Clinical Case Reports and Reviews

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Salicylate intoxication during treatment for rheumatic fever complicated by endocarditis

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Case report

A previously healthy 15-year-old girl presented with intermittent fever (up to 39°C), migratory polyarthritis, arthralgia, orthopnea and progressive dyspnea in the last 30 days that led to dyspnea at rest and a new heart murmur requiring hospitalization. Admission tests showed normocytic anemia, neutrophil leukocytosis, and also an increase in erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels (Table 1).

After 48 hours, the patient developed pulmonary congestion and tachycardia at rest being transferred to the intensive care unit. Bedside transthoracic echocardiogram showed rupture of the chordae tendineae of the anterior leaflet of the mitral valve with severe mitral regurgitation and moderate tricuspid regurgitation. Infective endocarditis and rheumatic fever were considered as likely diagnoses, and urgent mitral valve replacement with a bioprosthesis was performed with concomitant use of ceftriaxone, gentamicin, oxacillin and rifampicin for the first, and aspirin (total dose of 4 g/day) for the second raised diagnostic suspicion. After five days, despite the improvement of arthritis, she developed tinnitus, vomiting, decreased level of consciousness and convulsive seizure requiring orotracheal intubation and mechanical ventilation. Arterial blood gas analysis showed metabolic acidosis with an increased anion gap. Neurological examination revealed conjugate gaze deviation to the left, positive Babinski sign, mydriatic pupils and involuntary movements in upper and lower limbs (with normal head scan). It was then suspected the diagnosis of salicylate intoxication or Reye's syndrome. Serum salicylate levels were 18.3 mg/dL. The patient presented elevated liver transaminases and prolonged prothrombin time (PT), with gradual improvement in subsequent days after supportive measures and use of sodium bicarbonate to alkalization of blood and urine. She was discharged after completion of antibiotic treatment for endocarditis in good clinical condition.

Table 1. Results of laboratorial tests on admission.

Analyte	Reference range	Result
Hemoglobin (g/L)	121-151	92
Mean corpuscular volume (fL)	80-100	85
White cell count (per mm^3)	4,500-10,000	13,700
Differential count (%)		
Neutrophils	40-75	81.9
Band forms	< 10	13
Lymphocytes	20-50	5
Monocytes	2-10	0.1
Basophils	< 1	0.2
Eosinophils	< 6	0.3
Platelet count (per mm^3)	140,000-360,000	110,000
CRP* (mg/L)	< 5	45
ESR† (mm/h)	< 18	78

*CRP – C-reactive protein

†ESR - Erythrocyte sedimentation rate

Salicylates are ubiquitous agents. Intoxication by this drug remains a significant clinical problem. Aspirin have analgesic, antipyretic and anti-inflammatory properties. This agent can be used for the treatment of soft tissue and joint inflammation and vasculitis such as acute rheumatic fever and Kawasaki disease. Salicylate is available for ingestion as tablets, capsules, liquids and for topical application. Fatal aspirin poisoning may occur after the ingestion of 10 to 30 g by adults and as little as 3 g by children. Early recognition is the key to successful management. The diagnosis must be considered in any patient with a suspected drug overdose and also in those with an unexplained increase in the anion gap. The diagnosis is made based on the history, physical examination and acid-base findings. Confirmation of the diagnosis requires measurement of serum salicylate concentration.

Prominent early clinical features of acute salicylate poisoning include: tinnitus, vertigo, vomiting and diarrhea; more severe intoxication can cause altered mental status, hyperpyrexia, coma, noncardiac pulmonary edema, and death. These signs and symptoms can be arranged in three phases: up to the first 12 hours there is hyperventilation, which is followed by a paradoxic aciduria (due to a potassium loss by the kidneys) and at last there is dehydration,

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hypokalemia and progressive metabolic acidosis. However, a variety of acid-base disturbances can occur with salicylate poisoning. Most adults have either a primary respiratory alkalosis or a mixed primary respiratory alkalosis and primary metabolic acidosis.

Therapeutic serum salicylate concentrations are 10 to 30 mg/dL (0.7 to 2.2 mmol/L); values above 40 mg/dL (2.9 mmol/L) may be associated with toxicity. In patients with clinical signs of salicylate intoxication, serum concentrations should be measured every two hours until two consecutive levels show a continuous decrease from the peak measurement, the most recent concentration falls below 40 mg/dL, and the patient is asymptomatic with a normal respiratory rate and effort.

Chronic salicylate intoxication is often misdiagnosed and a high level of suspicion is needed, especially in high risk patients such as children and the elderly. When intubation is necessary due to primary respiratory failure, care must be taken to ensure appropriately high minute ventilation and maintain alkalemia with serum pH range from 7.50 to 7.59. Adults with salicylate poisoning and clinical signs of toxicity should be treated with alkalinization of the serum and urine. Alkalinization is the mainstay of therapy. We use intravenous sodium bicarbonate treatment. Early nephrology consultation should be obtained and hemodialysis considered for all patients with clinical evidence of severe intoxication. No specific antidote is available for salicylates.

Aspirin-poisoned patients may be hypotensive. Aggressive volume resuscitation is recommended, unless cerebral edema or pulmonary edema is present. Another important aspect is that aspirin intoxication may decrease cerebral glucose concentrations despite of normal serum glucose. Therefore, we suggest that adults with salicylate poisoning who are hypoglycemic or manifest alterations in mental status, regardless of their serum glucose concentration, are treated with supplemental glucose.