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An adult can still die of salicylate poisoning in France in 2008

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Severe salicylate poisoning is potentially fatal in adults. Even if its incidence is low, it is necessary to determine serum salicylate concentration when the classic triad (hyperventilation, tinnitus, epigastralgia) or more subtle manifestations are noticed.

A 74-year-old schizophrenic patient was admitted to intensive care unit for impaired consciousness. At admission, the patient was agitated, confused with a slurry speech. There was no sign of circulatory failure. Temperature was 33.8°C. The rest of clinical examination was normal.

Arterial blood gases on room air were: pH: 7.43, HCO_3^-: 11 mmol/L, $PaCO_2$: 16 mmHg, PaO_2: 91 mmHg. Blood samples showed lactate: 1.7 mmol/L, glucose: 10.1 mmol/L, Na^+: 143 mmol/L, Cl^-: 107 mmol/L, K^+: 5.1 mmol/L, urea: 18.5 mmol/L, creatininemia: 171 µmol/L, leucocytes count: 13.6 G/L. Urinary strip revealed presence of ketone (+++). Cerebrospinal fluid analysis and cerebral tomodensitometry were normal.

Four hours later, arterial pressure decreased to 79/30 mmHg requiring intravenous norepinephrin (1 µg/kg/min). Respiratory rate was 21/min, epigastralgia occurred and temperature rose to 39.2°C. Septic shock was considered and antimicrobial therapy was started. Arterial gases analysis showed: pH: 7.29, HCO_3^-:13 mmol/L, $PaCO_2$: 27 mmHg, PaO_2: 84 mmHg. Plasmatic osmolality was 332 mosm/L. Toxicological screening was performed. The patient was placed on mechanical ventilation.

Two hours later asystolia occurred and remained irreversible despite resuscitation.

We found thereafter plasma salicylate concentration to be 876 mg/L (severe toxicity is considered if >750 mg/L 6 hours after the overdose [1]).

This case highlights the difficulty to diagnose salicylate poisoning in atypical situations. The dosage was only performed 6 hours after admission. There were several explanations to this delay:

First, salicylate poisoning is very uncommon in France. Some French experts even wrote that salicylate poisoning may not be included in aetiologies of exogenous metabolic acidosis in adults [2].

Second, there were no symptoms to suspect poisoning (blister, letter or depression).

The confusion made the classic triad of salicylate poisoning difficult to recognise.

Metabolic acidosis with an increased anion gap without osmotic gap could have made a salicylate poisoning suspected. However, these anomalies were attributed to renal failure and starvation ketosis. The mixed acid-base disturbance was misunderstood and the patient was considered to compensate adequately for a metabolic acidosis. In fact, a normal pH at the admission with an abnormal serum HCO_3^- concentration suggests a mixed disturbance since compensation rarely brings the pH back to normal in acute situation. An associated central respiratory alkalosis, a classical sign of salicylate poisoning, should have been considered. Acidemia appears late in salicylate poisoning evolution, and we should not wait for it to mention this diagnosis. Rapid alkalinisation by sodium bicarbonate would have been useful for decreasing the amount of salicylate crossing through the blood brain barrier and increase renal clearance [3].

Finally fever and shock made the diagnosis of septic shock likely. Patients who present in the late phases of salicylate toxicity are often misdiagnosed with sepsis [4].

Severe salicylate poisoning should be quickly considered in case of both confusion and metabolic acidosis, leading to a rapid alkalinisation and haemodialysis [3]. In all the published cases, a delayed diagnosis was associated with increased mortality [5].

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