Title:
Hepatic veno-occlusive disease induced by Chinese medicinal herbs

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Dear Editor,

The potential hepatotoxic effects of medicinal herbs is well known and these products are frequently used without an adequate control by the health authorities. We report a case of toxic hepatic veno-occlusive disease (HVOD) which was presumably associated with the use of these herbal remedies. In Asia, pyrrolizidine alkaloids found in herbal medicines are a common cause of HVOD (1,2).

Case report

A 62-year-old Asian man was admitted to hospital due to hepatomegaly and jaundice (bilirubin, 23.6 mg/dl) with SGPT/SGOT at 1,301/506 IU/l and GGT at 527 IU/l. Vascular, obstructive, infectious and autoimmune conditions were ruled out. The liver biopsy was consistent with HVOD (Fig. 1). The patient reported the habitual use (until admission) of over-the-counter, unlabeled “laxative” capsules that he had purchased in China. An analysis performed at the Medicament Inspection and Control Department revealed that the capsules contained proline, anthraquinones and other unidentifiable compounds. He was discharged with no symptoms and only persistent mild cholestasis (GGT, 170 IU/l; Bb, 1.9 mg/dl).
Conclusion
Preparations containing Chinese medicinal herbs that are frequently obtained for the management of minor symptoms usually have a mixed, nonspecific formulation that lack precise information with regard to content. Given their widespread use, the highly variable hepatotoxicity induced by these products (3) is rather uncommon (4). Selected alkaloids produce a toxic destruction of sinusoidal endothelial cells which results in the obstruction of terminal venules (5). Diagnosing the toxic origin of HVOD requires histological confirmation, the exclusion of other causes and a clear temporal relationship. In mild/moderate cases, exposure discontinuation usually leads to a favorable outcome but high mortality rates have been reported on occasions (3). Language and culture barriers contribute to an even more challenging diagnosis.

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References

Fig. 1. A. Mild periportal lymphocytic infiltration (*) with a preserved zone-1 structure (1) which is in contrast to the sinusoidal dilation and congestion that results in hepatocyte collapse/degeneration (2). The central vein is not recognizable due to obliteration (arrow) (hematoxylin-eosin [HE], x4). B. Focal macrovesicular steatosis (1), detachment of damaged endothelial cells (2) and isolated lymphocytes within the central zone (3) (HE, x20). C and D. Severe sinusoidal fibrosis (long arrows) inducing venous flow obstruction with patchy dilation and congestion around degenerated (short arrows) and atrophic liver cells (*). The centrlobular parenchyma is preserved (1) (Masson’s trichrome stain, x10 and x20, respectively).