by methoxyproges- terone acetate in a 15-year-old girl.4

There are several other diseases which present themselves as telangiectases but derive from hyperplastic tumoral capillaries. They are the senile haemangioma, Cutis marmorea telangiectatica,5 and the spider6, and generalised essential telangiectasia (unpublished personal observation).

13 Beranek JT. Histogenesis of vascular spiders. Pathogenesis hypothesis.

Ranitidine noce (Rhs) v ranitidine mane and noce (Rbid)

Str.—We wish to comment on the abstract by Dobrilla et al. (Gut 1989; 30: A726) stating that there is no significant difference between Rhs and Rbid healing rate at two weeks but the authors did not mention which statistical test and significance level they used.

We found, however, using the χ2 test, without Yates correction, a significant difference between the two treatments at two weeks (χ2 = 5.2 resulting in a p value < 0.05).

It would be interesting to know why, according to the authors, the difference in healing rate was not significant at two weeks.

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Reply

Str.—With regard to the letter of VanWilder and Jan Plas, the comment of these authors is correct but there is an error in our abstract.

In fact, while the reported ranitidine bid healing percentage by week two is correct (64%), the absolute figures are 102/159 and not 107/159, as indicated. Using the χ2 test on the correct number of patients, the difference between the two treatment groups proves to be statistically not significant.

Further details can be found in the full paper which now has been published. (Clin Trial J 1989; 26: 153–62.)

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H pylori and duodenal ulcer

Str.—Dr Carrick and colleagues are to be congratulated on quantifying in their 137 subject the strength of the risk factor for duodenal ulceration (relative risk = 51) of duodenal infec-
Osler's disease is not a telangiectasia but a tumour.

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