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Control of Life-Threatening Head and Neck Hemorrhage After Dental Extractions: A Case Report

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Report of a Case

A 54-year-old man presented to the North Central Bronx Hospital Dental Clinic (Bronx, NY) on October 30, 2007, with a complaint of pain in his right mandible. The patient's vital signs were unremarkable on presentation with a blood pressure of 140/75 mm Hg and a pulse rate of 85 beats/min. The patient reported having a stroke 10 years previously that was managed with aspirin therapy for 7 years. The patient denied taking any medications. He had no known drug allergies but admitted to occasional alcohol use. He denied any drug or tobacco use on the medical intake questionnaire and during the initial consultation. He noted a history of bruising easily but reported having previous dental extractions without incident. A panoramic radiograph (Fig 1) showed multiple nonrestorable teeth (maxillary right third molar, maxillary right first premolar, maxillary right canine, maxillary left second premolar, maxillary left first molar, mandibular left first premolar, mandibular right second molar, and mandibular right third molar) with bone loss consistent with generalized periodontal disease. Clinical examination showed gross caries and gross mobility of the maxillary right third molar, maxillary right first premolar, maxillary right canine, maxillary left second premolar, maxillary left first molar, mandibular right second molar and mandibular right third molar, as well as a fractured crown and an exposed endodontic metal post in the

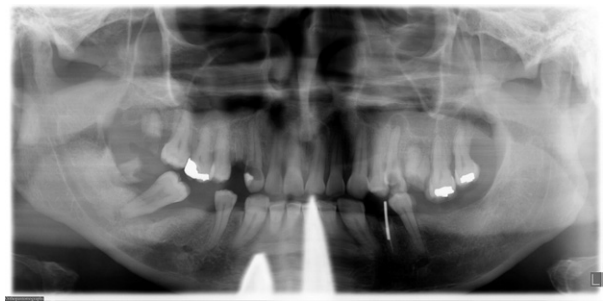

FIGURE 1. Preoperative panoramic radiograph (October 30, 2007).

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root of the mandibular left first premolar. The mandibular right second and third molars were painful on palpation.

After the radiographic and intraoral examination, at approximately 2:30 PM, informed consent was obtained for extraction of the mandibular left first premolar and mandibular right second and third molars. Approximately 7.2 mL of 2% lidocaine with 1:100,000 epinephrine was administered, and the mandibular right second and third molars were extracted with minimal force or disturbance of the surrounding tissues by use of an elevator and forceps. The surrounding granulation tissue was removed with a curette, and gauze was packed over the extraction sites. Attention was then drawn to the mandibular left first premolar. A full-thickness mucoperiosteal flap was reflected, and the tooth was extracted with the assistance of a surgical handpiece.

It was noted after the extractions that the patient had persistent oozing from the posterior sites. The patient's medical history was reviewed again, but he did not disclose any further information. On removal of the gauze, persistent oozing of the extraction sites was seen. No pulsatile bleeding was noted. The mandibular left first premolar extraction site was also seen to be oozing unusually. Absorbable gelatin powder (Gelfoam; Pharmacia, Kalamazoo, MI) was initially placed into the sockets of the mandibular left first premolar and mandibular right second and third molars to attempt hemorrhage control. Gauze was then placed over the sites and held firmly in place. This did not control the bleeding. Oxidized regenerated cellulose (Surgicel; Ethicon, Somerville, NJ) was then introduced into the sockets with gauze pressure over the sockets. The bleeding persisted, and microfibrillar collagen hemostat (Avitene; Davol, Cranston, RI) was obtained and placed into the sockets with placement of gauze soaked with topical thrombin of bovine origin (Thrombin-JMI; GenTrac, Middletown, WI) over the sockets. This was removed temporarily to facilitate placement of multiple sutures to obtain primary closure.

During the course of this treatment, the patient ingested blood, causing gastrointestinal distress. This resulted in an episode of emesis. Monitors were applied to the patient to obtain a second set of vital signs. The blood pressure was recorded as 140/83 mm Hg with a heart rate of 81 beats/min.

Despite aggressive local measures, the slow oozing continued. It was then decided to transport the patient to the emergency department (ED), where laboratory values could be obtained and appropriate medical management could be instituted because a significant bleeding diathesis was suspected. Shortly after the patient's arrival in the ED, it was observed that the bleeding was causing elevation of the floor of the mouth. Because of the threat to the patient's airway, anesthesiology was consulted.

Members of the oral and maxillofacial surgery, anesthesiology, and emergency medicine teams agreed that the patient should be intubated prophylactically for airway control. Before intubation, it was also noted that the edema had extended to the patient's right buccal and right submandibular spaces. Concurrently, the patient continued to have oozing intraorally from both the left and right extraction sites. At this time, the patient was sedated with propofol, and the airway was secured with an oral endotracheal tube. It was estimated that the patient lost 500 mL of blood before intubation. The intubation was performed approximately 3 hours after his extractions were completed.

The patient's sister, who accompanied him to the oral surgery clinic that afternoon, was consulted regarding her brother's medical history. Although she was not aware of any pre-existing medical condition, she reported that her brother had been an alcoholic for many years and, to her knowledge, was not under the care of any physician.

Baseline laboratory values obtained in the ED showed a platelet count of 21,000/mm^3 (normal range, 150-450,000/mm^3), hemoglobin level of 10.1 g/dL (normal range, 3.5-17.5 g/dL), and hematocrit level of 30% (normal range, 41%-53%). In addition, liver function enzyme tests showed an aspartate aminotransferase level of 308 U/L (normal range, 8-20 U/L), alanine transferase level of 86 U/L (normal range, 0-35 U/L), alkaline phosphatase level of 141 U/L (normal range, 20-70 U/L), and total bilirubin level of 2.0 mg/dL (normal, <1 mg/dL). The decision was made to transfer the patient to the medical intensive care unit (MICU) for observation and hematologic evaluation. Before admission to the MICU, the patient was transfused with 3 U of pooled platelets and 2 U of fresh-frozen plasma (FFP). Ten milligrams of vitamin K were administered intramuscularly. On admission to the MICU, the patient's physical examination (with the exception of his head and neck examination) was unremarkable. No hepatosplenomegaly was appreciated. His abdomen was soft and slightly distended, with no rebound or shifting dullness. His pulmonary and cardiovascular examinations were also normal.

Overnight in the MICU, an additional 5 U of platelets, 4 U of FFP, and 25 μg of desmopressin (DDAVP; Sinopep Pharmaceutical, Hangzhou, China) IV soluset were administered. Steroids were considered, but they were not administered because they are only useful in cases of autoimmune splenic sequestration and do not have a role in alcohol-induced sequestration. Piperacillin-tazobactam (Zosyn; Pfizer, New York, NY) and vancomycin (Vancocin; Alpharma Pharmaceutical, Fort Lee, NJ) antibiotic therapy was initiated for pulmonary coverage and for potential infection of a hematoma. Vital signs on admission to the MICU and for the next 24 hours were documented closely and are shown in Table 1.

Despite these various pharmacologic coagulation and hemostatic efforts, the patient continued to exhibit persistent oozing from the extraction sites. He subsequently lost an estimated 1,500 mL of blood in the following 12 hours. In consultation with the hematology team, additional rounds of FFP, packed red blood cells (PRBCs), DDAVP, and platelets were administered. Embolization of the bleeding was considered not to be an option because of the medical nature of the bleed. Aminocaproic acid (EACA) (Amicar;

Table 1. HEMODYNAMIC PARAMETERS (START TIME, OCTOBER 30, 6 PM)

	1 h	4 h	8 h	12 h	16 h	20 h	24 h	24-h Range
Blood pressure (mm Hg)	136/86	123/68	122/78	127/80	113/80	103/59	166/90	103-166/59-90
Heart rate (beats/min)	128	122	129	130	143	133	123	122-143
Temperature (°F)	101.6	103.2	101.8	100.9	101.5	101.8	101.3	100.9-103.2

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Table 2. FLUIDS GAINED AND LOST (START TIME, OCTOBER 30, 6 PM)

	1 h	4 h	8 h	12 h	16 h	20 h	24 h	24-h Totals
Fluids	600 mL normal saline (bolus)	600 mL normal saline (150 mL/h)	600 mL normal saline (150 mL/h)	600 mL normal saline (150 mL/h)	200 mL dextrose 5% in water (50 mL/h) 250 mL normal saline / aminocaproic acid (bolus)	200 mL dextrose 5% in water (50 mL/h) 300 mL normal saline / aminocaproic acid (50 mL/h)	200 mL dextrose 5% in water (50 mL/h) 200 mL normal saline / aminocaproic acid (50 mL/h)	3,150 mL normal saline 600 mL dextrose 5% in water
Blood products		1,900 mL	2,050 mL		3,300 mL			7,250 mL
Urine output	Not measured	260 mL initial Foley output	~50 mL/h	~38 mL/h	~30 mL/h	~25 mL/h	~37 mL/h	1,050 mL
Blood loss	~500 mL	Not measured	Not measured	1,500 mL	Not measured	Not measured	1,500 mL	~3,500 mL

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Pfizer) was introduced as both a systemic and topical antifibrinolytic agent. The patient was administered an initial bolus dose of 4 g and received an additional 10 g of EACA infused over a period of 10 hours. Topically, gauze soaked with 5 g of EACA was placed at the extraction sites.

Over the course of 24 hours, the patient's condition was extremely guarded because of the onset of hypovolemic shock. The patient exhibited a steady decrease in urine output and blood pressure, as well as a corresponding elevation in heart rate (Tables 1, 2). The patient was febrile throughout the night and had a maximum temperature of 103.2°F. Despite aggressive replacement therapy, the patient's complete blood count showed little improvement because of the ongoing bleeding and the apparent splenic sequestration of platelets (Table 3). Compounding the patient's deteriorating physiologic state, the extraction site continued to ooze throughout the night. An estimated total of 3,500 mL of blood loss was recorded over the 24-hour period after extractions.

Twenty-four hours after the extractions, there was no alleviation of the intraoral bleeding. The hematology team recommended the use of recombinant factor VIIa (rFVIIa), which was administered at a dose of 30 μg/kg. After the administration of rFVIIa, the patient showed a substantial decrease in intraoral oozing and hemostasis was achieved (Figs 2, 3). The first 24 hours' transfusions are documented in Table 4.

The patient's condition remained guarded as a result of the physiologic sequelae of distributive shock, and further transfusions were administered in an attempt to meet target goals (Table 5). The patient's vital signs were labile (Table 2), and he exhibited severe cervicofacial edema and anasarca (a generalized infiltration of edema fluid into subcutaneous connective tissue) that confined him to intubation in the MICU. A series of failed cuff-leak tests and computed tomography scans of the patient's airway clearly showed the severity of edema (Fig 4). A repeat computed tomography scan was completed before extubation (Fig 5). After 13 days, there was a clinical and radiographic decrease in upper airway edema, and the patient was extubated without complication on November 12, 2007, in the presence of members of the respiratory, anesthesia, MICU, otolaryngology, and oral and maxillofacial surgery teams. The patient was transferred from the MICU to the medicine service for further observation and management (Figs 6, 7).

The patient was ultimately diagnosed with ethanol-induced cirrhosis and secondary factor VII deficiency. The results of hepatic viral tests did not indicate a significant role for active viral infection in the patient's thrombocytopenia or liver dysfunction: hepatitis C, nonreactive; hepatitis B surface antigen, nonreactive; hepatitis B surface antibody, reactive (>10 IU/mL); hepatitis A antibody immunoglobulin, reactive; and hepatitis A antibody immunoglobulin, nonreactive.

The patient was discharged from the hospital 34 days postoperatively in stable condition with furosemide (Lasix; DAVA Pharmaceuticals, Fort Lee, NJ) therapy and a daily vitamin regimen including a multivitamin, folic acid, and thiamine.

Table 3. COMPLETE BLOOD COUNT AND COAGULATION VALUES—FIRST 24 HOURS (START TIME, OCTOBER 30, 6 PM)

	October 30: 0-6 h	October 30: 6-12 h	October 31: 12-18 h	October 31: 18-24 h (After Factor rVIIa Administration)
Platelets	21	66	88	64
Hemoglobin	10.1	9.4	6.4	6.5
Hematocrit	30	27.3	18.7	19.1
White blood cells	4.8	6.4	6.8	2.4
Fibrinogen			173	157.6
Prothrombin time	15.3	14.7	14.0	9.9
International Normalized Ratio	1.52	1.45	1.38	0.94
Partial thromboplastin time	28.7	28.7	25.0	31.0

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FIGURE 2. Post-intubation photograph (October 31, 2007). Note severe facial swelling.

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As of April 2009, the patient continues to be followed up by the hematology, medicine, and oral and maxillofacial surgery clinics on an outpatient basis. It should be noted that he no longer abuses alcohol and is in stable physical condition (Fig 8). As a result, his laboratory values have shown a considerable improvement in terms of hepatic function and hematologic parameters (Table 6). The patient is scheduled to undergo additional extractions of his remaining nonrestorable teeth in the operating room under general anesthesia with rFVIIa on standby.

Discussion

Alcoholism can result in deleterious multiorgan system disorders such as central nervous system defects, hepatitis, cirrhosis, and coagulopathies. In our case report of a patient with unidentified alcoholism with alcohol-induced liver hepatitis, occult coagulation disorders led to a near-fatal intraoral hemorrhage after multiple tooth extractions. This life-threatening bleed resulted in prophylactic intubation and MICU admission of the patient during which time a variety of pharmacologic agents and blood products were administered.

As exhibited in this case report, the physiologic manifestations of chronic alcohol consumption can cause detrimental effects on multiple major organ systems. In the case of our patient, alcoholic liver disease resulted in thrombocytopenia and a relative factor VII deficiency.

Clinical manifestations of hepatitis and cirrhosis include malaise, jaundice, and portal hypertension. Resultant laboratory values include elevated transaminase (alanine transferase/aspartate aminotransferase), elevated bilirubin, and decreased albumin levels. Alcohol reduces the intestinal absorption of glucose and vitamins, resulting in nutritional deficiencies and hematologic disorders.[1] Disorders of hematologic function are among the most commonly observed in the alcoholic patient. Thrombocytopenia develops as a combined result of bone marrow depression and hypersplenism. Coagulopathies develop because of impaired vitamin K absorption, thereby affecting the synthesis of clotting factors II, VII, IX, and X.[2] In fact, factor VII plasma levels are among the first of the coagulation factors to plummet in liver disease because of their short half-life of 3 to 6 hours.[3]

Although none was observed on examination, coagulation disorders may present themselves within the oral cavity. Oral manifestations include oral mucosal petechiae, hematoma, gingival bleeding, gingival jaundice, and glossitis.[1] In retrospect, if the patient had given a more accurate medical history, we may have identified him as an alcoholic, therefore altering the course of treatment. He acknowledged a history of social drinking of 2 to 3 drinks per week with no associated medical complications. A CAGE (criticized, annoyed, guilty, eye-opener) questionnaire (Table 7) may have elicited a different response from the patient. Once a clear history of alcoholism is established, the Child-Pugh classification (Tables 8, 9) may be used to determine the severity of liver disease and life expectancy.

Dental and surgical management of a known alcoholic or a patient with liver disease should include a thorough medical and dental survey. Specifically, one should investigate for hepatitis, jaundice, human immunodeficiency virus/acquired immunodeficiency syndrome, family history, medications, alco-

FIGURE 3. Photograph from November 2, 2007.

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Table 4. BLOOD PRODUCTS AND PROCOAGULANTS ADMINISTERED—FIRST 24 HOURS (START TIME, OCTOBER 30, 6 PM)

	October 30, Tuesday: 0-6 h	October 31, Wednesday			24-h Totals
		6-12 h	12-18 h	18-24 h	
FFP	2 U	4 U	6 U		12 U
Pooled platelets	6 U	2 U	2 U		10 U
Packed red blood cells		2 U	4 U		6 U
DDAVP (desmopressin acetate)		25 μg	25 μg		50 μg
Vitamin K	10 mg intramuscular	5 mg IV soluset	10 mg IV soluset		25 mg
Amicar (EACA)			4 g IV soluset in 250 mL normal saline (bolus) 5 g IV soluset in 250 mL normal saline (1 g/h) Gauze soaked with 5-g topical application	5 g IV soluset in 250 mL normal saline (1 g/h) Gauze soaked with 5-g topical application	14 g systemic 10 g local/topical
rFVIIa				2.4 mg (30 μg/kg)	2.4 mg
Rho (D) immune globulin				300 μg	300 μg

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hol intake, recreational drug use, sexual history, and bleeding tendencies. Patient testing should include complete blood count, prothrombin time, International Normalized Ratio, partial thromboplastin time, and liver function tests. Any abnormal values detected should be reviewed, and a primary care physician should be consulted before any surgical treatment. A platelet count of 100,000/μL is desirable for major surgical procedures, and minor oral surgical procedures may be performed with little risk with a platelet count of 50,000/μL.

Platelet transfusion should be considered before surgery in the thrombocytopenic patient. One unit of platelets yields an average increase of 5,000 to 8,000/μL in 1 hour. Approximately one third of platelets are sequestered in the spleen; therefore, the surgeon should be aware that patients with splenomegaly will sequester these transfused platelets more rapidly. Fifty-eight percent of platelet transfusions administered to splenomegalic patients produce a corrected count increment of less than 7,500.[4]

Table 5. BLOOD PRODUCTS AND PROCOAGULANTS ADMINISTERED—HOSPITAL DAYS 3-7 (START DATE, NOVEMBER 1)

	November 1, Thursday: 24-48 h	November 2, Friday: 48-72 h	November 3, Saturday	November 4, Sunday	November 5, Monday
FFP		2 U	2 U		
Pooled platelets	1 U	8 U	1 U		
Packed red blood cells			1 U		
DDAVP					
Vitamin K	10 mg	10 mg	10 mg	10 mg	10 mg
Amicar (EACA)	20 g IV soluset in 1 L dextrose 5% in water (1 g/h) Gauze soaked with 5-g topical application 3 times per day	5 g IV soluset in 500 mL dextrose 5% in water (500 mg/h) Gauze soaked with 5-g topical application 3 times per day			
rFVIIa					
Rho (D) immune globulin					300 μg

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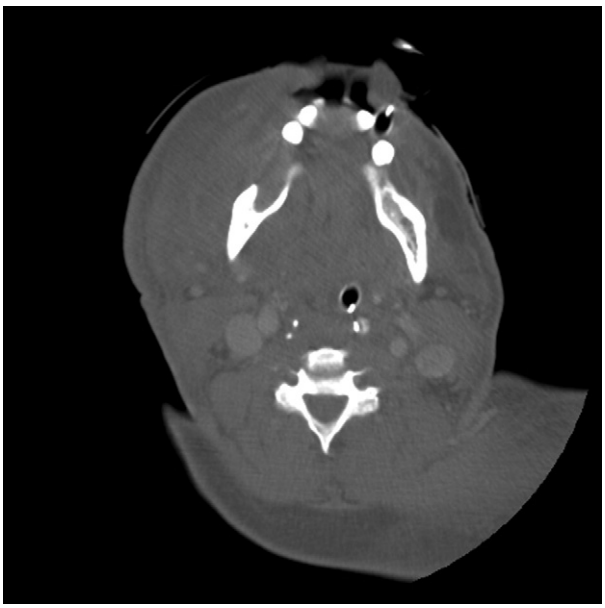

FIGURE 4. Computed tomography scan from November 5, 2007. Note lack of air space around endotracheal tube.

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In cases in which platelet transfusion appears inadequate, systemic pharmacologic intervention may be necessary. Recommended agents include DDAVP and lysine analogs (EACA and tranexamic acid). DDAVP (desmopressin) is a synthetic analog of antidiuretic hormone that increases the plasma levels of factor VIII and stimulates the release of von Willebrand factor from platelets. The recommended intravenous dose of desmopressin to decrease bleeding time is 0.3 μg/kg. EACA and tranexamic acid are antifibrinolytics that inhibit plasminogen conversion to plasmin. Thus they prevent clot lysis. The recommended loading dose of EACA is approximately 10 g, followed by a maintenance dose of 1 g/h. Tranexamic acid is approximately 10 times more potent than EACA. The recommended loading dose of tranexamic acid is also approximately 10 g, followed by an infusion of 250 mg/h.[5]

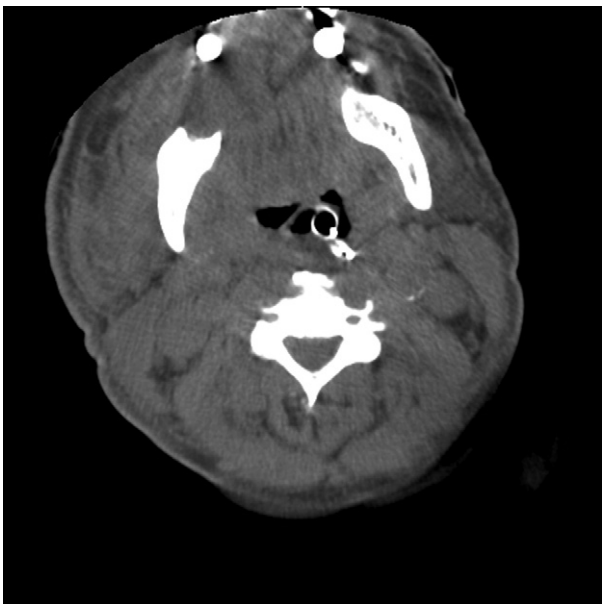

FIGURE 5. Computed tomography scan from November 11, 2007. Note air space in vallecula and piriform sinuses around endotracheal tube.

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FIGURE 6. Post-extubation extraoral photograph from November 16, 2007.

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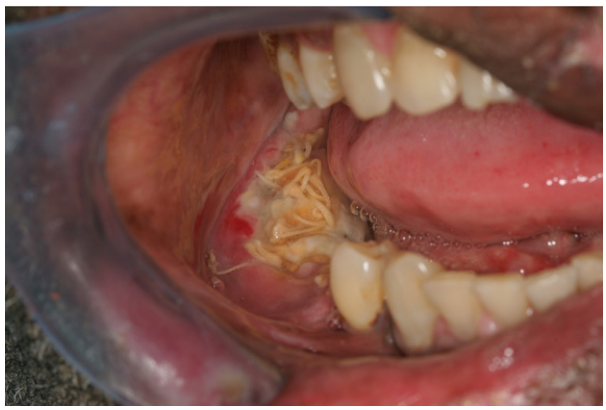

FIGURE 7. Post-extubation intraoral photograph from November 16, 2007.

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FIGURE 8. Five-month follow-up photograph from February 27, 2008. Note extreme change in facial form compared with Figure 2.

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In cases of acute hemorrhage in which local measures failed to promote hemostasis, alternate methods of coagulation therapy have been suggested in the literature. NovoSeven (Novo Nordisk, Princeton, NJ), a recombinant human coagulation factor VIIa (rFVIIa), is a vitamin K–dependent glycoprotein structurally similar to human plasma-derived factors VIIa. It is thought to promote local hemostasis by activating the extrinsic pathway of the coagulation cascade on the surface of activated platelets at the site of tissue injury (NovoSeven package insert). NovoSeven was initially developed for the treatment of bleeding in patients with hemophilia who had antibodies inactivating factor VIII or IX.[6] Previous FVIII and FIX replacement therapies for patients with hemophilia A and B resulted in the production of alloantibodies (inhibitors), which rendered the therapy ineffective. It was found that recombinant activated factor VII was structurally similar to human plasma-derived factors VIII and IX and showed an efficacy of 90% to 95% in the treatment of bleeding episodes in patients with congenital hemophilia without the production of alloantibodies.[7]

Food and Drug Administration–approved usage for rFVIIa is limited to the treatment and prophylaxis of bleeding episodes in patients with a known hemophilia A or B inhibitor or deficiency. Anecdotal evidence and case reports have shown success in the treatment of life-threatening hemorrhage in patients with thrombocytopenia, trauma, and liver disease.[8] rFVIIa does not induce systemic activation of the coagulation system and is not affected by circulating inhibitors, and its in vitro

Table 7. CAGE (CRITICIZED, ANNOYED, GUILTY, EYE-OPENER) QUESTIONNAIRE

Are you criticized about the amount of alcohol you consume?
Are you annoyed or angered by this criticism?
Do you feel guilty about your drinking?
Do you need an "eye opener" in the morning to get you going or to relieve a hangover?

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Table 6. COMPLETE BLOOD COUNT, LIVER FUNCTION TESTS, AND COAGULATION VALUES

	October 30, 2007: Admission	November 1, 2007	December 3, 2007: Discharge	March 7, 2008: Follow-Up	March 19, 2009
Platelets (/μL)	21,000	13,000	72,000	87,000	108,000
Hemoglobin (g/dL)	10.1	8.8	9.9	13.1	15.0
Hematocrit (%)	30	24.7	29.2	36.6	44
White blood cells (/μL)	4,800	4,300	9,800	5,900	5,800
Fibrinogen	173.0	Clotting		222.4	
Prothrombin time	15.3	Clotting	19.7	13.5	12.3
International Normalized Ratio	1.52	Clotting	2.01	1.32	1.21
Partial thromboplastin time	28.7	Clotting	33.4	30.9	29.0
Aspartate aminotransferase	308	319	41	54	41
Alanine transferase	86	72	26	34	29
Total bilirubin	2.0	9.5	1.4	1.5	1.0
Albumin	2.3	2.1	2.0	3.4	4.0

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Table 8. CHILD-PUGH CLASSIFICATION

Parameter	Points Assigned 1	2	3
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dL)	<2	2-3	>3
Albumin (g/dL)	>3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds over control	1-3	4-6	>6
INR	<1.8	1.8-2.3	>2.3
		Grades	Grades
Encephalopathy	None	1-2	3-4

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Table 9. CHILD-PUGH SURVIVAL

Grade	Points	1-yr Patient Survival (%)	2-yr Patient Survival (%)
A: Well-compensated disease	5-6	100	85
B: Significant functional compromise	7-9	80	60
C: Decompensated disease	10-15	45	35

A total score of 5-6 is considered grade A (well-compensated disease); 7-9 is grade B (significant functional compromise); and 10-15 is grade C (decompensated disease). These grades correlate with 1- and 2-year patient survival.

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production eliminates the risk for transfusion infection. These characteristics of rFVIIa make it an appealing option for treatment of bleeding disorders outside the realm of hemophilia.[9]

The hypothetical interaction between platelets and rFVIIa forms the basis of the therapeutic use of high-dose factor VIIa in treating hemophilic patients with inhibitory antibodies.[10] High-dose factor VIIa has been found to shorten the bleeding time in thrombocytopenic patients who bleed despite receiving platelet transfusions.[11] Depending on the cause for the thrombocytopenia, often a transfusion will increase the quantity of platelets available to begin the hemostatic process. In many cases of thrombocytopenia, however, the cause is splenic sequestration; transfusions will be ineffective or only partially effective in overcoming this problem. In vitro evidence shows that rFVIIa increases thrombin generation on the activated platelet surface and shortens the lag phase of further platelet activation.[6]

Several case reports exist showing success in reducing major blood loss in patients with trauma by administering rFVIIa. Geeraedts et al[12] report a complete cessation of uncontrollable hemorrhaging in 6 of 8 patients who sustained blunt force trauma after rFVIIa therapy and a substantial decrease in the amount of transfused blood products as a result of the hemostasis.

The use of rFVIIa for the treatment of spontaneous or surgical bleeding has shown to be efficacious in obtaining hemostasis and decreasing the amount of blood products transfused in a number of case reports and clinical trials.[13] It should be noted that dosages and timing of administration are not standardized, and off-label usage of this potent hemostatic agent should be done with caution because detrimental thrombotic events, though uncommon, can occur. In the case of our patient, who had a persistent bleed after a routine tooth extraction, recombinant FVIIa was used when other local and systemic methods of hemostasis had failed.

Off-label use of rFVIIa has grown extensively since the original report of a nearly miraculous recovery of a gunshot victim given rFVIIa after becoming coagulopathic.[14] Indeed, a registry is now operational to record the results of hemorrhage control using rFVIIa off label.[15] A number of randomized controlled trials have been carried out using rFVIIa or placebo in diverse coagulopathic patients without hemophilia. A meta-analysis of 22 such trials involving 3,184 patients showed that additional transfusions were significantly less likely to be required in patients receiving rFVIIa than those receiving placebo.[16] However, use of rFVIIa in bleeding dental patients has seldom been reported.

Life-threatening hemorrhage after a tooth extraction is a rare and frightening event. The most effective preparation for such a catastrophic event is prevention. It may be necessary despite all attempts at prevention to use alternate means to control such bleeding. This may include both local and systemic measures to achieve hemostasis.

It is imperative to obtain a clear and comprehensive medical history on all patients who are to receive surgical treatment. Vital signs should be recorded, and one must be able to recognize when a patient requires further preoperative testing. The risks and benefits of more extensive preoperative testing should also always be considered. The urge to obtain screening-type preoperative tests must be tempered by concerns about their sensitivity and specificity. In addition, crucial is the ability to sense a patient's resistance to present a complete medical history, as well as to be able to determine the safety of proceeding with surgical treatment with the information at hand. In our case presentation, despite our patient's severe coagulopathy, he did not manifest any overt clinical signs or symptoms of liver disease.

The use of rFVIIa was initiated in this case after all local and systemic agents failed to alleviate the bleed-

ing. Although rFVIIa is not available in a private office setting, clinicians should be aware of its use to control hemorrhage. Originally developed for the prophylactic treatment of hemophilic patients with inhibitors, rFVIIa has been shown to be successful in a number of emergent bleeding situations in several anecdotal case reports. It should also be noted that the use of rFVIIa in cases of spontaneous and surgical bleeding is not Food and Drug Administration approved, and anecdotal evidence suggests varying therapeutic doses and possible uses for this life-saving pharmacologic agent. This is one of only a handful of case reports of factor VIIa being used in the oral surgery setting for either prevention[17,18] or treatment[19,20] of postsurgical hemorrhage.

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