

cell-mediated reaction to either a specific virus or several viruses, some of them hepatotropic" is unwarranted on the evidence provided. In the authors' own words, these data are "too vague or speculative" to provide any real insight into the enigma of the relationship of lichen planus and hepatic disease.

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#### REFERENCES

1. Powell FC, Rogers RS, Dickson ER, Moore SB. An association between HLA DRI and lichen planus. *Br J Dermatol* 1986;114:473-478.
2. Seehafer JR, Rogers RS, Fleming CR, Dickson ER. Lichen planus-like lesions caused by penicillamine in primary biliary cirrhosis. *Arch Dermatol* 1981;117:140-142.
3. Powell FC, Rogers RS, and Dickson ER. Primary biliary cirrhosis and lichen planus. *J Am Acad Dermatol* 1983;9:540-545.
4. Rebora A, Rongioletti F, Canepa A. Chronic active hepatitis and lichen planus. *Acta Dermatol* 1982;62:351-352.
5. Mobacken H, Nilsson LA, Olsson R, Sloberg K. Incidence of liver disease in chronic lichen planus of the mouth. *Acta Derm Venerol (Stockh)* 1984;64:70-73.
6. Rebora A, Rongioletti F. Lichen planus and chronic active hepatitis: a retrospective study. *Acta Derm Venerol (Stockh)* 1984;64:52-56.

#### HOW DOES ENDOSCOPIC SCLEROTHERAPY ALTER SPLANCHNIC HEMODYNAMICS?

*Bourbon P, Zarski JP, Kitmacher P, Bourlard P, Machecourt J, Denis B, Rachail M.* Esophageal varice sclerotherapy: influence on portal pressure and azygos blood flow. *Gastroenterol Clin Biol* 1990;14:244-247.

#### ABSTRACT

Endoscopic sclerotherapy is widely employed for esophageal variceal hemorrhage. However it has side effects and can aggravate portal hypertension by suppression of portosystemic shunt. The purpose of the present investigation was to study the effect of variceal thrombosis on hepatic venous pressure gradient and azygos blood flow. Eight alcoholic cirrhotic patients with a first variceal hemorrhage were included. According to Child Pugh's classification, 4 patients were group A, 2 group B and 2 group C. At each session 40 to 60 ml of 1 p. 100 polidocanol were injected into the varices. A hemodynamic study was performed in each patient before and about 1 week after variceal obliteration (mean 3.3 procedures). Mean value of hepatic venous pressure gradient was  $16.6 \pm 5.5$  mm Hg and  $17.0 \pm 3.8$ , respectively, before sclerotherapy and after eradication of varices; azygos blood flow  $663 \pm 506$  ml/min before and  $682 \pm 522$  after; cardiac output was  $6.5 \pm 0.7$  ml/min before and  $6.5 \pm 0.8$  after. None of these differences were significant. These results suggest that endoscopic sclerotherapy using polido-

canol does not change hepatic venous pressure gradient and azygos blood flow, and does not lower blood flow through the gastroesophageal collaterals draining into the azygos vein. This is consistent with the hypothesis that thrombosis remains localized.

#### COMMENTS

In his first report of endoscopic sclerotherapy (EST) of esophageal varices (EV) in 1947, Moersch suggested that EST may increase the risk of bleeding from gastric varices, and he considered that the presence of gastric fundal varices was a contraindication to the use of EST. Since then, the potential hazard of EST in aggravating the consequences of portal hypertension has been debated. The hypothesis is that EST, by occluding EV and, perhaps, para-EV, could thereby reduce the collateral vessels that shunt blood from the gastric veins. Another hazard of EST that has been suggested is the occurrence of thrombosis in a major vessel of the portal venous system, which could increase the portal venous pressure.

Bourbon et al. suggested, in the work under discussion, that EST using polidocanol did not significantly alter the hepatic venous pressure gradient and azygos blood flow in eight patients with alcoholic cirrhosis who had recently bled from ruptured EV. This interesting study deserves several comments. First, no control group existed. As stated by the authors, this was not thought possible for ethical reasons. However, it greatly limits the conclusions achievable because it has been suggested that a progressive decrease in portal pressure occurs after a bleeding event (1) and that portal venous pressure falls spontaneously in alcoholic cirrhotic patients who have not bled from varices, especially in those patients who abstained from alcohol (2). Second, the number of patients is low, and large intraindividual variations were observed (e.g., portal venous pressure fell by 59% in one patient). Third, the use of the thermal dilution method for prolonged periods to detect chronic variations in azygos blood flow has not been established. This measurement is technically difficult, and the displacement of the catheter between serial measurements may introduce methodological errors because it is not clear that blood flow is measured in precisely the same way. Finally, the authors' results cannot be extrapolated to those with other sclerosing agents because it has been suggested that EST with ethanolamine decreases azygos blood flow by almost one fourth after eradication of EV (3). It has also been suggested that ethanolamine is superior to polidocanol for the eradication of EV and the prevention of rebleeding (4), even though it has also been suggested that the recurrence of EV in patients receiving EST with ethanolamine is caused by incomplete obliteration of the venous feeders of the varices (5).

In three other studies, EST did not significantly alter portal venous pressure (6). On the other hand, several reports have noted gastric or ectopic variceal bleeding after EST (6), and it has been suggested that EST significantly increases the prevalence of large spleno-

renal shunts (7). Moreover, D'Amico et al. (8) have suggested that EST is an independent risk factor for the development of portal hypertensive gastropathy. Finally, the role of EST in inducing portal vein thrombosis is debated (9, 10).

In conclusion, some discrepancies exist between hemodynamic and clinical studies on the venous consequences of EST. EST appears to enhance the development of large shunts or congestive gastropathy and to occlude para-EV in some patients. On the other hand, EST does not have a significant effect on splanchnic hemodynamics. These discrepancies could be explained by differences in the nature of the sclerosing agents used and by the limitations of the hemodynamic findings in relation to the time of bleeding.

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#### REFERENCES

1. Pommier-Layargues G, Villeneuve JP, Willems B, Huet PM, Marleau D. Systemic and hepatic hemodynamics after variceal hemorrhage: effects of propranolol and placebo. *Gastroenterology* 1987;93:1216-1224.
2. Colman J. Portal venous pressure (PVP) falls with time in patients with alcoholic cirrhosis [Abstract]. *HEPATOLOGY* 1988;102:1244.
3. Mastal R, Bosch J, Bordas JM, Bruix J, Kravetz D, Viola C, Rodes J. Efecto de la esclerosis de varices esofagicas sobre el flujo sanguinero de la vena azigos en pacientes con hipertension portal [Abstract]. *Gastroenterol Hepatol* 1985;8:174A.
4. Balanzo J, Sainz S, Espinos JC, Such J, Segura J, Guarner C, Cusso X, et al. Efficacy of ethanolamine and policonanol in the eradication of esophageal varices: a prospective randomized trial. *Endoscopy* 1989;21:251-253.
5. Takase Y, Shibuya S, Chikamori F, Oril K, Iwasaki Y. Recurrence factors studied by percutaneous transhepatic portography before and after sclerotherapy for esophageal varices. *HEPATOLOGY* 1990;11:348-352.
6. Trinchet JC. La sclérose endoscopique des varices oesophagiennes entraine-t-elle une majoration de l'hypertension portale? *Gastroenterol Clin Biol* 1990;14:241-243.
7. Dilawari JB, Raju GS, Chawla YD. Development of large spleno-adreno-renal shunt after endoscopic sclerotherapy. *Gastroenterology* 1989;97:421-426.
8. D'Amico G, Montalbano L, Traina M, Politi F, Pagliaro L. Role of sclerotherapy as risk factor of congestive gastropathy [Abstract]. *J Hepatol* 1989;9:S141.
9. Hunter GC, Steinkirchner T, Burbige EJ, Guernsey JM, Putnam CW. Venous complications of sclerotherapy for esophageal varices. *Am J Surg* 1988;6:497-501.
10. Kawasaki S, Henderson JM, Riepe SP, Brooks WS, Hertzler G. Endoscopic variceal sclerosis does not increase the risk of portal venous thrombosis [Abstract]. *Gastroenterology* 1990;98:A597.