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Salicylate Poisoning: Why a Century-Old Drug Continues to Challenge Clinical Toxicology

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Editorial

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For more than a century, aspirin has remained one of the most trusted medications in clinical practice. It has transformed the management of pain, fever, inflammation, and cardiovascular disease, earning a place on the World Health Organization's list of essential medicines. Yet, beneath its remarkable therapeutic value lies an uncomfortable reality—salicylate poisoning continues to present a significant challenge for clinical toxicologists worldwide. In an era of sophisticated diagnostics and advanced critical care, it is surprising that toxicity from such an old and inexpensive drug still contributes to emergency department visits, intensive care admissions, and preventable deaths. This paradox highlights an important truth in toxicology: familiarity with a drug often leads to underestimation of its potential danger.

The widespread availability of aspirin is both its greatest advantage and its greatest weakness. Because it is sold over the counter in many countries, many individuals assume that it carries minimal risk. Patients frequently exceed the recommended dose while attempting to relieve persistent pain or fever, unaware that repeated therapeutic overdoses can be as dangerous as a single massive ingestion. Children remain vulnerable to accidental exposure despite improvements in child-resistant packaging, while intentional overdose continues to be encountered in adolescents and adults presenting after self-harm. Chronic salicylate toxicity is especially concerning among older adults, where prolonged use combined with dehydration or impaired renal function may produce life-threatening toxicity despite seemingly appropriate dosing.

From a clinical toxicology perspective, salicylate poisoning is remarkable because it affects virtually every physiological system. Unlike toxins that act through a single receptor or pathway, salicylates produce a cascade of metabolic disturbances. Early in poisoning, direct stimulation of the respiratory center in the medulla results in hyperventilation and respiratory alkalosis. As toxicity progresses, salicylates uncouple oxidative phosphorylation within mitochondria, impairing cellular energy production while increasing oxygen consumption and heat generation. The result is a mixed acid-base disorder that challenges even experienced clinicians. Patients often transition rapidly from compensatory physiological responses to profound metabolic derangement, emphasizing the importance of early recognition before irreversible complications develop.

The clinical manifestations of salicylate poisoning illustrate why this condition is frequently misdiagnosed during its initial stages. Early symptoms—including tinnitus, nausea, vomiting, dizziness, diaphoresis, and tachypnea—are nonspecific and may resemble viral illness, gastroenteritis, anxiety disorders, or sepsis. Unfortunately, these vague complaints can delay appropriate laboratory evaluation, particularly in busy emergency departments. As toxicity advances, neurological symptoms such as confusion, agitation, delirium, and seizures become increasingly prominent. Pulmonary edema, hyperthermia, renal dysfunction, and cardiovascular instability represent severe systemic involvement and substantially increase mortality. Chronic poisoning is particularly deceptive because serum salicylate concentrations may appear only moderately elevated despite significant tissue toxicity, reinforcing the importance of clinical judgment over laboratory values alone.

Modern clinical toxicology emphasizes that diagnosis extends beyond measuring serum drug concentrations. Although serum salicylate levels remain an important diagnostic tool, they must always be interpreted in the context of symptom progression, acid-base status, renal function, and repeated measurements. Arterial or venous blood gas analysis provides critical insight into evolving metabolic disturbances, while electrolyte assessment often reveals hypokalemia that complicates management. Blood glucose should never be overlooked because central

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nervous system glucose depletion may occur even when serum glucose concentrations remain within normal limits. Consequently, successful diagnosis depends on integrating laboratory findings with careful bedside assessment rather than relying on isolated numerical values.

Treatment strategies for salicylate poisoning have evolved substantially over recent decades, yet the principles remain grounded in sound toxicological practice. Immediate stabilization of airway, breathing, and circulation remains the highest priority, followed by aggressive correction of fluid and electrolyte abnormalities. Activated charcoal continues to play an important role when patients present early after ingestion and have no contraindications to gastrointestinal decontamination. However, the hallmark of salicylate management is urinary alkalinization using intravenous sodium bicarbonate. By increasing urine pH, ionized salicylate becomes trapped within the renal tubules, significantly enhancing elimination while simultaneously reducing penetration into the central nervous system. Maintaining adequate serum potassium is equally important because potassium depletion limits the kidney's ability to produce alkaline urine, thereby reducing treatment effectiveness.

Despite the success of urinary alkalinization, clinicians must recognize situations in which conservative management is insufficient. Hemodialysis remains the definitive treatment for severe salicylate poisoning and should never be viewed as a measure of last resort. Patients with severe metabolic acidosis, altered mental status, pulmonary edema, renal impairment, refractory electrolyte abnormalities, or markedly elevated serum salicylate concentrations benefit substantially from early extracorporeal removal. Beyond eliminating circulating salicylates, hemodialysis rapidly corrects acid-base imbalance and restores physiological stability. Delayed referral for dialysis remains one of the most preventable contributors to poor clinical outcomes, highlighting the need for early consultation with nephrology and clinical toxicology specialists.

Although advances in emergency medicine have significantly reduced mortality, prevention remains the most effective strategy. Public health initiatives should continue emphasizing responsible medication use, particularly among individuals who routinely self-medicate for chronic pain. Pharmacists occupy a unique position in educating patients about maximum daily doses, potential drug interactions, and early warning signs of toxicity. Likewise, healthcare providers should exercise greater caution when prescribing long-term aspirin therapy to elderly patients or individuals with chronic kidney disease, where physiological reserve is reduced and toxicity may develop gradually.

Clinical toxicology also has an educational responsibility. Salicylate poisoning offers an excellent teaching model because it integrates pharmacology, physiology, acid-base interpretation, nephrology, emergency medicine, and critical care. Simulation-based education and case-based learning should continue to emphasize recognition of mixed acid-base disorders, indications for sodium bicarbonate therapy, and timely initiation of hemodialysis. Improving clinician confidence in managing these complex patients may ultimately reduce diagnostic delays and improve survival.

Looking ahead, research in salicylate poisoning should move beyond simply refining treatment protocols. Future investigations should explore biomarkers capable of identifying tissue toxicity earlier than conventional serum concentrations. Artificial intelligence-assisted decision support systems may also help clinicians recognize subtle patterns suggestive of chronic poisoning before severe complications develop. Furthermore, strengthening poison information centers and expanding access to toxicology consultation services could improve patient outcomes, particularly in rural and resource-limited healthcare settings.

Ultimately, salicylate poisoning reminds the medical community that no medication is inherently safe simply because it has existed for generations. Aspirin remains one of medicine's greatest therapeutic achievements, but its benefits must always be balanced against the risk of toxicity. For clinical toxicologists, the challenge extends beyond treating poisoned patients—it involves promoting rational drug use, strengthening public awareness, and ensuring that healthcare professionals maintain a high index of suspicion when evaluating unexplained metabolic disturbances. The continued presence of severe salicylate poisoning in modern healthcare is not merely a toxicological issue; it is a reflection of how easily familiarity can overshadow vigilance. As long as aspirin remains accessible worldwide, clinical toxicology must remain equally prepared to recognize, manage, and ultimately prevent its potentially devastating consequences.

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