

Utilization of Point of Care (POC) Test: Blood Gas in Diagnosing Aspirin Overdose: A Case Report

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Abstract: Aspirin overdose is not very common in India, but it is still challenging to Emergency Physicians to diagnose, when presented to Emergency Room due to lack of qualitative/quantitative measures in many hospitals. Careful interpretation of history, clinical examination and use of "Point of Care (POC)" testing like Blood Gas analysis helps in diagnosing an Aspirin overdose. Hereby presenting a case where blood gas interpretation helped in diagnoses of a case of Aspirin overdose. A young lady presented with history of consumption of multiple unknown tablets, totally amounting to 60 in number about 20 hours before presentation to ER. On further history patient revealed that she had also taken 5 tablets of Paracetamol (650mg) amongst them. Patient was anxious, febrile, had nausea, epigastric pain, headache. This couldn't be correlated with paracetamol overdose alone. On investigating further, ABG showed primary respiratory alkalosis, primary metabolic acidosis, and hyperchloremic acidosis. Going with clinical history, signs, symptoms and ABG findings we made a provisional diagnosis of Aspirin over dose. Getting a detailed history and interpretation of the point of care investigations appropriately, will help in diagnosis and decreasing mortality and morbidity.

Keywords: Aspirin, blood gas, Point of Care, acidosis, alkalosis, overdose, ER (Emergency Room)

1. Introduction

Aspirin is among the oldest medication remaining in the clinical practice. The use has declined in recent years due to its association with Reye's syndrome in children and availability of other newer drugs like nonsteroidal anti-inflammatory drugs (NSAIDs)¹. It is still a novel antiplatelet drug. Use of aspirin as an analgesic in India is less, which explains lesser case reports of aspirin overdose in India. Salicylate an active metabolite from aspirin is responsible for the toxic effects².

2. Case Presentation

A 25 year old lady brought to Emergency Room with history of consumption of multiple unknown tablets amounting to 60 in number, about 20 hours before presentation. On taking further history, patient revealed that she had also taken 5 tablets of Paracetamol 650mg amongst them, the rest she could not remember. She presented with history of pain abdomen, multiple episodes of vomiting. Her last menstrual period was four days back. No history of any unprotected sex. On examination she was diaphoretic and agitated. She was tachypnic with respiratory rate around 30/min. Pulse rate of 130/min. Blood pressure 124/78mmHg, Spo₂: 98% in room air, temperature (oral): 102°F. Systemic examination revealed mild tenderness in the epigastrium, was moving all four limbs, mildly agitated. Rest of the examination was normal. The history and examination did not correlate with the paracetamol overdose. On further investigating, Blood Gas Analysis (Table 1) revealed mixed acid base disorder, primary respiratory alkalosis, primary metabolic acidosis and hyperchloremic acidosis. The common drug which can cause this finding in blood gas is Aspirin. Provisional diagnosis of Aspirin overdose was made, her symptoms correlated with the moderate toxicity (table 2). Treatment initiated in Emergency Room with urinary alkalization, intravenous potassium supplementation, tepid sponging, anti-emetics, along with proton pump inhibitors. Patient was admitted to intensive care for further care and

management. On following up patient showed improvement in sensorium by the second day and revealed that she had consumed aspirin tablets (75mg), 55 in quantity. Patient discharged from the hospital on 4th day after psychiatric counseling.

Point of care tests performed showed following findings

Table 1: Blood Gas analysis of patient

pH	7.396
pCO ₂	26
Na/K/Cl	140/3.1/123
Lactate	1.8
HCO ₃	15.6
Glucose	77mg/dl
Serum creatinine	0.7

pCO₂: partial pressure of carbon dioxide; Na: Sodium; K: Potassium; Cl: Chloride; HCO₃: Serum Bicarbonate

Table 2: severity grading of salicylate toxicity in adults

	Mild	Moderate	Severe
Acute ingestion dose (mg)	Less than 150mg/kg	150-300mg/kg	More than 300mg/kg
Salicylate levels in blood(mg/dl)	45-65mg/dl	65-90mg/dl	90-110mg/dl
End organ toxicity	Tinnitus Hearing loss Dizziness Nausea/vomiting	Tachypnea Hyperpyrexia Diaphoresis Ataxia Anxiety	Abnormal mental status Seizures Acute lung injury Renal failure Cardiac arrhythmias Shock

mg/kg: milligram/kilogram; mg/dl: milligram/deciliter

3. Discussion

Salicylate poisoning is much less common now a days. A Delay in diagnosis is associated with a mortality of around 15% compared to a much lower rate in those patients in whom diagnosis and initiation of therapy is made early¹. There is no antidote to salicylate poisoning, and management is directed towards preventing further absorption and enhancing elimination of the drug³.

The diagnosis of salicylate overdose is usually suspected from the history, physical examination and acid base findings. Confirmation of the diagnosis requires measurement of the serum salicylate concentration.

Early symptoms of acute aspirin toxicity include tinnitus, vertigo, nausea, vomiting and diarrhea; symptoms of more severe intoxication include altered mental status ranging from agitation to lethargy, hyperpyrexia, non-cardiogenic pulmonary edema and coma (table 2)⁷⁸. Early symptoms are typically present within one to two hours after a single acute ingestion, but various factors can affect the symptom onset, such as multiple aspirin separated by time, ingestion of enteric coated preparations and co ingestions. Therefore, neither the diagnosis of aspirin toxicity nor an estimation of overdose severity should be based upon the timing of symptoms and signs.

Lethal intoxication can occur after ingestion of 10 to 30g by adults and as little as 3g by children. Although toxicity does not correlate completely with serum salicylate concentration and symptoms, most patients exhibit signs of intoxication when serum level exceeds 40 to 50mg/dl (2.9 to 3.6mmol/L); the usual therapeutic range is 10 to 30mg/dl (0.7 to 2.2mmol/L)⁴. Approximately one third of adults who intentionally overdose on aspirin also ingest one or more other medications, many of which are respiratory depressants⁵.

Aspirin is eliminated almost exclusively via kidneys; so serum creatinine concentration should be checked in all patients with salicylate toxicity. Renal failure is an absolute indication of dialysis in salicylate toxicity (table 3)⁶. Hypokalemia should be corrected to achieve effective urinary alkalization with bicarbonate⁹.

Most adults have either a primary respiratory alkalosis or, more commonly, a mixed primary respiratory alkalosis-primary metabolic acidosis. A pure metabolic acidosis is unusual in adults with aspirin overdose.

As with all poisoned patients, treatment involves quick assessment and stabilization of airway, breathing and circulation. This should be followed by gastrointestinal decontamination if indicated. The specific treatment of aspirin consists of alkalization of plasma and urine and in some cases hemodialysis.

Treatment and continued diagnostic testing is no longer necessary when the patient has clinically improved, their acid base status has normalized and blood salicylate level is no longer in the toxic range.

Our experience with this patient is that in the absence of adequate history and quantitative tests to measure plasma salicylate levels, we suggest careful interpretation of the examination findings and point of care tests like blood gas analysis to diagnose the aspirin overdose.

Table 3: Indications of dialysis in aspirin overdose

Altered mental status
Pulmonary edema
Cerebral edema
Acute / chronic kidney injury
Severe acidemia pH ≤ 7.20
Serum salicylate levels more than 90mg/dl
Clinical deterioration despite aggressive and appropriate supportive care

mg/dl: milligram/deciliter.

References

- [1] Done AK. Salicylate intoxication. *Pediatrics*. 1960 Nov 1;26(5):800-7.
- [2] Levy G. Clinical pharmacokinetics of salicylates: a re-assessment. *British journal of clinical pharmacology*. 1980 Apr 1;10(S2).
- [3] Dargan PI, Wallace CI, Jones AL. An evidence based flowchart to guide the management of acute salicylate (aspirin) overdose. *Emergency Medicine Journal*. 2002 May 1;19(3):206-9.
- [4] Hill JB. Salicylate intoxication. *New England Journal of Medicine*. 1973 May 24;288(21):1110-3.
- [5] Gabow PA, Anderson RJ, Potts DE, Schrier RW. Acid-base disturbances in the salicylate-intoxicated adult. *Archives of internal medicine*. 1978 Oct 1;138(10):1481-4.
- [6] Juurlink DN, Gosselin S, Kielstein JT, Ghannoum M, Laverne V, Nolin TD, Hoffman RS, Workgroup EX. Extracorporeal treatment for salicylate poisoning: systematic review and recommendations from the EXTRIP workgroup. *Annals of emergency medicine*. 2015 Aug 31;66(2):165-81.
- [7] Proudfoot AT. Toxicity of salicylates. *Am J Med* 1983;75:99-103
- [8] Done AK. Salicylate intoxication. *Pediatrics*. 1960 Nov 1;26(5):800-7.
- [9] Pearlman BL, Gambhir R. Salicylate intoxication: a clinical review. *Postgraduate medicine*. 2009 Jul 1;121(4):162-8.

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Funding: none
Conflict of interest: None
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