## **Abstract**

Hump nosed viper (Hypnale species) bites are an important cause of mortality and morbidity in southern India and Sri Lanka accounting for 27 and 77% of venomous snake bites respectively. (Ariaratnam CA, 2008 Nov;) Previously they were known to be moderately venomous snakes with predominant local envenomation, but since recently severe systemic envenomation incidents has been reported including hemostatic dysfunction, microangiopathic hemolysis, kidney injury, myocardial toxicity and death.

Here we report a case of hump nosed viper bite complicated with type 2 myocardial infarction and acute pulmonary oedema secondary to acute heart failure in a Sri Lankan female presented to National Hospital of Colombo, Sri Lanka.

39-year-old previously healthy female from Kegalle, was transferred to our hospital for further management of her condition from a local hospital. She gave a history of snake bite 3 days back and the offending snake was identified to be a hump nosed viper. She complained of chest tightness on day 3 of the illness and progressive shortness of breath with associated orthopnea and paroxysmalnocturnal dyspnea. She had bilateral lower limb oedema. She denied of pasthistory of similar episodes and her past medical history was insignificant. Herconscious level was stable, but she was tachypneic and tachycardic withrespiratory rate of 32/min and 120bpm respectively. She was hemodynamically stable with a blood pressure of 100/60mmHg. She had bilateral lower zone finecrepitations in the lungs with elevated jugular venous pressure and her heartsounds were normal. She had oxygen dependency with on air saturation of ~85%. Upon monitoring her urine output was adequate. Her ECG showed lateral lead STsegment depressions (I, avL, V2-V6 leads) and T wave inversions. Her cardiactroponin I titer was elevated with a value of 5.9 ng/ml. her renal functions were normal 0.8 mg/dl and chest X-ray showed evidence of pulmonary oedema and 2Dechocardiogram showed an ejection fraction of 60% with mild lateral wallhypokinesia. CT coronary angiography was carried out with the metabolicscreening which revealed normal coronary arteries and a negative metabolicscreening. She was managed with supportive therapy with loop diuretics, oxygenand follow up 2D echocardiogram showed complete recovery of the cardiac function and she was asymptomatic at 3 months in to follow up. Therefore, sincethe CT coronary angiogram showed normal coronary arteries, the case was concluded as a case of venom induced type 2 myocardial infarction leading toheart failure with acute pulmonary oedema.