



APAP & ASPIRIN TOXICITY CASE STUDIES

Clinical Toxicology Lab. / 5th Stage

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Case (1): APAP Toxicity

- 39 year old Female presented to the ED awake and alert with mild Right Upper Quadrant pain. She reports that > 24 hours ago she ingested a large number of pills; about 20-30 pills of (Tylenol). Past Medical History includes bipolar disorder and schizophrenia with previous suicide attempts, cocaine abuse. Lab investigations revealed:
 - Na 134 (135-145 mEq/L)
 - K 3.3 (3.3 – 4.8 mEq/L)
 - Bicarb 14 (23 – 30 mmol/L)
 - Cl 103 (95 – 105 mEq/L)
 - BUN 9 (5-25 mg/dL)
 - Creatinine 1.17 (0.7 - 1.5 mg/dL)
 - Glucose 350 (70 – 110 mg/dL)
 - T. Bilirubin 2.1 (0.2 – 1.2 mg/dL)
 - AST 468 (4 – 40 U/L)
 - ALT 100 (4-40 U/L)
 - ALP 100 (4 – 110 U/L)
 - PT 21.7 (11.8-14.5 sec)

Case (1): continue

- Answer the following questions:
 1. Is it acute or chronic toxicity of APAP?
 2. Which organ is most likely affected by APAP toxicity?
 3. How APAP produces its toxic effect?
 4. Would the patient benefit from administration of (activated charcoal)?
 5. Does the patient require hemodialysis?
 6. From the supplemented lab. Data, which are indicative of hepatic injury?
 7. Would the patient condition improve after NAC administration (yes or no) and why?
 8. What is the preferred time to give NAC in order to prevent liver injury?
 9. Can Rumack-Mathiew nomogram be applied to this case (yes or no) and why?
 10. What is the acute overdose of APAP in adults?

Case (2): APAP Toxicity

- 18yr old man presented to ED at 9am with complaint of nausea, vomiting, dizziness and abdominal cramps. Claimed to have taken painkillers early yesterday afternoon and then proceeded to drink a bottle of wine. Lab data revealed:
 - Na = 149 (135-145 mEq/L)
 - K = 3.6 (3.3 – 4.8 mEq/L)
 - Bicarb = 30 mmol/L (23 – 30 mmol/L)
 - BUN = 36 mg/dL (5-25 mg/dL)
 - Creatinine = 1.4 mg/dL (0.7 - 1.5 mg/dL)
 - pH = 7.4
 - INR = 1.6
 - AST = 200 U/L (4 – 40 U/L)
 - ALT = 250 U/L (4-40 U/L)
- Drug screen: Acetaminophen 85 mg/L (5 – 30 mg/L); Ethanol 30 mg/dL. salicylates, opiates, benzodiazepines, cocaine and amphetamines negative

Case (3): APAP Toxicity

- A 57-year-old male (weight 180lb) presented with **nausea and emesis** to a hospital. On examination the patient was slightly febrile and had abdominal tenderness with evidence of **hepatosplenomegaly** , **ascites, and mild jaundice**. The patient admitted to a history of **moderate to high alcohol intake** more than 12 drinks/week for more than 10 years. He stated that he had recently been taking approximately 8 acetaminophen tablets (500mg) during the day for the past several weeks because of persistent headache, which he believed resulted from out breakfast and lunch as part of recent diet, laboratory analysis revealed markedly elevated serum ALT(535IU/L), AST(430IU/L)levels (normal values: 4-51 IU/L and 15-45 IU/L) respectively a bilirubin level of 41 micromol/L (normal < 17 micromole/L, a blood glucose level 2.0mmol/L (3.5-5.8mmol/L) and a **blood acetaminophen concentration of 58 microgram/ml**. the patient was admitted to the hospital and administered an intravenous infusion included **glucose and NAC**.

Case (4): ASA (Aspirin) Toxicity

- A 61-year-old woman presented to the Emergency Department after awakening with left-sided weakness. She had a history of ischemic stroke with an associated seizure disorder. The patient denied recent seizure, and brain magnetic resonance imaging (MRI) showed no evidence of an acute stroke. Following her arrival, she became acutely confused and complained of tinnitus, shortness of breath, and blurred vision. On direct questioning, she gave a history of excessive use of salicylate for the previous two to three weeks. Lab investigations revealed:

▪ Sodium (mEq/L)	138	136-145
▪ Potassium (mEq/L)	2.7	3.5-5.1
▪ Chloride (mEq/L)	106	98-107
▪ Bicarbonate (mEq/L)	12	22-32
▪ BUN (mg/dl)	8	6-20
▪ Creatinine (mg/dl)	0.71	0.44-1.03
▪ Glucose (mg/dl)	115	70-139
▪ Lactic acid	0.9	0.5-2.0
▪ pH	7.46	7.33-7.43
▪ pCO ₂ (mmHg)	21	38-50
▪ pO ₂ (mmHg)	32	30-50
- Her initial serum salicylate level was significantly increased at 78.1 mg/dl (upper therapeutic limit, 19.9 mg/dl).

Case (4): continue

- Answer the following questions:
 1. Is it an acute or chronic toxicity?
 2. Which of the mentioned signs that were indicative of ASA toxicity?
 3. Most cases of ASA toxicity are associated with acid-base disturbance, which lab data are indicative for such disturbance?
 4. Would Rumack-Mathiew nomogram be applied to this case?
 5. Would the patient benefit from the decontamination methods?
 6. Do you think the patient require a hemodialysis at this level of serum salicylate (yes or no) and why?
 7. Why lactic acidosis was measured?
 8. How would you manage such toxicity?