Cholinesterase toxicity:

Acute toxicity from organophosphorus agents presents with manifestations of **cholinergic excess**. The dominant clinical features of acute cholinergic toxicity include: **bradycardia, miosis, lacrimation, salivation, bronchorrhea, bronchospasm, urination, emesis, and diarrhea**

SLUDGE/BBB – **S**alivation, **L**acrimation, **U**rination, **D**efecation, **G**astric **E**mesis, **B**ronchorrhea, **B**ronchospasm, **B**radycardia

DUMBELS – **D**efecation, **U**rination, **M**iosis, **B**ronchorrhea/Bronchospasm/Bradycardia, **E**mesis, **L**acrimation, **S**alivation

Other issues include:

Cardiac issues — Cardiac arrhythmias, including heart block and QTc prolongation, are occasionally observed in organophosphorus agent poisoning

Respiratory issues — Fatalities from acute organophosphorus agent poisoning generally result from respiratory failure due to a combination of depression of the CNS respiratory center, neuromuscular weakness, excessive respiratory secretions, and bronchoconstriction

Intermediate (neurologic) syndrome — Ten to 40 percent of patients poisoned with organophosphorus develop a distinct neurologic disorder 24 to 96 hours after exposure. This disorder, referred to as the "intermediate syndrome," consists of characteristic neurological findings including neck flexion weakness, decreased deep tendon reflexes, cranial nerve abnormalities, proximal muscle weakness, and respiratory

Delayed and long-term neuropathology — Organophosphorus agent induced delayed neuropathy (OPIDN) typically occurs one to three weeks after ingestion of one of a small number of specific organophosphorus agents, including chlorpyrifos insufficiency

AKI

Pancreatitis