



Original Research Article

Endoscopic Assessment of Upper Gastrointestinal Lesions in Patients of Hemotoxic Snake Envenomation

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ABSTRACT

Background: Snake bite is a common medical emergency in tropical countries. Mucosal bleeding, especially from gastrointestinal tract is very common in these patients.

Aims: To study the incidence of upper gastrointestinal lesions in patients of hemotoxic snake envenomation by endoscopy and to determine the type and size of these lesions.

Material and Methods: Sixty patients of hemotoxic envenomation admitted in government hospital during 1 year period w.e.f. November 2010 to October 2011 were subjected to upper gastrointestinal endoscopy. Their demographic, clinical and upper gastrointestinal endoscopic findings were noted and analyzed.

Results: Majority of patients were young (75%) between 20 to 50 years of age, male (71.7%) and belonging to rural population (73.3%). All the cases were bitten over the limbs. Fang marks (86.6%) and local swelling (83.3%) was the most common local manifestations. Haemorrhagic syndrome was seen in 56.7% of patients, in which gum bleed (47%) and upper gastrointestinal bleed (23.5%) were the most common presentations. 76.7% of the patients had upper gastrointestinal lesions. Erosive gastritis (63.3%) was the most common lesion, 8.4% patients had duodenitis. Oesophagitis and gastric ulcer were seen in 2 and 1 patient, respectively. Only 14 patients (23.3%) had normal study. In the stomach, fundus (71%) and antrum (63.1%) were the most commonly involved sites.

Conclusions: Mucosal involvement, especially of the gastrointestinal tract is very common among patients of hemotoxic snake envenomation and should be recognized and treated appropriately. To our knowledge, this is the first study of upper GI Endoscopic assessment of snake bite patient in the world. Further research is needed in this aspect of snake envenomation.

Keywords: Snake bite, upper gastrointestinal endoscopy, hemotoxic, gastritis.

INTRODUCTION

Alexander the Great invaded India in 326 BC and was greatly impressed by the skill of Indian physicians, especially in the treatment of snake bites. India is home to some of the most poisonous snakes in the

world, most of which are found in rural India. [1] India has the highest number of deaths due to snake bites in the world with 3500 to 50000 people dying per year, according to WHO direct estimates. However, existing epidemiological data

remains fragmented and the true impact of snakes is likely to be underestimated. [2]

Based on their osteology, mycology, sensory organ system, arrangement of scales, dentition, etc., snakes are categorized into families. The families of venomous snakes are: Atractaspididae, Elapidae, Hydrophidae and Viperidae. The major families in the Indian subcontinent are: Elapidae, Viperidae and Hydrophidae (the sea snakes). [3]

Patients with snake bites should undergo comprehensive workup to look for possible hematologic, neurologic, renal and cardiovascular abnormalities. [4] Clinical effects of snake bites range from mild local reaction to life threatening systemic reaction depending on the species, size of snake, amount of venom injected, size and overall health of the victim, location and depth of bite physiology and reaction of bite victim (panics, remains calm, delays trip to hospital). Various bleeding manifestations include bleeding from recent wounds, gums, epistaxis, haemoptysis, haematemesis, melaena, rectal bleeding, vaginal bleeding, bleeding into the skin (petechiae, purpura, ecchymoses) and intracranial haemorrhage.

Among the challenges faced in improving snake bite treatment and reducing morbidity and deaths is that the research in this field has been neglected. [5] Not much work seems to have been done in evaluation of the gastrointestinal lesions of hemotoxic snake bite poisoning. The exact site of bleeding and the lesion from which bleeding takes place in gastrointestinal tract, is yet to be established specifically. This study is taken up as an attempt to evaluate the upper gastrointestinal lesions in patients of hemotoxic snake bite poisoning.

MATERIALS AND METHODS

The study was conducted on 60 patients of hemotoxic snake envenomation admitted in our center (Government Medical

College, Jammu) during 1 year from November 2010 to October 2011. The patients showed systemic features of hemotoxic envenomation as evidenced by spontaneous bleeding from any mucocutaneous site or laboratory findings suggestive of hemotoxic snake envenomation *i.e.* prolonged clotting time for more than 20 minutes.

Patients who were excluded from the study included those suffering from acid peptic disease, peptic ulcer, chronic liver disease, bleeding disorders prior to snake bite, hemodynamically unstable patients as evidenced by shock, hypotension and those who refused to give consent for the study.

A detailed history and physical examination was conducted. Investigations like haemoglobin estimation, TLC, DLC, PBF and platelet count, blood grouping and cross matching, renal function tests, liver function tests, urine routine examination, clotting time and prothrombin time were carried out. Clot quality and grading were performed as per Reid et al. [6] grading:

Grade 1: Normal – cell deposit does not rise above the bottom curve of the tube, clot approximates 50% of the original whole blood volume.

Grade 2: Slight defect – cell deposits increase above the bottom curve of the tube, up to 30% of the original blood volume, clot size is diminished in proportion.

Grade 3: Moderate defect – cell deposit is 30-50% of the original whole blood volume, clot size is about half of the size of a contracted normal clot.

Grade 4: Severe defect – cell deposit is 50% or more of the original volume, clot is a small speck.

Grade 5: No clot

Upper gastrointestinal (UGI) endoscopy was performed in the gastroenterology section, using Fujinon EG-265 WR endoscope, within 24 hours of presentation, after taking consent for the

procedure. The site and nature of lesions were recorded properly.

RESULTS

The age of the patients ranged from 16 to 80 years. Most of the patients (75%) were in the age group of 20-50 years of age. Males were more frequently affected with male: female ratio being 2.5:1. This is likely due to the fact that snake bite is an occupational hazard in farming and men are more commonly involved in farming as opposed to females. Field predominance of the victims was observed with 73.3% of cases being from rural area with rural to urban ratio being 2.74:1.

76.6% of the patients presented within 12 hours of snake bite. Four of the cases reported after 3 days. This could be explained by the geographical and mountainous terrain in the area with a long travel time to the health centers. Many of the victims first reported to village healers and reported to late to the hospital. All the patients had bite over the limbs with upper limbs (56.7%) being more commonly involved than lower limbs (43.3%). Fang marks (86.6%) and local swelling (83.3%) was the most common local symptoms. Bleeding from the site of bite and color changes were seen in 50.1% and 26.7% cases.

Among the systemic signs and symptoms, haemorrhagic syndrome was seen in 56.7% of cases, nausea and vomiting was seen in 53.3% and pain abdomen in 23.3% cases. Gum bleeding (47%), upper GI bleed (23.5%), hematuria (17.7%) and hemoptysis (14.7%) were the most common manifestations of haemorrhagic syndrome in the present study.

Anemia (46.6%), leucocytosis (33.3%), proteinuria (50%) and azotemia (30.7%) were the commonest laboratory abnormalities, apart from prolongation of

clotting time and PTI (80%), 86.6% had Grade 5 clot at admission.

UGI endoscopic lesions were seen in 76.7% patients and in the rest the study was normal. Erosive gastritis (63.3%) was the most frequently seen lesion, followed by duodenitis (8.4%), esophagitis (3.3%). One patient had a prepyloric ulcer (Table 1).

In the stomach, fundal gastritis (71%) was most common. Antrum and body of stomach were involved in 63.1% and 34.2% cases (Table 2).

Table 1. UGI endoscopic findings of the cases involved in the present study

UGI Endoscopic Findings	No. of Cases	Percentage (%)
Erosive gastritis	38	63.3
Duodenitis	5	8.4
Oesophagitis	2	3.3
Gastric ulcer	1	1.7
Normal study	14	23.3
Total	60	100

Table 2 (a). Distribution of gastritis

Site	No. of Cases	Percentage (%)
Fundus	10	26.3
Body	1	2.6
Antrum	7	18.4
Fundus and body	3	7.9
Fundus and antrum	8	21.1
Body and antrum	3	7.9
Pan gastritis	6	15.8
Total	38	100

Table 2 (b). Distribution of gastritis*

Site	No. of cases	Percentage (%)
Fundus	27	71
Body	13	34.2
Antrum	24	63.1

*multiple sites may be involved in a single patient

DISCUSSION

Demographic profile and clinical profile of the patients was similar to the previous studies done by Bhat, [7] Kulkarni and Anees, [8] Hayat et al. [9] and Alkaaabi et al. [10] Variation in haemorrhagic manifestations among different studies can be explained by sub-species differences of snake venom. As far as the endoscopic findings were concerned, not much data was available for comparison. Only one case reported by Lee et al. [11] showed a broad erosive mucosal change in the esophagus,

congestive mucosal changes, edema and fresh hemorrhage at antrum suggestive of gastritis. No definite ulcer or visible vessel was noted.

As to the mechanisms responsible for the gastric and duodenal lesion, one should consider that snake venom is not a single toxin but cocktail of many components – enzymes, polynucleotides, toxins, non-toxin proteins, carbohydrates, metal, lipids, free aminoacids and biogenic amines. [12]

The mechanisms responsible for the gastric and duodenal erosions and hemorrhages can include the following: direct toxic effects of the venom, arterial obstruction due to fibrin deposition and thrombosis, dysautonomia, shock and thrombocytopenia.

Pro-coagulant enzymes are the major factors in viper venom; these stimulate blood clotting and consumption of fibrinogen as a result of disseminated intravascular coagulation resulting in incoagulable blood. The stimulation of blood clotting results in formation of fibrin in the blood. Most of this is immediately broken down by the body's own fibrinolytic system. These fibrin deposits could be responsible for thrombosis of blood vessels leading to ischemia of the gastrointestinal tract. Iwakiri et al. [13] reported a case of thrombosis of internal iliac artery due to DIC from viper toxin resulting in ischaemic colitis. Rosenthal et al. [14] reported a case of ischaemic colitis after a viperine snake bite which resulted in emergency laparotomy revealing a necrotic ileum and caecum. Intracranial hemorrhage and ischaemic stroke following snake bite have been reported. [15]

Haemorrhagins are the zinc metalloproteinases, they damage the endothelial lining of the blood vessels causing spurting of red blood cells and spontaneous systemic bleeding. Cytolytic or

necrotic toxins damage the cell membranes and stimulate apoptosis. The digestive hydrolases, polypeptide toxins and other factors increase permeability resulting in local swelling and non-healing ulcers and gangrene. Haemolytic and mucolytic phospholipase A2 damage cell membranes, endothelium, skeletal muscle, nerve and RBCs. All these enzymes are responsible for the direct toxic effects of the venom over the GI mucosa and endothelium.

Dysautonomia: Autonomic dysfunction can result in vomiting and abdominal pain and markedly exaggerated circulatory responses in many snake envenomation cases.

Shock: Severe hypotension can enhance the ischemia and gastritis can result from ischemic insult.

Thrombocytopenia: Severe thrombocytopenia (<20000) can also contribute to GI bleeding.

No fatalities were seen in the present study due to the fact that the hemodynamically unstable patients were excluded from the study. So death rate and complications were less as compared to the other studies where death rate was high.

The clinical profile of snake bite varies greatly. Mucosal bleeding, especially from GI tract is very common in these patients. Erosive gastritis, especially fundal and antral gastritis is a common finding in the patients of hemotoxic snake envenomation. In our study, the incidence of UGI endoscopic lesions was 76.4%. Mostly the patients had mixed lesions. The suggested mechanisms for these lesions include increased vascular permeability and endothelial damage due to snake venom toxins and thrombosis caused by fibrin deposits. Gastritis should be appropriately treated with proton pump inhibitors and H₂ blockers. Judicious use of NSAIDs should be done as they tend to aggravate gastritis. To our knowledge, this is the first study of upper GI Endoscopic

assessment of snake bite patient in the world. Further research is required in this field.

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