Letters

Correspondance

Exacerbation of hemochromatosis by ingestion of milk thistle

couple of details in the Case Report "Exacerbation of hemochromatosis by ingestion of milk thistle" by Dr Whittington¹ caught my eye. The hepatotoxicity of milk thistle was assumed by the sudden improvement in the patient's liver function upon discontinuation of milk thistle ingestion. Milk thistle, however, was not the only potential toxin removed from the diet at that time. As was clearly stated, the patient also stopped taking "2 extra-strength acetaminophen pills every 2 or 3 days" and "a can of cola every day." The type of cola was not identified, but it seems likely in an obese patient that the cola would be a diet cola, with aspartame as the sweetener.

Two extra-strength acetaminophen pills (about 1 g) every 2 or 3 days would seem to be an innocuous dose, at least in a healthy individual. And, despite the early fears about aspartame's hepatotoxicity, there is little strong evidence that it poses a serious risk, at least by itself. In certain individuals, however, toxicity can be experienced at "therapeutic" doses of acetaminophen of less than 4 g/day.2 And there is some evidence that aspartame can act synergistically with other food additives to produce neurotoxicity.3

A substantial portion of my practice involves diagnosis and treatment of chronic pain. In my experience, isolated elevations of γ -glutamyl transpeptidase, or a disproportionate elevation of γ -glutamyl transpeptidase relative to the other liver enzymes, is common. Most of

the time it is associated with acetaminophen ingestion at therapeutic or subtherapeutic doses.

I cannot disagree with Dr Whittington's advice to "be cautious about ingesting milk thistle in order to improve liver function," but I think this advice could also apply to many other biologically active substances in patients with borderline liver function. Because of the coincidental cessation of acetaminophen and (possibly) aspartame in this case, blaming the milk thistle might be premature.

> —Robert Kidd MDCM Renfrew, Ont by e-mail

References

- 1. Whittington C. Exacerbation of hemochromatosis by ingestion of milk thistle. Can Fam Physician 2007;53:1671-3.
- 2. Amar PJ, Schiff ER. Acetaminophen safety and hepatotoxicity-where do we go from here? Expert Opin Drug Saf 2007;6(4):341-55.
- 3. Lau K, McLean WG, Williams DP, Howard CV. Synergistic interactions between commonly used food additives in a developmental neurotoxicity test. Toxic Sci 2006;90(1):178-87. Epub 2005 Dec 13.

Response

r Kidd's is a fair comment. Originally the article title included "possible exacerbation," but this was edited. I see many hemochromatosis patients and this patient was unusual. She had a dysmetabolic hepatosiderosis in conjunction with hemochromatosis. Most hemochromatosis patients have a low or normal body mass index. The role of toxins in hemochromatosis has been debated since Gilbert and Grenet implicated alcohol as the culprit in 1896. It took 100 years and the cloning of the HFE gene to dispel the myth