Commentary

Snake envenomation is one of the preventable causes of death. Although a major public health problem in

many countries, the epidemiology of snakebite is still fragmentary, mainly due to the lack of statistical data. This is compounded by the fact that the majority of victims come from rural areas, out of reach of the available medical facilities. It is estimated that snakebites may exceed 5 million per year, out of which approximately 100,000 develop severe sequelae. Early in 2009, snakebite was finally included in the WHO's list of neglected tropical diseases, confirming the experience in many parts of the world that snakebite is a common occupational hazard of farmers, plantation workers, and others, resulting in tens of thousands of deaths each year and many cases of chronic physical handicap.[1] Much is now known about the species of venomous snakes responsible for these bites, the nature of their venoms, and the clinical effects of envenoming on human patients.[2]

In the present case report, a 48-year-old lady developed cerebellar infarction following Russell's viper bite.[3] Following viper bites, haemorrhagic complications are more commonly encountered, but ischaemic complications are also possible and are reported. This is due to the pro coagulant effect of the venom. Most of the ischemic complications after viper bites are reported in the anterior cerebral artery territory. There are only two previous reports of ischemic insult affecting posterior cerebral circulation: brainstem infarct by Lee *et al*[4] and cerebellar infarct by Mugundhan *et al*.[5]

Another unusual feature I observed is that in all previously reported post snakebite ischemic strokes, the event occurred within a few hours (acute phase) of bite, but in this report it has occurred after 48 h of envenomation (sub-acute phase).

Pulmonary oedema reported in this case is also a rare complication. Singh *et al*[6] has already reported a case of severe anaphylactic reaction after the ASV infusion in a cobra envenomation, which rapidly progressed into acute pulmonary oedema necessitating assisted ventilation. In that case report, pulmonary oedema occurred within minutes of ASV infusion. But in the present case report, pulmonary oedema has occurred 48 h later, with no history of ASV hypersensitivity. Here, pulmonary oedema has occurred along with cerebellar infarct, so possibly neurogenic in origin. This is the first time that neurogenic pulmonary oedema has been reported following a viper bite.

The message to be highlighted is the timely administration of ASV . Ideally it should be within 4 h of a bite,[2,7] with maximum doses[8] to be given within the "stipulated time" as per the national protocol[8] or latest WHO protocol.[2] Antivenom neutralizes only the free flowing venom. Once it attaches to the tissues, inflammatory cascade will be initiated leading to all sorts of complications.[2,8] People should be made aware to "get to hospital"[2] at the earliest after any suspected snakebite, since the initial hours are precious with regard to morbidity and mortality. Any modality of treatment other than ASV is of no use. Adequate community awareness programs should be conducted in this regard. Doctors also should be trained in the snakebite treatment protocol . This will improve the effective use of ASV and prevent the wastage of this precious drug.

Despite its importance, there have been few proper clinical studies of snakebite than of almost any other tropical disease. Snakebites probably cause more deaths in the region than do *Entamoeba histolytica* infections, but only a small fraction of the research investment in amoebiasis has been devoted to the study of snakebites. It is recommended that governments, academic institutions, pharmaceutical, agricultural, and other industries and other funding bodies should actively encourage and sponsor properly designed clinical studies of all aspects of snakebite.[2]

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