

## Letter to the Editor

# Topical use of Hemocoagulase (Reptilase): A simple and effective way of managing post-extraction bleeding

Sir,  
Bleeding is a troublesome, embarrassing sequel encountered during or after surgical procedure and can cause distress, agony, and discomfort to the patient and the surgeon both.

Hemocoagulase is the enzyme complex, based fundamentally on coagulative and anti-hemorrhagic properties of fractions isolated from the poison of Bothrops Jararaca or Bothrops atrox.<sup>[1]</sup>

We conducted a study to evaluate the efficacy of hemocoagulase for the management of post-extraction bleeding. A total of 200 patients who underwent symmetrical bilateral orthodontic extractions, were analyzed for bleeding stoppage time (BST). After extraction of teeth, on one side, standardized pom-pom impregnated with placebo [1ml Normal Saline (NS)] was applied and on the other side, hemocoagulase (1 IU) was applied. Visual observation of extraction site was done initially after 1 min and thereafter repeated every 30 s till no bleeding was seen using double blind method. Mean BST value of study and control side were 1.67 and 3.03 min, respectively. Statistical analysis was carried out using unpaired 't' test which revealed  $P < 0.001$  for BST in study group which is highly significant.

Hemocoagulase has thrombin and thromboplastin like action on blood coagulation. *In vitro*, the thrombin-like activity of hemocoagulase transforms fibrinogen into fibrin by gradually splitting off fibrinopeptide-A, giving rise to des-A-fibrin monomers, which polymerize end-to-end to fibrin.<sup>[1-4]</sup> However, unlike thrombin, this activity of hemocoagulase is not inhibited by heparin. The des-A-fibrin monomer produced by the thrombin-like enzymatic activity in the circulating blood remains in solution because it forms a complex with native fibrinogen. These complexes of high molecular weight accelerate the platelet aggregation and reduce capillary permeability at the site of the vascular lesion. The presence of platelet factor III, released from thrombocytes aggregating at a bleeding site, is essential for the thromboplastin-like enzymatic activity of hemocoagulase to activate factor X. This activated factor Xa then supports thrombin formation at the site of hemorrhage.

In the absence of platelet factor III and of factor X *in vitro*, the thromboplastin-like enzymatic activity activates prothrombin to thrombin, which converts fibrinogen to fibrin. This effect is independent of the presence of calcium, though it is accelerated in the presence of calcium ions. Thus, hemocoagulase shortens the bleeding and coagulation time so that blood loss is reduced. *In vivo*, therapeutic doses of hemocoagulase are not shown to initiate intravascular coagulation.<sup>[2]</sup>

Hemocoagulase is easy to use as compared to other local and biologic hemostatic agents like gel foam, absorbable collagen,

microfibrillar collagen, thrombin etc.<sup>[3]</sup> Hemocoagulase is used by some authors in managing capillary bleeding in cleft-palate and skin grafting operations.<sup>[4]</sup>

Hemocoagulase (2U/ml) mixed in fibrin glue can be filled in post-extraction sockets to reduce bleeding. This technique reduces post-extraction bleeding in hemophilic patients with 80% success reducing preoperative factor VIII infusion in hemophilic patients undergoing dental extraction. Hemocoagulase also holds good prospect in managing post-extraction bleeding in cardiac patients on aspirin without stopping aspirin before extraction. Its topical use provides faster hemostasis in patients undergoing dental extraction without any systemic or local adverse effects.

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