Origins of the Fright Of Snakes and Its Ties with Human Evolution

Fright of snakes is probably one of the most primitive human traits that has puzzled the experimental psychologists (Ohman A, 2007) and evolutionists (Isbell LA, 2006). Lack of data is one of the reasons for which concrete steps to venture into this uncharted territory was made difficult (Warrell DA, 2010). In 2009, snake bite was recognised for the first time by WHO as a neglected tropical disease, later dropped from the list and also rejected by the Bill & Melinda Gates Foundation for funding. However, in June 2017, WHO was forced to reinstate snake-envenoming to the list of neglected tropical diseases (NTDs), under category A (Chippaux J-P, 2017). This inclusion was necessary for boosting initiatives taken in this field.

Per year, 5 million snake bite results in 2.7 million envenoming, 81,000 to 138,000 deaths, 300 000 permanent disability and 1•8–2•7 million serious clinical condition (Chippaux J-P, 2017; Editorial, The Lancet, 2017). In India alone, 200,000 cases of snake bite envenomation are reported each year, resulting in 35,000–50,000 deaths (Cruz et al., 2009). However, the true picture is underestimated due to poor hospital records and dependence on traditional folk treatments. Moreover, while in developed nation snake bite occurs during recreational activities, in developing nation it is a occupational hazard affecting younger people (Cruz et al., 2009).

Snake bite mediated renal failure has been observed in victims of mainly Viperidae bite, and 13% to 32% of pit viper or Russell's viper bite results in renal complications (Chugh KS, 1989). Though anti snake venom serum (ASVS) given within first couple of hours are expected to prevent renal failure in Russell’s viper envenomation, ASVS given within the first hour of bite does not always prevent renal failure (Lewin et al., 1985) which accounts for a significant number of death from Russell’s viper.

Developing countries, hold the lion’s share of global annual mortality estimate i.e. 40,000/yr. India contributes 1/4th of the death rate (Chugh KS, 1989) and the high death rate results from scarcity of ASVS, poor rate of hospitalization, dependence on untested folk medicine and other infrastructural problems. The treatment protocol designed for developed countries hence has little relevance in India. Acute kidney injury, AKI, formerly known as acute renal failure (ARF) from venomous snakes and insects together constituted approximately 3% of all cases of acute renal failure (Chugh KS, 1987) but studies on venom induced ARF are only few. Moreover the mechanism of renal failure by snake envenomation is still unclear.

Although ASVS is effective in keeping the mortality low in developed countries, in developing countries the same solution is rendered ineffective by several factors typical to neglected tropical diseases. Snake bite, despite its response to antivenom treatment, turns fatal in developing countries for infrastructural deficiencies, particularly when envenomation progress into renal complications. 85% of total death from AKI that is contributed by developing countries could have been spared. In this context, International Society of Nephrology has stated ‘0 by 25’ as an oath of not allowing a single death from acute kidney injury or AKI untreated in low-resource regions by 2025 (Mehta et al., 2015). Medical strategies tend to fail in poorer countries often due to lacking in critical consideration of cost.

While biomarker for AKI is a medical challenge, it can also serve strategic role in developing treatment protocol in developing countries. Snake bite patients are treated with ASVS in primary health care centres but dialysis to treat renal complication can be given only at tertiary health care centres. Since serum creatinine or creatinine albumin ratio, the trusted AKI markers, do not change till about 50% loss of renal function, which is often too late to transport the patient from primary to tertiary health care centres, which may take even 8-10 hrs. Moreover, decision of dialysis often relies on clinical observation due to lack of a sensitive biomarker. Sensitive and specific markers for early detection of AKI, viz cystatin C, neutrophil gelatinase associated lipocalin (NGAL), etc are too expensive for this set up.

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