OP – 35

Thrombotic microangiopathy following *Daboia* and *Hypnale* envenomation: a descriptive study from Sri Lanka

Eranga Wijewickrama[1], Hasindu Jayathunga[1], Kasun Karunarathne[1], Lalindra Goonarathna[1], Ariaranee Gnanadasan[1], Indika Gawarammana[2], Geoff Isbister[3]

[1]*Faculty of Medicine, University of Colombo*

[2]*Faculty of Medicine, University of Peradeniya*

[3]*Hunter Area Toxicology Service*

Objective: Russell's (*Daboia*) and Hump-nosed (*Hypnale*) viper envenomation is known to cause acute kidney injury (AKI). Thrombotic microangiopathy (TMA) is frequently observed in patients with AKI following snake bites and studying the underlying pathophysiology of TMA may lead to new insights in to venom induced AKI. This study was carried out to assess the incidence of TMA, the spectrum of clinical manifestations associated with TMA, and the relationship between TMA and acute kidney injury in the setting of *Daboia* and *Hypnale* envenomation.

Methods: This is an ongoing prospective descriptive study carried out at Base Hospital, Horana, Sri Lanka. Patients presented to the hospital following *Hypnale*, *Daboia* and unknown snakebites were included in the study. Blood samples for full blood count, reticulocyte count and blood film were obtained on presentation and daily thereafter. Serum creatinine, twenty minute whole blood clotting time (20WBCT), prothrombin time (PT), activated partial thromboplastin time (APTT) and thrombin time (TT) were performed in all. TMA was diagnosed in the presence of thrombombcytopaenia (platelet count <100,000/ml) and microangipathic haemolytic anaemia (MAHA).

Results: A total of 144 patients were enrolled during the period from July 2016 to June 2017 (male 99, female 45). Eighty four cases were due to *Hypnale* and twenty five cases were due to *Daboia* envenomation. In thirty five the biting snake was unidentified. Coagulopathy developed in five patients following *Daboia* envenomation and they were given antivenom. Thrombocytopenia was present in six patients (*Daboia* 1, *Hypnale* 1 and Unidentified 4). Mean haemoglobin level on admission and at 24 and 72 hours was 13.03, 12.89 and 12.53 g/dL respectively. Seven patients developed AKI (Daboia 1, Hypnale 2 and Unidentified 4) and six of them had co-existent TMA. There were four males and three females. Median age was 50. Two of them received antivenom (*Daboia* 1, unknown 1). $6/7$ had TMA. Among those who developed AKI, median lowest Hb level was 6.92 g/dl. Dialysis was done for all 7 patients (median 4 sessions). TMA was not observed in any of the patients without AKI.

Conclusion: TMA is an uncommon manifestation of *Daboia* and *Hypnale* envenoming and appears to be closely associated with AKI. Further studies are required to define the exact role of TMA in the causation of AKI following *Daboia* and *Hypnale* envenomation.