PP6 WHITE TABLET WITH YELLOW OUTCOME – IS IT REVERSIBLE?

YY WONG[1], NF ABD RAHMAN[1]

[1] *HOSPITAL SIBU, SARAWAK, MALAYSIA*

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 Description

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. 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 micrograms/mL. Intravenous NAC was initiated and continued in the ward for three days. The patient eventually recovered with improving liver functions (AST 111 units/L and ALT 760 units/L) and his INR normalized to 0.89. He was discharged home after eight days.

Discussion

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

Conclusion

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

Keywords: Acetaminophen, overdose, NAC