Nephrotoxicity associated with acute paracetamol overdose: a case report and review of the literature

CS Loh and R Ponampalam

A 29-year-old, 65 kg, Chinese man presented to hospital 10 hours after ingesting 30 g of paracetamol (462 mg/kg body weight). The blood paracetamol level was 145 µg/ml at 10 hours post-ingestion. He had no known risk factors for hepatotoxicity and was treated with intravenous N-acetylcysteine (NAC). Serum creatinine level rose to a maximum of 455 µmol/L on day 8; it gradually declined without the need for dialysis. Little is known of the risk factors for nephrotoxicity, which may occur with or without concurrent liver damage, suggesting possible primary toxic effects on the kidney. The use of NAC in this case may have prevented the progression to liver failure and reduced the severity of the nephrotoxic effects. (Hong Kong j. emerg.med. 2006;13:105-110)

Keywords: Acetaminophen, acetylcysteine, acute kidney failure, analgesics, overdose

Introduction

N-acetyl-para-aminophenol (paracetamol) was discovered in 1889. It was introduced into the United Kingdom market as an analgesic and antipyretic in 1956. Paracetamol is an active metabolite of phenacetin, a compound that was used for its good analgesic and antipyretic properties until it was implicated in analgesic-abuse nephropathy. As paracetamol is well tolerated, it is commonly used as an over-the-counter analgesic and antipyretic for headache and minor musculoskeletal pain.

Hepatotoxicity is the most remarkable feature of paracetamol overdose. Renal effects of paracetamol overdose are less commonly seen than hepatic effects. However, renal impairment may be more common than previously recognised. The overall incidence of acute renal failure in patients with paracetamol poisoning is less than 2%, and acute renal failure occurs in 10 to 40% of patients with severe hepatic necrosis. In 45 adolescents aged 12 to 18 years, mild and severe nephrotoxicity was observed in 8.9% and 2.2% of patients respectively. While many reports associate the renal effects of large doses of paracetamol

---

Keywords: Acetaminophen, acetylcysteine, acute kidney failure, analgesics, overdose

Introduction

N-acetyl-para-aminophenol (paracetamol) was discovered in 1889. It was introduced into the United Kingdom market as an analgesic and antipyretic in 1956. Paracetamol is an active metabolite of phenacetin, a compound that was used for its good analgesic and antipyretic properties until it was implicated in analgesic-abuse nephropathy. As paracetamol is well tolerated, it is commonly used as an over-the-counter analgesic and antipyretic for headache and minor musculoskeletal pain.

Hepatotoxicity is the most remarkable feature of paracetamol overdose. Renal effects of paracetamol overdose are less commonly seen than hepatic effects. However, renal impairment may be more common than previously recognised. The overall incidence of acute renal failure in patients with paracetamol poisoning is less than 2%, and acute renal failure occurs in 10 to 40% of patients with severe hepatic necrosis. In 45 adolescents aged 12 to 18 years, mild and severe nephrotoxicity was observed in 8.9% and 2.2% of patients respectively. While many reports associate the renal effects of large doses of paracetamol.