□ CASE REPORT □

Fatal Splenic Laceration in a Young Woman Caused by Idiopathic Isolated Splenic Vein Thrombosis

Tetsuya Nomura¹, Natsuya Keira¹, Yota Urakabe¹, Daisuke Naito¹, Satoko Enomoto¹,
Susumu Nishikawa¹, Hiroaki Matsubara² and Tetsuya Tatsumi¹

Abstract

A 38-year-old woman died of hemorrhagic shock caused by idiopathic bleeding in the abdominal cavity. At autopsy, more than 5,000 mL hemoperitoneum was detected. There was no remarkable bleeding site except for a small tear in the surface of the spleen. Microscopic examination suggested that isolated splenic vein thrombosis induced coagulative necrosis of the spleen and subsequently caused splenic laceration. Whenever a case of hemoperitoneum is encountered, splenic rupture should be included in the differential diagnosis; it is imperative to manage such cases as promptly as possible.

Key words: hemoperitoneum, splenic laceration, splenic vein thrombosis

(Inter Med 48: 907-910, 2009)

(DOI: 10.2169/internalmedicine.48.1843)

Introduction

Isolated splenic vein thrombosis (SVT) is a rare pathology; it is reported to usually induce left side portal vein hypertension, which sometimes results in bleeding from gastric varices (1). However, trauma is the most frequent cause of splenic rupture, and very few reports have previously demonstrated SVT-induced splenic rupture (2).

Case Report

A 38-year-old woman complained of epigastralgia after supper and called for an ambulance about 10 hours after the onset of symptoms. When paramedics arrived, her face was pale and she was in a cold sweat. Her consciousness was clear, but her blood pressure was 76/46 mmHg and her pulse rate was 120/min with a regular rhythm. In the ambulance her consciousness level rapidly decreased, and her pulse became undetectable. When transported to our emergency room, electrocardiogram demonstrated pulseless electrical activity (PEA). Advanced cardiac life support (ACLS) was quickly carried out and she was eventually resuscitated with approximately a 10-minute practice of ACLS.

After resuscitation her systolic blood pressure was about 40 to 50 mmHg and her pulse rate was about 110/min with a sinus rhythm even under maximum dosage of dopamine. She was in comatose state (Glasgow coma score =3). APACHE II (Acute Physiology Age and Chronic Health Evaluation) score was calculated at 38 on intensive care admission. Her conjunctivae were quite anemic and her abdomen was remarkably distended. Plain abdominal computed tomography (CT) demonstrated massive peritoneum and a large mass beside the uterus (Fig. 1). There were no major findings in the brain, other thoracic or abdominal organs. Laboratory study showed severe anemia (Table 1). Although she was transfused with 8 units of red cell concentrate in mannitol-adenine-phosphate (RC-MAP) and 750 mL of 5% human serum albumin in all, her low blood pressure persisted and she never regained consciousness. Because she had no relatives and lived alone, there was very limited information about her personal background available. Exams and physical findings suggested that massive hemoperitoneum induced hemorrhagic shock. It was important to clarify the bleeding site and to stop the bleeding as promptly as possible. Transcatheter arterial embolization (TAE) to determine the bleeding site is usually the first choice in such a clinical setting. However, predictive mortality from

¹Department of Cardiovascular Medicine, Nantan General Hospital, Nantan and ²Department of Cardiovascular Medicine, Kyoto Prefectural University School of Medicine, Kyoto

Received for publication November 6, 2008; Accepted for publication February 16, 2009

Correspondence to Dr. Tetsuya Nomura, t2-ya@za2.so-net.ne.jp

Figure 1. Plain abdominal CT on admission demonstrated massive peritoneum (A: arrows) and a large mass adjacent to the uterus (B: arrows).

Table 1. A Case of Splenic Rupture in a Young Woman

WBC	22550 / μ L	LDH	420 U/L
RBC	2.01 10^6 / μ L	ALP	107 U/L
Hgb	6.2 g/dL	T-Bil	0.2 mg/dL
Hct	19.8 %	CRP	0.1 mg/dL
PLT	190 10^3 / μ L	BS	522 mg/dL
BUN	17.3 mg/dL	TropT	negative
Cre	1.16 mg/dL	hCG	<0.1 ng/mL
Na	148 mEq/L		
K	6.9 mEq/L	(10L/min O ₂ inhaled)	
Cl	104 mEq/L	pH	6.669
Amy	61 IU/L	pCO ₂	23.8 mmHg
CPK	36 U/L	pO ₂	591.1 mmHg
AST	158 U/L	HCO ₃	2.7 mmol/L
ALT	150 U/L	BE	-34.3 mmol/L

APACHE II score was relatively high and neurological prognosis was also less promising even if she survived. Her surety did not desire for further examination and intensive treatment. Over time, multiple organ failure induced by systemic lower perfusion emerged and she died on day 3 of hospitalization.

To investigate the cause of death, autopsy was performed with the permission of her surety. More than 5,000 mL hemoperitoneum was confirmed. The spleen weighed 350 g, was moderately swollen and the capsule was stretched and partially torn (Fig. 2A). Microscopic findings demonstrated splenic infarction characterized by coagulative necrosis and the capsule was partially lacerated (Fig. 2B, C). In particular, the splenic vein was filled with coagulative thrombi which uniformly consisted of funicular fibrin and rich red blood cells. They were mostly anchored to vessel walls which is in contrast with postmortem coagulation characterized by mold formation in the vessels (Fig. 2D). Large leiomyoma of the uterus measuring 10×10×9 cm was recog-

nized, but there was no bleeding from the tumor. There was congestion of multiple abdominal organs secondary to hemorrhagic shock. Splenic small laceration was the only finding that indicated bleeding into the abdominal cavity. The pathological diagnosis was idiopathic isolated splenic vein thrombosis which induced splenic infarction, followed by splenic laceration, which resulted in severe hemorrhagic shock.

Discussion

The cause of isolated splenic vein thrombosis (SVT) is reported to be frequently derived from pancreatic diseases such as pancreatitis, tumor, abscess, and cyst (3). Causes other than these range widely, including trauma, lymphoma, sarcoma, metastatic tumor, venous anomaly, renal diseases, and clotting system disorder (4). While splenic rupture often occurs in cases of abdominal injury, endogenous occurrence of splenic rupture is not often encountered. Furthermore SVT-induced splenic rupture is one of the rarest of possible causes (2, 5).

The clinical course described here is so rare that the pathophysiology is not fully elucidated and many mysterious findings remain to be resolved. What induced isolated splenic vein thrombosis in such a young woman? Previous papers have reported that various backgrounds can become the basis of thrombosis, for example, some disorders of the clotting system (2), the influence of abdominal compression and vibration (6), or taking drugs that induce hypercoagulability such as steroids or various hormonal medications. Here, was as much as 5,000 mL of bleeding from such a tiny laceration of the spleen? It was an undeniable fact that no bleeding point other than the splenic laceration was found in the abdominal cavity. Therefore, we suppose the existence of a clotting system disorder again from such a clinical setting. Because she had no relatives and lived alone, we could not obtain detailed information about this patient. We did not store a specimen of the patient's blood serum, and her clinical background remains merely a speculation.

The causal relationship between splenic vein thrombosis and splenic rupture remains controversial. Some investiga-

Figure 2. The findings at autopsy. The spleen was congestive and moderately enlarged. The capsule was stretched and partially broken (A: arrows). Microscopic image showed a small laceration of the capsule (B: arrows). Highly magnified specimen demonstrated splenic infarction characterized by coagulative necrosis (C) and coagulative thrombi filling the splenic veins (D: arrows).

tors have reported that widely distributed thrombi from the splenic vein to the portal vein could be related to splenic rupture (5). That is to say widely distributed thrombi tend to induce hypoplasia of the collateral flow followed by congestion of the spleen. Circulatory disorders in the spleen induce splenic infarction and finally produce rupture of the splenic capsule. In the present case, we recognized relatively localized splenic vein thrombi as well as hypoplasia of the collateral veins, which means that the splenic vein occlusion was completed so suddenly that there was no time for collateral veins to be organized. This fact is consistent with the clinical course of our patient in terms of the acute onset.

APACHE II score, a severity of disease classification sys-

tem, indicates a general measure of severity of disease. An increasing score is closely correlated with the subsequent risk of hospital death (7). Because of the relatively high APACHE II score in our patient we speculated that the possibility of saving her life was very low. Moreover, it is reported that a low Glasgow coma score in early neurological evaluation of comatose survivors after cardiac arrest predicts an unsatisfactory outcome (8).

However, if we could have performed TAE as an emergent procedure, there might have been a ray of hope that she could survive. For that purpose, a quick and accurate diagnosis is always required in the scene of an emergency.

References

1. Covarelli P, Badolato M, Boselli C, et al. Splenic vein thrombosis complicated by massive gastric bleeding: treatment with arterious embolization. *Am Surg* **74**: 184-186, 2008.
2. Williams N, Gerrard C, London NJ, Chapman C, Bell PR. Splenic rupture following splenic vein thrombosis in a man with protein S deficiency. *Postgrad Med J* **68**: 928-929, 1992.
3. Hiraiwa K, Morozumi K, Miyazaki H, et al. Isolated splenic vein thrombosis secondary to splenic metastasis: a case report. *World J Gastroenterol* **12**: 6561-6563, 2006.
4. Koklu S, Koksak A, Yolcu OF, et al. Isolated splenic vein thrombosis: an unusual cause and review of the literature. *Can J Gastroenterol* **18**: 173-174, 2004.
5. Zimmerman D, Bell WR. Venous thrombosis and splenic rupture in paroxysmal nocturnal hemoglobinuria. *Am J Med* **68**: 275-279, 1980.
6. Tzur I, Almozni-Sarafian D, Dotan E, et al. Splenic vein thrombosis following abdominal compression and vibration: a case report. *Angiology* **59**: 514-516, 2008.
7. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. *Crit Care Med* **13**: 818-

- 829, 1985.
8. Edgren E, Hedstrand U, Kelsey S, Sutton-Tyrrell K, Safar P. Assessment of neurological prognosis in comatose survivors of cardiac arrest. BRCT I Study Group. *Lancet* **343**: 1055-1059, 1994.

© 2009 The Japanese Society of Internal Medicine
<http://www.naika.or.jp/imindex.html>