

Acetaminophen (paracetamol) hepatotoxicity with regular intake of alcohol: analysis of instances of therapeutic misadventure.

[Hepatology](#). 1995 Sep;22(3):767-73.
Erratum in: [Hepatology](#) 1995 Dec;22(6):1898.

[Zimmerman HJ](#), [Maddrey WC](#).

Hepatic injury in alcoholics due to intake of acetaminophen (APAP or acetylparaaminophenol) with therapeutic intent has been reported, but the extent of the phenomenon is not clear, pertinent details of the association remain insufficiently clarified, and the importance of the phenomenon is not widely appreciated. The present report describes 67 patients who developed hepatic injury after ingestion of APAP with therapeutic intent. All were regular users of alcohol. Sixty-four percent of the patients were considered to be "alcoholic" or reported intakes greater than 80 g/d, 35% took 60 g/d or less, and the remainder were vague in their reporting. Doses of APAP were in the "nontoxic" range (< 6 g/d) in 60% of the group, within the recommended range (< 4 g/d) in 40%, and at 4.1 to 6 g/d in 20%. Characteristic feature was the towering level reached by aspartate transaminase (AST) with figures ranging from 3,000 to 48,000 IU in more than 90% of cases. Almost 20% of the patients died. The data on these patients were similar to 94 cases of injury from APAP taken with therapeutic intent reported in the literature. This study provides further evidence of hepatic injury in regular uses of alcohol, especially chronic alcoholics, who take APAP with therapeutic intent. Susceptibility is presumably caused by induction of cytochrome P-4502E1 by ethanol and by depletion of glutathione (GSH) because of the effects of alcohol, the malnutrition often associated with alcoholism, and the depletion associated with chronic use of APAP and impaired glucuronidation caused by fasting perhaps as well. The syndrome of liver injury is distinctive, marked by uniquely elevated levels of AST, and poses a significant threat.(ABSTRACT TRUNCATED AT 250 WORDS)



