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【 CASE REPORT 】

Vasovagal Reaction and Ischemic Colitis Following Blood Donation

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Abstract:
Vasovagal reactions are the most common type of adverse reaction after blood donation; however, there are no reports of ischemic colitis as an adverse reaction after blood donation. A previously healthy 55-year-old woman suffered loss of consciousness at the end of her first plasma donation. She was diagnosed with a vasovagal reaction and received hydration. However, she developed persistent left flank pain and watery diarrhea, followed by bloody diarrhea. Abdominal computed tomography confirmed ischemic colitis. She was asked to fast and was eventually discharged 7 days later. We should consider the possibility of ischemic colitis if patients develop persistent abdominal pain after transient hypotension, such as that observed during a vasovagal reaction.

Key words: blood donation, hypotension, ischemic colitis, vasovagal reaction

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Introduction

Although blood donation is considered safe, the prevalence of adverse reactions, such as vasovagal reactions (VVR), hematomas, citrate reactions, and nerve injuries, is approximately 1% (1). However, ischemic colitis has not been recognized as an adverse reaction of blood donation. We herein report a case of ischemic colitis after a blood donation-related VVR.

Case Report

A previously healthy 55-year-old woman made her first plasma donation, having donated whole blood more than 20 times without complication. She had no relevant medical history and was not taking any medications. She was 158.2 cm in height, weighed 52.8 kg, and her BMI was 21.1 kg/m^2. At the end of the plasma donation session, she reported a feeling of discomfort and subsequently experienced a loss of consciousness accompanied by convulsions for 10 seconds. Her heart rate and blood pressure were measured immediately after the event and were found to be 62 beats/min and 113/82 mmHg, respectively. She was diagnosed with VVR and received hydration intravenously followed by observation; however, she developed left flank pain and began vomiting 1 hour later. She presented to our hospital 4 hours after the onset of the VVR, due to an increase in abdominal pain.

On arrival, her temperature was 36.7°C, her heart rate was 85 beats/min, and her blood pressure was 126/68 mmHg. A physical examination revealed left abdominal tenderness without signs of peritonitis. The laboratory findings are shown in Table. Electrocardiography and cardiac ultrasonography revealed no abnormalities. She defecated after arrival, and her stool was small and solid. Metoclopramide (10 mg) and scopolamine butylbromide (20 mg) were administered intravenously but she continued to have flank pain. Furthermore, both intravenous famotidine (20 mg) and d-chlorpheniramine maleate (20 mg) were ineffective for relieving her abdominal pain. Thus, we performed abdominal CT on the day of admission. Non-contrast CT revealed pericolonic fat stranding along with bowel wall thickening at the splenic flexure, both of which were suggestive of ischemic

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Table. **Laboratory Findings on Admission.auto-c**

Blood cell counts		Biochemistry		Venous blood gas	
WBC	15.3 10^3/μL	ALB	4.3 g/dL	pH	7.521
RBC	523 10^4/μL	UN	14.7 mg/dL	pCO_2	26.9 mmHg
Hb	15 g/dL	CRE	0.56 mg/dL	HCO_3	22 mmol/L
Ht	43.7 %	Na	141 mmol/L	BE	0.6 mmol/L
PLT	24.1 10^4/μL	K	3.6 mmol/L	AG	14.2 mmol/L
		Cl	105 mmol/L	Ca^{2+}	1.12 mmol/L
Blood coagulation		Ca	9.1 mg/dL	Lac	32 mg/dL
PT	11.7 s	TB	0.81 mg/dL		
APTT	25.8 s	AST	16 U/L		
D-dimer	0.4 μg/mL	ALT	9 U/L		
		LD	198 U/L		
		CK	59 U/L		
		CRP	0.02 mg/dL		

WBC: white blood cell count, RBC: red blood cell count, Hb: hemoglobin, Ht: Hematocrit, PLT: platelet, PT: prothrombin time, APTT: activated partial prothrombin time, ALB: albumin, UN: blood urea nitrogen, CRE: creatinine, Na: serum sodium, K: serum potassium, Cl: serum chloride, Ca: serum calcium, TB: total bilirubin, AST: aspartate amino transferase, ALT: alanine amino transferase, LD: lactic dehydrogenase, CK: creatine kinase, CRP: C-reactive protein, BE: base excess, AG: anion gap, Ca^{2+}: ionized serum calcium, Lac: lactic acid

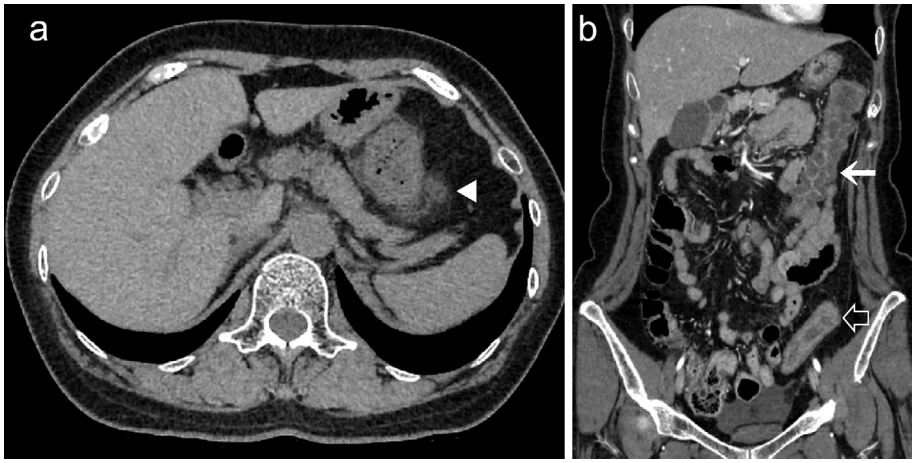

Figure. **(a) Axial computed tomography scan on day 1 and (b) coronal on day 2 showing pericolonic fat stranding of the splenic flexure (arrowhead), thumbprinting of transverse colon (white arrow), and bowel wall thickening of sigmoid colon (open white arrow).**

colitis (Figure a).

The patient was asked to fast and cefmetazole sodium (1 g) was intravenously administered twice on day 1. She suffered watery diarrhea 9 hours after the VVR, followed by bloody diarrhea 16 hours after the VVR; however, a stool culture was normal. On the second day of admission, contrast abdominal CT revealed bowel wall thickening and thumbprinting from the transverse colon to the sigmoid colon (Figure b). There was no occlusion of the inferior mesenteric artery or the superior mesenteric artery. These findings helped confirm the diagnosis of ischemic colitis. On the fourth day of admission, her abdominal pain resolved, and fasting was ceased. The patient recovered and was discharged 7 days after admission. We planned to perform colonoscopy during admission, but the patient refused to undergo further evaluation.

Discussion

The present case highlights two important issues. First, a blood donation-related VVR may cause ischemic colitis. Ischemic colitis is caused by a diminution of the colonic blood supply, resulting from hypotension and hypovolemia, leading to systemic hypoperfusion (2, 3). This is the first reported case of ischemic colitis caused by a VVR; however, ischemic colitis caused by hypotension has been reported previously, particularly in cases of anaphylactic shock (4-6). In this case, we did not detect hypotension; nevertheless, a

loss of consciousness accompanied by convulsions suggested transient hypotension. We initially considered the patient's abdominal pain to be a symptom of a citrate reaction or citrate allergy; however, she had no rash, her calcium ion levels were normal, and antihistamines were not effective. Moreover, the patient had not been treated with vasopressors, which are known to occasionally cause intestinal ischemia (2, 3). In addition, the patient had no risk factors for ischemic colitis (e.g., hypertension, hyperlipidemia, diabetes, or constipation). With the exception of transient hypotension during VVR, there was no clinical evidence of other known causes of ischemic colitis. Second, CT is a useful diagnostic modality for detecting early ischemic colitis. Typically, ischemic colitis develops in elderly patients, and presents with rapid-onset abdominal pain and diarrhea, followed by hematochezia within 1 day of the onset of abdominal pain. Without changes in the condition of the feces, the diagnosis is clinically challenging. In this case, CT findings of pericolonic fat stranding at the splenic flexure led to a diagnosis of ischemic colitis. Ischemic colitis most often occurs at watershed areas, such as the splenic flexure and the rectosigmoid junction (2, 3).

Vasovagal syncope has been the main focus of blood donor safety and the risk factors for VVRs have included blood donation for first-time donors, young age, female sex, lower BMI, short sleep time, and apheresis donation (1). In this case, we believe that first time plasma donation was an important cause of the patient's VVR. Even though the donor had frequently donated whole blood without adverse reactions, this was the first component donation, which may cause a VVR.

Conclusion

Transient hypotension, such as with a VVR, may cause ischemic colitis. VVRs are the most common adverse reactions during blood donation and do not usually require hospitalization. However, we should consider the possibility of ischemic colitis if patients develop persistent abdominal pain after a VVR.

The authors state that they have no Conflict of Interest (COI).

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