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Malignant drug-induced rhabdomyolysis

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Recently, several cases of poisoning following the different drug poisoning have been referred to our emergency department with more severe conditions that different and more intense in contrast to cases have been reported previously. These patients nearly almost died despite ICU care. Due to not-so-long-term toxicity it appears that another mechanisms than mentioned before are involved in the occurrence and progression of this syndrome. In order to figure out the causes of this syndrome future studies are recommended.

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Dear Editor,

he syndrome of rhabdomyolysis is the result of skeletal muscle injury that alters the integrity of the sarcolemma and leads to the final release of creatine phosphokinase (CPK), lactate dehydrogenase (LDH) and myoglobin into the interstitial space and plasma. There are numerous causes that can lead to acute rhabdomyolysis and many of patients present with multiple causes include muscle trauma (vigorous exercise, crush injuries, battering, or seizures), inadequate blood perfusion, heat stroke, electrolyte imbalance, hereditary enzyme deficiencies, infections and intoxication of drugs and toxins(1).

Serious drug poisoning is frequently associated with rhabdomyolysis. The mechanisms involved depend on the drug. General anesthetic agents and overdosage with CNS depressants drugs, such as narcotics, cyclic antidepressants, benzodiazepines, antihistamines and barbiturates, cause rhabdomyolysis by pressure-induced ischemia due to prolonged immobilization. Drugs such as LSD, sympathomimetics and phencyclidine, which induce delirium or agitation, and those which cause prolonged involuntary muscle

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contraction (e.g., phenothiazines and butyrophenones), lead to increased ATP demand and eventual exhaustion of its stores (2).

The incidence of drug-induced rhabdomyolysis is uncertain, largely because most of it is unreported. Similarly, the mortality rates are unknown.

Although drug-induced rhabdomyolysis was uncommon in the past, it is no longer rare, due to the introduction of more and increasingly potent drugs into clinical practice and there are numerous reports about different aspect of this syndrome in literature.

Recently, several cases of poisoning following the different drug poisoning (usually methadone poisoning with other drugs) have been referred to our clinical toxicology emergency department with more severe conditions that different and more intense in contrast to cases have been reported previously (according to laboratory and clinical findings on admission and during the course of hospitalization).

Unlike before, these patients subsequent a short period of immobilization, about a few hours, due to loss of consciousness from drug overdosage presented with signs of decreased level of consciousness(GCS= 4-5), multiple necrotic skin lesions in dependent area(Fig1), hyperthermia, respiratory distress, unstability of cardiovascular system .Elevated liver function tests more than 10 folds than normal value, creatinine phosphokinase (CK) increased more than 1000 than normal, an elevation in prothrombin time(PT) and partial thromboplastin time (PTT) and INR, acute renal failure with severe hyperkalemia marked rise in uric acid and hypocalcemia, gasometrical parameters revealed severe metabolic acidosis. These patients nearly almost died despite ICU care.

Due to not-so-long-term toxicity it appears that another mechanisms than mentioned before are involved in the occurrence and progression of this syndrome. In order to figure out the causes of this syndrome future studies are recommended.



Figure1: Multiple necrotic skin lesions in dependent area.

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