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ACUTE INTOXICATION CAUSED BY OPIOID DRUGS: COMPLICATIONS AND PREVENTION

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Introduction: Acute opioid poisoning remains a challenge in Critical care and toxicology, with the growing diversity of potent synthetic opioids and long-acting substances. Opioid-induced severe respiratory failure can initiate hypoxic and metabolic disturbances that extend beyond the initial toxic effect. Although naloxone is a cornerstone of treatment, its shorter duration of action compared with certain opioids may cause renarcotization, a period of transient clinical improvement. Consequently, survival depends not only on reversing opioid effects but also on early recognition and preventing secondary complications.

Aims: To characterize the major life-threatening complications associated with severe acute opioid poisoning, examine their underlying pathophysiological mechanisms, and identify clinical priorities for reducing complication-related morbidity and mortality.

Materials and Methods: A retrospective cohort analysis was undertaken using clinical records from a critical care toxicology department. The study population comprised patients with acute severe opioid poisoning complicated by coma and respiratory failure. Documented outcomes included aspiration, respiratory failure, toxic-hypoxic encephalopathy, rhabdomyolysis, acute kidney injury, compartment syndrome, cardiovascular failure, and cardiac arrest. Complications occurring in patients who received naloxone were also considered. Serum lactate concentration, duration of coma, and length of intensive care unit (ICU) stay were examined as clinically relevant indicators of illness severity and treatment course.

Results: The clinical analysis focused on the complex interaction between opioid-induced hypoventilation, tissue hypoxia, impaired consciousness, and secondary organ damage. Persistent or recurrent respiratory depression may contribute to neurological injury, while aspiration can further compromise pulmonary function. Prolonged immobilization during coma may promote muscle ischemia, rhabdomyolysis, and acute kidney injury, with compartment syndrome

representing a further potentially serious consequence. Elevated lactate concentrations may provide additional evidence of impaired tissue perfusion. The occurrence of complications despite naloxone administration underscores the need for continued monitoring and comprehensive supportive care.

Conclusion: Severe opioid poisoning should be approached as a potentially multisystem toxicological emergency rather than an isolated episode of respiratory depression. Early airway management, effective ventilation, appropriately titrated naloxone, surveillance for recurrent toxicity, and systematic assessment for secondary organ injury are fundamental components of care. Individualized treatment directed toward the complications identified in each patient may reduce preventable organ failure. Further evaluation of clinical predictors, including lactate concentration and coma duration, may help improve risk complications and guide intensive care management.

Keywords: opioid poisoning, hypoxia, renarcotization.