Case type- Clinical Tips

Title: Endodontic management of factor x deficient patient

Authors-

Dr Vineet Gupta, Junior Resident, Department of Conservative Dentistry & Endodontics, Faculty of Dental Sciences, King George's Medical University UP Lucknow

Dr. Promila Verma, Professor & Head, Department of Conservative Dentistry & Endodontics, Faculty of Dental Sciences, King George's Medical University UP Lucknow

Dr. A.P. Tikku Former Professor & Head Department of Conservative Dentistry & Endodontics, Faculty of Dental Sciences, King George's Medical University UP Lucknow

Dr Ramesh Bharti, Professor, Department of Conservative Dentistry & Endodontics, Faculty of Dental Sciences, King George's Medical University UP Lucknow

Abstract:

A hereditary hematologic condition called factor X deficiency, also known as Stuart-Prower factor deficiency, affects 1 in 2 million people. The gene causing this condition is autosomal recessive; thus, only those inheriting from both parents exhibit clinical symptoms, such as moderate bleeding, easy bruising and subcutaneous bleeding from mucous membranes. Patients with a considerable deficiency may experience severe internal or external bleeding as a result of trauma or other causes, which can result in consequences including hemarthrosis or hemorrhagic strokes. In this case report, the endodontic management for a patient with severe factor X

deficiency is discussed. It also highlights the importance of timely interdisciplinary communication and consultation to promote a successful outcome.

Keywords: Fresh Frozen Plasma, Hemostat, Clotting Factors.

Introduction:

Researchers found individuals with a hemorrhagic condition that resembled factor VII insufficiency first discovered the coagulation disorder factor X deficiency in the middle of the 1950s. It was given that name in honour of the first two patients (Stuart and Prower) who both exhibited it. Plasma contains roughly 1 mg/dL of factor X, a vitamin K-dependent clotting factor that is produced in the liver.(1) It has a significant impact on the coagulation cascade system as the initial participant in the last common pathway. Postoperative bleeding is frequently a part of surgical operations like dental extractions, but it's usually mild. Patients with known bleeding or clotting issues, however, can be particularly difficult for dentists to treat because excessive bleeding can be dangerous and upsetting for patients and their families.

Case Report:

A 19-year-old presented to the department of Conservative Dentistry & Endodontics, King George's Medical University with the chief complaint of sensitivity to hot and cold. His medical history revealed severe factor X deficiency (figure 1 and figure 2), spontaneous bleeding of joints and shoulders. At 17 years of age, he has undergone root canal treatment with respect 16, after fresh frozen plasma administration.

On intra-oral examination proximal caries was seen in relation to 37, positive for tender on percussion. Radiograph revealed proximal caries involving pulp with lamina dura widening (figure 3), diagnosing it with symptomatic apical periodontics and endodontic treatment was planned with 37. Preoperative recommendations by the haematologist included a fresh frozen plasma administration 6 hours before the procedure. As the half-life of factor X is 25-45 hours (Standard dose: 10-20 mL/kg).

Laboratory findings included prolonged Prothrombin Time(PT) and increased International Normalized Ratio (INR)

Following precautions were taken:

1) Instrumentation and filling should never be done beyond the apical region

2) Teeth should be treated at least 2-3mm short of radiographic apex.

3) Bleeding tendency can be aggravated by NSAIDs and hence safer alternatives for pain control paracetamol and cox-2 inhibitors.

4) Intra-osseous or intra-ligamentary LA administration should be preferred rather than nerve blocks.

5) Use of apex locators reduce the number of intra-operative radiographs thereby decreasing chances of soft tissue injury.

Medical consent prior to procedure was taken. Intraligamentary local aesthetic infiltration with 2 mL of 2% lignocaine with adrenaline 1:100 000 was done. Application of rubber dam was done (figure 4) to prevent soft tissue lacerations and high speed suction can injure the mucosa in the floor of mouth and cause hematoma. Working length of the root canal is calculated precisely to ensure that the instrument do not pass through the apex. WL was kept 1.5-2mm short of the radiographic apex to prevent any inadvertent bleeding and master cone radiograph was taken (figure 5). Biomechanical preparation was done followed by obturation (figure 6) in single sitting as the concentration of replaced factor X starts decreasing with time.

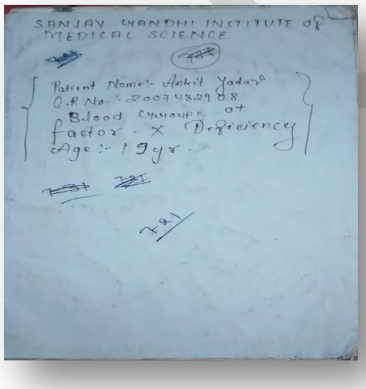

Figure 1: Patient's Case Records

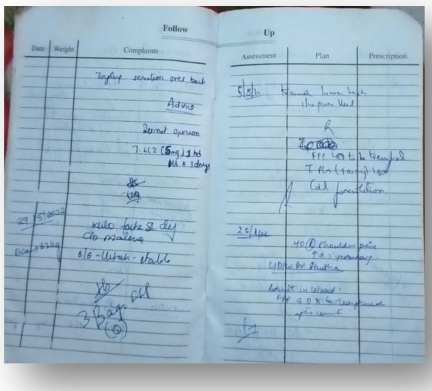

Figure 2: Fresh Frozen Plasma (FFP) Replacement Records

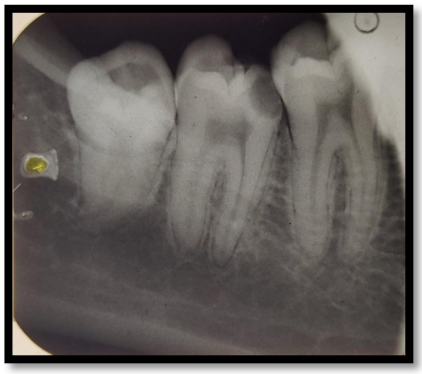

Figure 3: Pre-operative Radiograph.

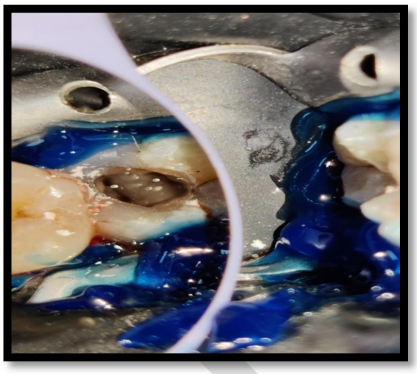

Figure 4: Access Cavity Preparation

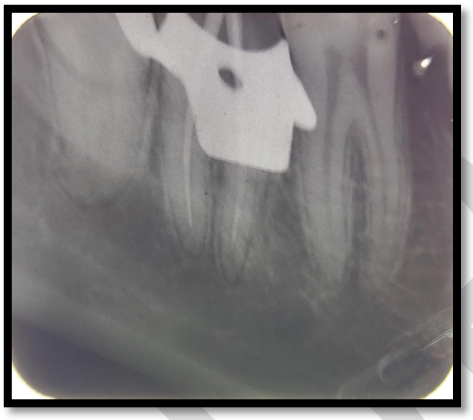

Figure 5: Master cone radiograph

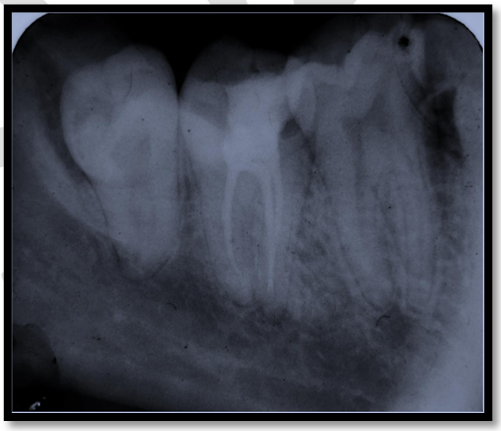

Figure 6: Post-operative radiograph

Discussion:

Factor X, along with factors II, VII, and IX, also needs vitamin K to be synthesised. Factor IXa, along with cofactor factor VIIIa, and factor VIIa, along with cofactor tissue factor, activate it to generate factor Xa. It has a 25–45 hour half-life. The factor X gene is found on the 13th chromosome (13q34) of humans.(3) A clotting disorder known as factor X deficiency occurs when the antigen's quantity and activity are both diminished (type I deficiency) or when the

antigen level is normal or nearly normal but the activity is low (type II deficiency).(2) Factor X deficiency is characterised by prolonged prothrombin time and activated partial thromboplastin time, with normal bleeding and thrombin times. It can manifest as epistaxis, hemarthrosis, or gastrointestinal bleeding.(4) For haemostasis, 10% factor X activity is the ideal level.4 Factor X activity levels in the mild type of the deficit range from 6% to 10%, and the illness may be unintentionally identified after menorrhagia or mild bruising. The severe form (activity level 1%) is typically diagnosed in the neonatal period with umbilical cord bleeding, subcutaneous deep tissue bleeding, or bleeding into body cavities shortly after injury. The moderate form (activity level 1% to 5%) may present with bleeding following a haemostatic challenge, such as surgery or trauma. Fresh frozen plasma (FFP) and prothrombin complex concentrate (PCC) both include factor X, despite the fact that it is not commercially available as a concentrate. The benefits of FFP include immediate accessibility, affordability, and a 20–40-hour extended half-life(5).

To stabilise localised clots in the extraction sockets by blocking the naturally occurring fibrinolytic enzymes in saliva, aggressive local measures and the administration of an antifibrinolytic agent, such as tranexamic acid or aminocaproic acid, are necessary in addition to the advised therapies. Aminocaproic acid is a derivative and analogue of the amino acid lysine, commonly known as -aminocaproic acid or 6-aminohexanoic acid. As a lysine analogue, it binds reversibly to the kringle domain of the enzyme plasminogen and inhibits fibrin's ability to bind to plasmin, which is typically activated. This substance functions as an anti-fibrinolytic or anti-proteolytic agent. Aminocaproic acid can be administered intravenously or orally to treat severe postoperative bleeding.(6)

Conclusion:

The rarity of factor X deficiency makes it impossible to design a specific product to replace it or conduct a formal clinical trial of any existing medication that might be used to treat it. Treatment of patients with uncommon medical conditions can be difficult in situations where there is no protocol for a pre-operative plan. We draw attention to this case to educate dental professionals about challenges associated with the treatment of a rare but serious condition. A successful outcome required prompt and effective communication between all team members.

References:

1. Gupta PK, Kumar H, Kumar S. Hereditary factor X (Stuart-Prower) deficiency. Med J Armed Forces India. 2008;64(3):286-7.

2. Girolami A, Scarparo P, Scandellari R, Allemand E. Congenital factor X deficiencies with a defect only or predominantly in the extrinsic or in the intrinsic system: a critical evaluation. Am J Hematol. 2008;83(8):668-71.

3. Sinha R, Garg R, Chhabra A. Isolated factor X deficiency, a rare occurrence: anesthetic concerns and perioperative management. The Internet Journal of Anesthesiology. 2007;13.

4. Kumar A, Mishra KL, Kumar A, Mishra D. Hereditary coagulation factor X deficiency. Indian Pediatr. 2005;42(12):1240-2.

5. Mori K, Sakai H, Nakano N, Suzuki S, Sugai K, Hisa S, and other. Congenital factor X deficiency in Japan. Tohoku J Exp Med. 1981;133(1):1-19.

6. Ermis B, Ors R, Tastekin A, Orhan F. Severe congenital factor X deficiency with intracranial bleeding in two siblings. Brain Dev. 2004;26(2):137-8.