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Active Bleeding in Uremic Patients after Temporary Catheter Implantation: A Case Report and Literature Review

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Abstract

Patients with uremia often develop bleeding diatheses at cutaneous or incisional sites, intra-abdominal or intracranial hemorrhages. We report active bleeding in a uremic patient after she received a temporary catheter for hemodialysis therapy. Active bleeding was considered to be due to platelet dysfunction, diagnosed by prolonged skin bleeding time. Her spontaneous bleeding gradually vanished after receiving blood transfusions with cryoprecipitate. The assessment of skin bleeding time in uremic patients with active bleeding is a crucial step to identify platelet dysfunction.

Keywords: Hemorrhage; Platelet dysfunction; Uremia

Introduction

Uremia is a serious subsequent complication of renal function deterioration, mostly caused by hypertension and diabetes [1]. Uremia causes a medical syndrome associated with fluid overloading, electrolyte and hormone imbalances, and metabolic aberrations. During the acute stage, hyperkalemia, metabolic acidosis, and generalized edema, especially pulmonary edema, cause cardiovascular diseases to worsen, which can lead to death. During the chronic phase, the macromolecule uremic toxins, such as parathyroid hormone, β2-microglobulin, polyamines, and advanced glycation end products, are considered to result in long-term organ damage in uremic patients [2].

If not properly treated, uremia can cause uremic bleeding, which leads to serious complications, including coma, seizures, cardiac arrest, and even death. Progressively severe uremia can cause clinically spontaneous bleeding, such as gastrointestinal hemorrhage, intracranial bleeding, and internal bleeding.

Uremic bleeding is due to untreated uremic toxins inhibiting intrinsic platelet anomalies and an impaired platelet–vessel wall interface, eventually leading to life-threatening conditions [3,4]. Herein, we describe a woman presenting with uremic bleeding and a literature review on the topic.

Case Presentation

A 44-year-old woman presented to the emergency department with nausea, vomiting, and shortness of breath. On examination, her mental status was drowsy. Her blood pressure was 210/100 mmHg, heart rate 103 beats/min, respiratory rate 28 breaths/min, and body temperature 37.1°C. The physical examination showed disorientation, incoherent speech (Glasgow coma scale E3V4M5), bilateral chest breathing rales, and pitting edema of both legs. A chest radiograph displayed cardiomegaly, pulmonary edema, and bilateral pleural effusions.

Table 1 Clinical characteristics in a uremic patient with spontaneous bleeding after temporary catheter implantation.

Variables	Day 1	Day 2	Day 7	Day 14
Blood Cell and Coagulation				
Hemoglobin (g/dL)	8	7.5	8.9	8.8
Hematocrit (%)	25.2	24.2	26.5	28.1
Platelet (103/mm^3)	230	207	286	263
aPTT (sec)	25.7	26	24.5	25.4
PT (sec)	10.9	26	10.7	10.2
Bleeding time (sec)	ND	12	7.5	ND
Fibrinogen (g/L)	2.08	3.66	3.97	3.51
D-dimer (mg/L)	5.15	4.81	4.2	3.22

Serum Biochemistries				
BUN (mg/dL)	>134.43	33.13	47.88	31.36
Creatinine (mg/dL)	15.51	10.39	7.23	8.82
AST (U/L)	24	33	32	16
ALT (U/L)	40	64	40	17
Albumin (g/dL)	3.35	3.32	3.51	4.1
Potassium (mmol/L)	5.64	4.1	4.38	4.56
Sodium (mmol/L)	137.2	141.7	141.1	138.2
Calcium (mg/dL)	8.8	8.4	8.46	8.92
phosphorus (mg/dL)	8.36	5.24	1.09	1.42
BNP (pg/mL)	>25000	>25000	12319	5046
Urine				
Protein	3+	2+	2+	2+
Occult blood	+-	-	+-	-
WBC (/HPF)	1–3	0–1	1–3	0–1
RBC (/HPF)	-	-	0–1	0–1
Daily urine (mL/day)	300	500	600	750
Arterial Blood Gas				
pH	7.424	7.397	7.445	7.382
PaO_2 (mmHg)	62.1	72.6	80.6	82.3
$PaCO_2$ (mmHg)	26.5	32.5	39.5	38.1
HCO_3 (mol/L)	17.1	19.6	26.7	24.8
BE (mol/L)	-6.1	-4.2	3	2.5
Vital Signs				
PR (bpm)	112	80	70	76
BP (mmHg)	160/110	170/110	135/80	140/80
RR (breaths/min)	28	24	17	16
BW (kg)	64.05	63.35	60.25	59.5
BT (°C)	36.4	36.3	36.4	36.6

Laboratory data are shown in **Table 1**. Uremia was diagnosed and treated with emergent hemodialysis (HD) after performing implantation of a temporary HD catheter over the right internal jugular vein. With supportive treatment, intermittent gingival bleeding and blood oozing over the insertion area of the temporary HD catheter was noted. On Day 1, platelet counts, prothrombin time, and activated partial thromboplastin time were at normal levels. On Day 2, a prolonged skin bleeding time was detected by using the Duke method. Afterwards, her bleeding symptoms gradually disappeared following blood transfusion with cryoprecipitate.

Discussion

The assessment of skin bleeding time in uremic patients with active bleeding is a crucial step to identify platelet dysfunction. With conservative and compression therapy, this patient with uremia had been oozing blood from the catheter incision site after receiving a temporary HD catheter. After a series of examinations, a prolonged skin bleeding time was detected; the diagnosis was platelet dysfunction. Her spontaneous bleeding gradually vanished after receiving blood transfusions with cryoprecipitate.

Platelet dysfunction in HD patients is one of the major complications of uremic bleeding [5]. Defective platelet function is caused by both intrinsic platelet anomalies and an impaired platelet–vessel interface. Ordinarily, the platelets respond to vascular damage with platelet activation, recruitment, adhesion, and aggregation. In uremia, increased endogenous fibrin fragments combine with GP II b-IIIa [6], further interfering in the platelet and von Willebrand factor interface; finally, reduced platelet aggregation causes bleeding diatheses [7]. In this case, the impaired platelet–vessel wall aggregation of platelets at the site of the vascular injury had caused spontaneous bleeding after a vascular procedure [8].

Traditionally, uremic platelet dysfunction has been recognized by a prolonged skin bleeding time [9]. Recently, other testing procedures, such as whole blood platelet aggregation, platelet function analyzer, thromboelastograph, and cone platelet analyzer have been reported to identify the status of platelet dysfunction. However, these assessments were poor predictors of prolonged skin bleeding time [10]. Soyoral et al. suggested that skin bleeding time could be an appropriate test for evaluating platelet dysfunction and predicting bleeding diatheses in uremic patients [11].

Dialysis can partially treat platelet dysfunctions and coagulopathies in patients with uremia, but it cannot fully eliminate these bleeding diatheses. Alternatively, cryoprecipitate can be used for treating active hemorrhages [12]. Plasma extracts rich in von Willebrand factor, factor VIII, and fibrinogen rapidly resolve bleeding diatheses in uremic patients. Additionally, desmopressin acetate and conjugated estrogen can be used to treat uremic bleeding in platelet dysfunction [13,14]. Achieving a hematocrit of 30% ameliorates bleeding diatheses in patients with uremia [15].

Conclusion

In conclusion, we report active bleeding in a uremic patient after she received a temporary HD catheter. Active bleeding was considered to be due to platelet dysfunction, diagnosed by prolonged skin bleeding time. Thus, in addition to considering platelet depletion and coagulopathy, tests for platelet dysfunction should be part of the work-up in uremic patients with bleeding diatheses.

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