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Case Report- Acetaminophen Poisoning

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ABSTRACT

Paracetamol is most common analgesic drug used all over the world. It is also single most commonly taken drug in over doses that leads to hospitalisation. paracetamol is rapidly absorbed from small intestine .Twenty Five percent of ingested dose undergoes first pass metabolism in gut wall. In normal conditions N-Acetyl-P-Benzoquinone imine (NAPQI) immediately bound by intracellular glutathione and eliminated in urine as mercapturic adducts. With increased paracetamol doses, greater production of NAPQI may deplete glutathione stores. When glutathione depletion reaches a critical level NAPQI binds to other proteins causing damage to hepatocytes, which may lead to hepatic injury. A 21 year old female presented to the casualty in SreeBalaji Medical College and Hospital with intoxicated state with ingestion of total amount of 7 g of acetaminophen in order to attempt suicide. She was brought to our hospital after half an hour from the time of consuming acetaminophen. The chief complaints were lower abdomen pain and nausea with giddiness and disorientation. Acute acetaminophen poisoning is a most common poisoning in our country due to its easy availability and cheap price. Acetaminophen is one of the drug frequently used for suicide. Activated charcoal seems the best choice to reduce absorption. NAC should be given to patients with overdose. Our patient treatment was successful with NAC.

Key words: Paracetamol, NAC, poisoning, NAPQI

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INTRODUCTION

Acetaminophen is most widely used drug in the population. It is classified as a mild analgesic. It is commonly used in the treatment of headaches and other body aches and symptomatic treatment in many diseases. While combining with opioid analgesics group, acetaminophen can also be used in the management of more severe pain such as post-surgical pain sequel and propalliative adjuvant care in advanced leukemic patients. But if taken in large doses the most important complication of acetaminophen is acute centrilobular hepatic necrosis, but only a small minority of patients is at risk. Analgesics were found to be the most common cause of intoxication in various studies [1-6].

Case

A 21 year old female presented to the casualty in SreeBalaji Medical College and Hospital with intoxicated state with ingestion of total amount of 7 g of acetaminophen in order to attempt suicide. She was brought to our hospital after half an hour from the time of consuming acetaminophen. The chief complaints were lower abdomen pain and nausea with giddiness and disorientation.

On examination she was conscious, general condition was fair, left arm brachial artery blood pressure was 130/90 mmHg and pulse rate was 92 beats per minute.GCS– 14/15.bilateral pupils equally reacting to light.CVS - s1 s2 heard.,RS- NVBS present.CNS-NFND., disorientation present.,P/A- mild tenderness present over right hypochondrial region. An intravenous line was started and infusion of isotonic solutions at 1500 ml for 24hours was maintained. Patients was under central system monitoring and evaluation was done every hour.

Blood was withdrawn for laboratory investigations. Patient’s laboratory findings are as follows :

Sl No	biochemical parameters	Investigations on admission	Investigations on discharge
1.	GLUCOSE	random- 125mg/dl	pp- 110mg/dl
2.	SERUM UREA	38mg/dl	19mg/dl
3.	SERUM CREATININE	0.9 mg/dl	0.7mg/dl
4.	TOTAL BILIRUBIN	0.8mg/dl	0.7mg/dl
5.	ALT	14 u/l	11 u/l
6.	AST	46 u/l	26 u/l
7.	LDH	468 u/l	512 u/l
8.	DIRECT BILIRUBIN	0.7 mg/dl	0.8 mg/dl
9.	BUN	20 mg/dl	14 mg/dl
10.	ALP	182 u/l	142 u/l
11.	PT	19.5 sn	19.7 sn
12.	INR	1.61	1.65
13.	WBC	5700 cu.mm	7900 cu.mm
14.	HCO3	19	22.5
15.	PCO2	46	34.8
16.	PO2	92.1	96
17.	SPO2	97 in rag	98 in rag
18.	PH	7.24	7.46

Metabolic acidosis was diagnosed with ABG reports.

Gastric ravage was done immediately when patient was brought to casualty using riles tube. One gram/ kilogram active charcoal was given via nasogastric tube. N-acetylcysteine (nac) was given intravenously as per protocol because of the alleged toxic levels (>7.5 g).bicarbonate infusions of 50mmol under strict ABG monitoring was performed. So she was on step down ICU care for 48 hours after admission and was shifted toward after 2 days and discharged after four days from the time of admission. One week later the patient came for review and liver function tests were normal. She was given psychiatric rehabilitation therapy.



DISCUSSION

Acetaminophen (paracetamol, n-acetyl-p-aminophenol) is probably the most widely used of all drugs in the world. Acetaminophen poisoning is frequently seen due to accidental consumption or suicidal attempt. A toxic exposure to acetaminophen is suggested when greater than 140 mg/kg is ingested in a single dose or when greater than 7.5 g is ingested within a 24-h period [7].

The clinical presentation of human acetaminophen toxicity can be approximately divided into four stages.

Stage 1

During the first 24 h after exposure patients often have minimal signs and symptoms of toxicity. Some may have minor, Nonspecific signs and symptoms such as anorexia, nausea, vomiting, pallor, and malaise.

Stage 2

Days 2 to 3, clinical signs of hepatotoxicity that may be increased in hepatotoxic patients including right hypochondrial pain and tenderness, and abnormal laboratory reports, such as elevated serum aspartate aminotransferase (ast), alanine aminotransferase (alt), and bilirubin. Even without treatment, most of these patients will recover without sequelae.

Stage 3

Days 3 to 4, however, the conditions of some patients will progress to fulminant hepatic failure. Characteristic findings include metabolic acidosis, coagulopathy, renal failure, encephalopathy, and recurrent gastrointestinal (GI) symptoms. Those patients who survive the complications of fulminant hepatic failure will begin to recover over the next week.

Stage 4

With complete resolution of hepatic dysfunction in survivors ref (7).the plasma acetaminophen concentration provides a good diagnostic indicator, and treatment is successful in patients presenting early with an accurate history, particularly with regard to time of ingestion which is essential for interpreting acetaminophen concentration (ref 2). If a plasma acetaminophen concentration is not measured, urine acetaminophen concentration can be measured. Acetylcysteine treatment was started due to its high toxic levels of acetaminophen.

Treatment measures

NAC can be used orally or intravenously. The standard 72-hour oral NAC regimen is a loading dose of 140 mg/kg followed by maintenance doses 70 mg/kg every 4 h for 17 doses. The 20-hour iv NAC protocol is 150 mg/kg loading dose over 15 minutes, followed by an additional dose of 50 mg/kg over 4 hours and then 100 mg/kg over 16 hours for a total dose of 300 mg/kg. If treatment is initiated within 8 h of acetaminophen ingestion, NAC is nearly 100% effective in preventing the development of hepatotoxicity, as defined by an AST level of greater than 1000 u/l.

In this case, the patient was administered NAC according to intravenous treatment protocol and discharged after 4 days of admission.

CONCLUSION

Acute acetaminophen poisoning is a most common poisoning in our country due to its easy availability and cheap price. Acetaminophen is one of the drugs frequently used for suicide. Activated charcoal seems the best choice to reduce absorption. NAC should be given to patients with overdose. Our patient treatment was successful with NAC.



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