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 mγъdy h osm m א r "ʒ k en ϒ ⍰ s, h ŕ ⍰

ASAT: 9910 UI/L) and toxic level of acetaminophen (73 mg/l) without any other abnormalities. Forensic autopsy was ordered. T e external examination revealed no dysmorphic features. T e 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 a f er death occurred) revealed a high level of acetaminophen (79 mg/l). Histological examination concluded to di f use microvacuolar steatosis with centrilobular necrosis.

T e 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, e f cacious 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 e f ect 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 di f erences in the drug's metabolism and its pathways of detoxi f cation [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 of lar þęlי etÎ_M ham`ag