**AS1-1**

**Advance in the management of acute human poisonings: new treatment modalities**

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**Background:** Acute poisonings remain a major problem in many countries. Timely and effective treatment is important in minimizing the mortalities associated with acute poisonings.

**Methods:** A Medline search of literature reports relating to insulin euglycemic therapy, intravenous lipid emulsion (ILE), and extracorporeal life support (ECLS) was conducted and summarized.

**Results:** Insulin euglycemic therapy was mainly used in the management of beta-blocker and calcium channel blocker poisonings and the mechanisms underlying the therapy include increased inotropy, increased intracellular glucose transport, and vascular dilatation. Both animal studies and numerous case reports suggest that high dose insulin is superior to conventional treatments. The usefulness of ILE in human poisonings was first reported in a patient with bupivacaine related cardiac arrest in 2006. In the past 5 years, many severely poisoned cases receiving ILE were reported and the major mechanism underlying ILE is the "lipid sink" theory. Although there are no published controlled trials (RCTs) on ILE, it seems that ILE maybe useful in patients manifesting life-threatening cardiotoxicity from lipophilic toxicants. The application of ECLS in poisoned patients is rarely reported. In a recently reported study, 2/14 (86%) patients receiving ECLS survived, as compared to 23/48 (48%) patients without ECLS ($p=0.02$). Given the limited reports, ECLS may benefit patients with hemodynamic instability not responding to conventional measures.

**Conclusions:** Limited literature reports suggest that several new treatments may be effective in rescuing severely poisoned patients. Well-designed multicenter prospective cohorts studies and/or RCTs are urgently needed to better understand the effectiveness of such treatments.

**AS1-2**

**Complement inhibition alleviates paraquat-induced acute lung injury**

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The widely used herbicide, paraquat (PQ), is highly toxic and claims thousands of lives from both accidental and voluntary ingestion. The pathological mechanisms of PQ poisoning–induced acute lung injury (ALI) are not well understood, and the role of complement in PQ induced ALI has not been elucidated. We developed and characterized a mouse model of PQ-induced ALI and studied the role of complement in the pathogenesis of PQ poisoning. Intraperitoneal administration of PQ caused dose- and time-dependent lung damage and mortality, with associated inflammatory response. Within 24 hours of PQ-induced ALI, there was significantly increased expression of the complement proteins, C1q and C3 in the lung. Expression of the anaphylatoxin receptors, C3aR and C5aR, was also increased. Compared with wild-type mice, C3-deficient mice survived significantly longer and displayed significantly reduced lung inflammation and pathology after PQ treatment. Similar reductions in PQ-induced inflammation, pathology, and mortality were recorded in mice treated with the C3 inhibitors, CR2-Crry, and alternative pathway specific CR2-fH. A similar therapeutic effect was also observed by treatment with either C3a receptor antagonist or a blocking C5a receptor monoclonal antibody. Together, these studies indicate that PQ induced ALI is mediated through receptor signaling by the C3a and C5a complement activation products that are generated via the alternative complement pathway, and that complement inhibition may be an effective clinical intervention for post exposure treatment of PQ-induced ALI.