Poster No. 19

WHITE TABLET WITH YELLOW OUTCOME – IS IT REVERSIBLE?

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INTRODUCTION

Acetaminophen overdose is a common cause of acute liver failure worldwide, leading to liver transplant or death. Its antidote, N-acetylcysteine (NAC) has shown great efficacy in treating acute acetaminophen overdose; however, its benefit in delayed presentations has been questioned.

CASE REPORT

A 31-year old intravenous drug user gentleman presented with epigastric pain and vomiting for four days. Upon examination, the patient was jaundiced and had epigastric tenderness. Further history revealed that the patient had deliberately ingested twenty tablets of 500-mg acetaminophen (ten grams) with alcohol, prior to the onset of illness four days ago. A diagnosis of delayed presentation acetaminophen overdose was made. The patient had deranged liver enzymes with aspartate transaminase (AST) of 6,628 units/L and alanine transaminase (ALT) of 5,774 units/L; coagulopathy with international normalized ratio (INR) of 5.77; and detectable serum acetaminophen level at 4.65 µg/mL. Intravenous NAC was initiated in emergency department and continued in the ward for three days. The patient eventually recovered well with improving liver functions. He was discharged home after eight days.

Investigation	Results			Units
	Day 1	Day 6	Day 8	
Haemoglobin	18.8	15.6		g/dL
White blood cells	19.4	10.8		$x10^3/\mu L$
Platelets	270	222		$x10^3/\mu L$
Sodium (Na^+)	127	132		mmol/L
Potassium (K^+)	4.9	4.0		mmol/L
Urea	5.2	2.2		mmol/L
Creatinine	98	58		µmol/L
Prothrombin time	51	14		seconds
INR	5.77	1.08		
Total bilirubin	206.6	166.3	150.8	µmol/L
Direct bilirubin	119.4	84.4	82.9	µmol/L
AST	6,628	207	111	U/L
ALT	5,774	1,278	760	U/L
Albumin	43	36	38	g/L
pH	7.314			
Bicarbonate ($HCO3^+$)	16.3			mmol/L
Base excess	-8.7			mmol/L

Table 1: Blood investigations and results.

DISCUSSION

The time of acetaminophen ingestion to NAC initiation is a major determinant of outcome in acetaminophen-induced hepatotoxicity. Early NAC therapy commenced within eight hours of acetaminophen overdose could significantly reduce serious hepatotoxicity and mortality.[1] NAC prevents hepatic injury by replenishing hepatic glutathione stores, by acting as sulphate precursor, or by directly reducing the toxic byproduct of acetaminophen, N-acetyl-p-benzoquinoneimine (NAPQI).[1] In delayed presentations beyond 24 hours following acetaminophen overdose, patients with fulminant liver failure who fulfill the King's College Criteria would ideally benefit from liver transplant, which is not readily available, especially in resource constraint areas such as in central Sarawak.[2,3] Alternatively, NAC therapy could enhance liver recovery by improving microcirculatory blood flow, increasing tissue oxygen delivery, and decreasing neutrophil infiltration.[1]

CONCLUSION

The effectiveness of NAC therapy in delayed presentations of acetaminophen-induced acute liver failure should not be undervalued and requires additional studies.

Acetaminophen-induced Acute Liver Failure
Arterial pH < 7.3 (irrespective of the grade of encephalopathy)
OR
Grade III or IV encephalopathy AND
Prothrombin time > 100 seconds AND
Serum creatinine > 3.4 mg/dL (301 µmol/L)

Table 2: King's College Criteria for liver transplantation.

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DECLARATION OF CONFLICT

All authors declare that there is no conflict of interest regarding this case presentation.

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