Evidence-Based Medicine: spinal immobilisation as a model of medical reversal

Medicina Basada en la Evidencia: la inmovilización espinal como paradigma de reversión médica

To the Editor,

The concept of "medical reversal" refers to the abandonment of an accepted and practiced intervention, not because a superior alternative has emerged, but due to evidence demonstrating its ineffectiveness or harm.[1] Since most healthcare interventions lack high-quality evidence,[2] one might expect that, when subjected to rigorous evaluation, many would undergo medical reversal.[1]

In this context, a recent review by the National Association of EMS Physicians (NAEMSP) could represent an important example of medical reversal in Emergency Medicine. This work concludes that "given the lack of evidence supporting its benefit and the magnitude of data demonstrating harm, spinal immobilization and movement restriction should not be maintained as a standard of care".[3]

Spinal immobilization originated as a hypothesis during World War II and became standard practice following the publication of two case series in 1957 and 1966. Decades later, the NAEMSP review found no study demonstrating definitive clinical benefit. On the contrary, it identified 55 studies showing harm attributable to this intervention.[3]

Such a well-established practice in emergency medicine—arguably one of its defining features—originated from premature conclusions based on retrospective analyses without control groups. Despite this, it became a universal standard and persisted for decades without adequate evidence of effectiveness.[3]

In the case of spinal immobilization, two particularly relevant and common causes of medical reversal can be identified: excessive reliance on pathophysiological reasoning and inappropriate use of surrogate outcomes.[4] This phenomenon is based on the assumption that if "cervical movement = neurological injury," then the inverse must also be true.

However, the complexity of human pathophysiology is such that mechanistic reasoning based on limited knowledge rarely accurately predicts clinical outcomes. Not even when the hypothesis seems as logical as imagining that a fractured vertebra, without perfect immobilization, would sever the spinal cord.

This reality highlights the importance of basing clinical practice on studies that go beyond mechanistic surrogate variables and instead measure clinically meaningful outcomes.[4]

The NAEMSP calls for the development of strategies to reduce the use of cervical collars and proposes limiting the use of spinal boards and vacuum mattresses to rescue situations. Although efforts to reduce indiscriminate immobilization began more than 20 years ago with tools such as the Canadian C-Spine Rule and NEXUS criteria, these tools were developed under the assumption that immobilization was effective. The findings of this review therefore require reconsideration of these tools and of prehospital trauma immobilization as a whole.

In a broader context, this review represents another lesson in humility and caution in the face of the complexity of medicine.[5] It also serves as a warning against the weak pathophysiological logic that often justifies our interventions.

In settings such as out-of-hospital emergency medicine—where research is challenging but decisions are critical—concepts such as medical reversal can help us better evaluate the evidence supporting each intervention, with the ultimate goal of improving clinical outcomes for our patients.

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Ethical Responsibilities:
All authors have confirmed their authorship, the nonexistence of external funding, and the maintenance of confidentiality and respect for patients' rights in the author's responsibilities document, publication agreement, and assignment of rights to Revista Española de Urgencias y Emergencias.

Editor in Charge:
Rafael Castro Delgado.

ARTICLE INFORMATION

Data Availability: Data are available upon request from the corresponding author.

Use of Generative Artificial Intelligence Tools: The author declares that no AI tools were used in the preparation of this article.

Article not commissioned by the Editorial Board and with external peer review.

Note of the editors: This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Rev Esp Urg Emerg. 2026;5:152-153. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

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Renal failure due to household insecticide, secondary to Ekbom syndrome

Insuficiencia renal por insecticida doméstico, secundario a Síndrome de Ekbom

To the Editor,

The most frequent pesticide exposure in the non-occupational population is due to pyrethrins and pyrethroids used in domestic settings.[1] These substances are considered to have very low toxicity in humans (toxicity category III);[2] however, they may produce adverse health effects at high doses (acute intoxication) or with prolonged exposure (chronic intoxication).

We present the case of an 81-year-old woman with a past medical history of hypertension, diabetes mellitus, and mild cognitive impairment, who was being treated with insulin, metformin, linagliptin, ferroglycine, doxazosin, amlodipine, fenofibrate, pravastatin, haloperidol, quetiapine, aripiprazole, trazodone, ebastine, and timolol. She lived alone. She consulted her physician for nausea and diarrheal stools after consuming artisanal cheese. After 48 hours with diet and oral hydration, symptoms resolved.

One week later, symptoms of nausea and diarrhea recurred, accompanied by asthenia, and she was referred to the emergency department (ED), where creatinine was 1.3 mg/dL with otherwise normal laboratory results and negative cultures, ruling out food poisoning, urinary tract infection, or diabetic decompensation. Supportive treatment was initiated, and she was discharged after 7 days.

Ten days later, symptoms recurred with weakness, dizziness, and dysuria, with a test suggestive of urinary infection, and outpatient treatment with amoxicillin–clavulanic acid was initiated. After progressive worsening with vomiting, loose stools, disorientation, and presyncopal sensation, she was again referred to the ED. On presentation: blood pressure, 146/75 mmHg; respiratory rate, 16 rpm; oxygen saturation, 95 %; temperature, 36°C. Laboratory findings included hematocrit 26 %, hemoglobin 8.7 g/dL, leukocytes 8,230/mm^3, platelets 190,000/mm^3, BUN 65 mg/dL, creatinine 7.04 mg/dL, glucose 111 mg/dL, total proteins 5.7 g/dL, Ca 8.7 mg/dL, Na 136 mEq/L, K 5.2 mEq/L, INR 0.94, prothrombin activity 100 %, fibrinogen 748 mg/dL. Blood gases: pH 7.46, pCO_2 29.0 mmHg, pO_2 143.0 mmHg, HCO_3 20.6 mmol/L. TCO_2: 21.5 mmol/L. Urinalysis showed urea nitrogen 98 mg/dL, urea 209.72 mg/dL, protein/creatinine ratio 14,996.81 mg/g, proteinuria 2,356 mg, osmolality 305 mOsm/kg, Na 129.90 mEq/L, K 14.48 mEq/L. Sediment: erythrocytes ++/µL, leukocyte esterase negative, nitrites negative, erythrocytes >100/field, leukocytes 3–5/field, bacteria negative. Urine and stool cultures were negative. Infectious serology and virology were negative. Autoimmune study was negative. Thyroid hormones, vitamin B12, folic acid, vitamin D, and cholecalciferol were within normal limits. Iron metabolism showed sideremia 45 µg/dL, transferrin 177 µg/dL, ferritin 393 ng/mL, transferrin saturation index 18.1 %. 24-hour urine: diuresis 3,370 mL, albuminuria 62 mg/24 hours, proteinuria 340.37 mg/24 hours, creatinine clearance 74.88 mL/min, sodium 256.12 mEq/24 hours, potassium 80.40 mEq/24 hours.

Abdominal ultrasound ruled out obstructive causes of elevated creatinine, suggesting parenchymal involvement.

On history-taking, the pa-

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Ethical Responsibilities:
All authors have confirmed their authorship, the nonexistence of external funding, and the maintenance of confidentiality and respect for patients' rights in the author's responsibilities document, publication agreement, and assignment of rights to Revista Española de Urgencias y Emergencias.

Editor in Charge:
Montserrat Amigó Tadín.

tient reported a several-month history of skin pruritus, which she attributed to mites. Her medical record included a probable diagnosis of delusional parasitosis (Ekbom syndrome), characterized by the conviction of being infested by parasites, insects, or microorganisms.[3] The patient stated that, to treat this, she had been applying household insecticides daily by spraying them onto her skin after showering, as well as on her bedding and throughout her home. A relative confirmed the use of 5–6 household containers every 15 days for months of a product containing permethrin and tetramethrin (0–0.249 %), propan-2-ol, isopropyl alcohol; isopropanol 0–14.99 %; d-phenothrin [(1R,3R) 2,2-dimethyl-3(2-methylprop-1-enyl)-3-phenoxybenzyl cyclopropanecarboxylate (0–0.249 %)]; ®-p-mentha-1,8-diene (0–0.249 %); β-pinene (0–0.99 %); camphene (0–0.249 %); α-pinene (0–0.99 %).

Previous renal function had been normal, with a creatinine of 1.3 mg/dL, rising to 7.04 mg/dL within approximately 10 days. The rapid progression of renal failure without a clear trigger such as urinary infection or evidence of prerenal failure suggests an acute process. This led to suspicion of a pharmacological/toxic origin, establishing the diagnosis of acute tubular necrosis and Ekbom syndrome.

The patient was admitted and treated with IV fluids, furosemide, general supportive care, and erythropoietin. Her clinical course was favorable within one week, with resolution of the condition and return of creatinine to baseline values. She was discharged asymptomatic, conscious, oriented, and tolerating oral intake. At 3-month follow-up, after eliminating insecticides from the home, symptoms had not recurred and renal function remained normal.

Pyrethroids exert their effect by prolonging the open phase of sodium channels, inhibiting calcium channels and Ca^{2+}–Mg^{2+} ATPase, affecting GABA-mediated inhibition, modulating nicotinic cholinergic transmission, increasing norepinephrine release, and acting on calcium ions. Exposure to insecticides can produce acute injury. Patients with acute kidney injury induced by nephrotoxic substances may acquire it through ingestion or inhalation of toxic agents. Acute kidney failure may result from toxic damage caused by animals,[4] plants,[5] and chemical products, including pesticides,[6] although the most frequent clinical manifestations of pyrethrin toxicity are respiratory (40 %), GI (33 %), and neurological (40 %).[7]

Although no blood samples were collected to measure pesticide levels, this case can be classified as intoxication due to such exposure, based on the Case Classification Matrix for Acute Illnesses Associated with Insecticides.[8]

Between 2015 and 2050, the proportion of the global population aged > 60 years is expected to nearly double, from 12 % to 22 %.[9] Social isolation and loneliness affect approximately one-quarter of older adults and represent a risk factor for health.[10] In this vulnerable and growing population, including lifestyle habits, environmental factors, and exposure to toxic substances (acute or chronic) in the medical history is essential.

ARTICLE INFORMATION

Data Availability: Data are available upon request from the corresponding author.

Use of Generative Artificial Intelligence Tools: The authors declare that no AI tools were used in the preparation of this article.

Article not commissioned by the Editorial Board and with external peer review.

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