Paracetamol Poisoning Resulting in Massively Raised Serum Transaminases

Sir,

Paracetamol poisoning is not a very frequently encountered problem in our population, as compared to the incidence in the west. Paracetamol (acetaminophen) is being used as an effective and safer analgesic as compared to non-steroidal anti inflammatory drugs, when administered in therapeutic dose. In contrast to aspirin, it is more likely to cause hepatotoxicity, when taken in larger than therapeutic dose.

We present a case of a 45 year old Caucasian female of British origin, who traveled to Pakistan, 15 days prior to this incident and presented with the history of ingestion of 30 tablets of paracetamol extra (Paracetamol and caffeine preparation) containing 15 gms of paracetamol during previous 23 hours. She vomited twice during this period. She was being treated for depression in England and took this preparation due to its mood elevating effect. Her physical examination was unremarkable. Her bilirubin was 1.6 g/dl, serum transaminases were raised. ALT being 1121 U/I and the prothrombin time (PT) was 16.1 seconds (INR 1.2). Hepatitis serology was negative. After admission to hospital, she was managed with intravenous fluids and oral N-Acetylcysteine (NAC) for the total of 17 doses. Two days later, the transaminases were markedly elevated with ALT of 14,544 U/I. Six days later, jaundice was more pronounced but transaminases were settling down. She was sent home and followed up in outpatient clinics. After 3 weeks, her liver function test were within the normal range.

The popular belief held by many physicians that paracetamol poisoning will not cause elevated liver enzymes within the initial 24 hour period has many exceptions mentioned in different studies. The maximum damage incurred upon the liver, is between 72 to 96 hours post ingestion. This becomes manifest as icterus, encephalopathy, prolonged PT and raised transaminases. History of ingestion is not of prime importance in the management of the paracetamol poisoning, due to lack of details regarding the amount of the drug lost in vomitus and the individual variations in the metabolism of the paracetamol. Thus serum paracetamol level 4 hours after ingestion serves as an important guideline. Unfortunately the test is not available here in Pakistan. After 12 hours, PT must be obtained as it is often the first liver function to become abnormal and thus carries a greater prognostic significance. The protective measures against the liver damage are limited. Glutathione repleting agents, Methionine and N-acetylcysteine, are the two available choices. Our case is important in highlighting the fact that the serum transaminases, after paracetamol poisoning may get raised within 24 hours post ingestion and may rise overwhelmingly within a couple of days without necessarily resulting in liver failure. PT must be followed in these cases as a marker to assess the severity of liver failure. Oral NAC available in pharmacies are good protective agents for the liver and early administration of this should be considered as it may save the patients from a very serious illness.

References


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