

BRIEF REPORT

Impact of serum acetaminophen concentration on changes in serum potassium, creatinine and urea concentrations among patients with acetaminophen overdose

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ABSTRACT

Background Acetaminophen overdose may be accompanied by electrolyte disturbances. The basis for electrolyte change appears to be due to increased fractional urinary electrolyte excretion.

Purpose This study investigated the impact of serum acetaminophen concentration on changes in serum potassium, creatinine and urea concentrations in patients with acetaminophen overdose.

Methods This was a retrospective cohort study which included patients admitted to the emergency department and hospital within 24 h of acetaminophen ingestion. The study was conducted over a period of 5 years from 1 January 2004 to 31 December 2008. Data are presented as mean \pm SD and as medians (interquartile range) and groups were compared using independent two-tailed Student *t*-test. Statistical Package for Social Sciences (SPSS) 15 was used for data analysis.

Results Two hundred and eighty-three patients were studied (44 males and 239 females), mean age 23 ± 7.5 years. Patients who had a serum acetaminophen concentration above a 'possible toxicity' treatment line were associated with an elevation in serum creatinine concentration ($p = 0.044$) and a reduction in the serum potassium concentration ($p < 0.001$) but were not associated with a reduction in serum urea concentration ($p > 0.99$). During the study period, 63.3% (179 patients) had serum potassium concentrations less than the normal concentration (3.5 mmol/l) and 31.4% (89 patients) had serum urea concentrations less than the normal concentration (2.5 mmol/l). The serum creatinine concentration in all patients was within the normal range.

Conclusions Acetaminophen appears to cause a concentration-dependent reduction of potassium concentrations and an elevation of creatinine concentrations of short duration (<24 h) after overdose. Copyright © 2010 John Wiley & Sons, Ltd.

KEY WORDS—acetaminophen; potassium; creatinine; urea; overdose

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INTRODUCTION

Acetaminophen is one of the most common antipyretics and analgesics used all over the world. It is easily accessible over the counter and thus intentional acetaminophen overdose is common. In fact, acetaminophen in large doses is capable of causing both hepatic¹ and renal failure.²

The most effective way to diagnose toxicity is by obtaining a serum acetaminophen concentration. A drug nomogram developed in 1975, called the Rumack–Matthew nomogram, estimates the risk of toxicity based on the serum concentration of acetaminophen at a given number of hours after ingestion. This nomogram, called 'normal' treatment line, describes serum acetaminophen concentrations between 200 mg/l at 4 h and 30 mg/l at 15 h.³ To determine the risk of potential hepatotoxicity, the acetaminophen concentration is traced along the nomogram. Use of a timed serum acetaminophen concentration plotted on the nomogram appears to be

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