

Lethal Acetaminophen Poisoning Related to Error Prescription: Medico Legal Implications - About a Case Report

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Abstract

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Acetaminophen, also known as paracetamol, is a widely used analgesic and antipyretic. It is a common cause of acute liver failure, especially when taken in high doses or for prolonged periods. This case report discusses a fatal case of acetaminophen poisoning related to a prescription error. The patient, a 45-year-old male, presented with symptoms of liver failure after taking a significantly higher dose than prescribed. The case highlights the importance of accurate medication management and the potential legal implications of prescription errors. The authors discuss the clinical presentation, laboratory findings, and the pathophysiology of acetaminophen-induced liver injury. They also explore the medico-legal aspects of the case, including the role of the prescribing physician and the potential for malpractice litigation. The case serves as a reminder for healthcare providers to exercise caution when prescribing acetaminophen and to be vigilant for signs of toxicity. The authors conclude that a thorough review of the patient's medication history and a clear understanding of the prescribed dose are essential to prevent such tragic outcomes. The case also underscores the need for ongoing education and training for healthcare professionals to minimize prescription errors and improve patient safety.

ASAT: 9910UI/L) and toxic level of acetaminophen (73 mg/l) without any other abnormalities. Forensic autopsy was ordered. The external examination revealed no dysmorphic features. The biometrical parameters (height, weight and cephalic perimeter) were within normal range. At dissection, the liver was palish and hemorrhagic. No infectious disease was detected macroscopically. Toxicological screening (22 h after death occurred) revealed a high level of acetaminophen (79 mg/l). Histological examination concluded to diffuse microvacuolar steatosis with centrilobular necrosis.

The availability of acetaminophen in many medical preparations and the contra-indication of aspirin containing products for pediatric use, have made acetaminophen one of the most commonly used analgesic-antipyretic medication in current pediatric medicine [4]. Although it is safe, efficacious and well-established, its ubiquitous presence in the house and easy availability makes it a substance with high potential for harm, both accidentally and non-accidentally [5]. In fact, paracetamol is one of the most commonly implicated substances for poisoning in both children and adults [6]. In children under the age of 5 years acute paracetamol toxicity is usually due to accidental ingestion. In some of cases, the children were given the drug by a parent for therapeutic effect at doses well above those recommended for age and weight. It may occasionally be due to attempted suicide in older children and is rarely due to intentional poisoning for infants. Children may be less prone to paracetamol hepatotoxicity because of developmental differences in the drug's metabolism and its pathways of detoxification [2]. In fact, acetaminophen is primarily metabolized by conjugation in the liver to non-toxic compounds that are eliminated in the urine. In acute overdose or when the maximum daily dose is exceeded over a prolonged period, metabolism by conjugation becomes saturated, and excess acetaminophen is oxidatively metabolized by the CYP enzymes (CYP2E1, 1A2, 2A6 and 3A4) to the hepatotoxic reactive metabolite, N-acetyl-p-benzoquinoneimine (NAPQI). NAPQI has an extremely short half-life and is rapidly conjugated with glutathione and then renal excreted. Under conditions

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