Two Cases of Minor Blunt Splenic Injury Accompanied with Unexpected Fatal Delayed Splenic Rupture

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Contrast blush (CB) is an area with a density higher than the organ parenchyma in the arterial phase of contrast-enhanced computed tomography (CT). CB may be a sign of contrast medium extravasation, pseudoaneurysm, arteriovenous fistula, or other conditions; however, the indications for treatment remain unclear. Nevertheless, CB could be used to indicate a fatal scenario, such as delayed splenic rupture. Here, we present two multiple-injury cases of fatal delayed splenic rupture following the nonoperative management of a minor splenic injury. In both cases, despite morphological CT findings being minor on admission, CB was observed, and both patients could not rest owing to factors such as older age, a head injury, and drunkenness. Furthermore, in the CB case that indicated pseudoaneurysm, delayed splenic rupture occurred much earlier after the injury compared to the other case without the possibility of pseudoaneurysm. In conclusion, we recommend transcatheter arterial embolization be urgently performed in a case wherein the presence of a pseudoaneurysm is highly probable and factors such as multiple injuries and inability to rest are involved.

Key words: contrast blush, delayed splenic rupture, nonoperative management, transcatheter arterial embolization

INTRODUCTION

An area having a density higher than that of the organ parenchyma in the arterial phase of contrast-enhanced computed tomography (CT) is called contrast blush (CB). It indicates contrast medium extravasation, pseudoaneurysm, or arteriovenous fistula [1]. Currently, nonoperative management (NOM) is the mainstay of treatment for hemodynamically stable blunt splenic injury, and transcatheter arterial embolization (TAE) plays an important role in completing NOM [2]. However, its absolute indications including when TAE should be performed are unclear, and some cases may be fatal if accompanied by severe complications. Thus, it is important to choose treatment not only according to damage morphology but also according to CB presence while considering the risk of complications. Here, we report two cases of splenic injury accompanied with delayed splenic rupture having a minor morphological damage.

CASE REPORT

Case 1

A 71-year-old woman sustained a fall injury. She had no past medical history and was not on antithrombotic drugs. On the third day after the injury, she visited a local physician with a complaint of abdominal pain and was admitted to the hospital with a diagnosis of splenic injury, bilateral traumatic hemothorax, and bilateral multiple rib fractures [Injury Severity Score (ISS) 34]. Contrast-enhanced CT at that time revealed no extravasation in the spleen; however, in the arterial phase, there was a region in the lower part of the spleen that was densely dyed compared with the surrounding areas. Based on the imaging findings, there was no laceration. Moderate subcapsular hematoma was observed and there was no active bleeding into the peritoneum (Fig. 1). Although she had been followed up conservatively, her health deteriorated after experiencing sudden hemorrhagic shock on the tenth day after the injury and was transferred to our hospital. At the time of emergency transport to our hospital, her blood pressure was 78/48 mmHg, but no obvious coagulation disorder was observed. Contrast-enhanced CT revealed extravasation in the lower part of the spleen and fluid retention around the spleen and liver (Fig. 2). An emergency transfusion of four red blood cell units was performed, and the systolic blood pressure (SBP) rose to 100 mmHg. Therefore, we decided to perform angiography. Angiographic examination revealed extravasation from the inferior terminal branch of the splenic artery; thus, TAE was performed using a microcoil (Fig. 3). Subsequently, the vital signs stabilized. No major problem occurred after that, and she was transferred for rehabilitation.

Case 2

A 50-year-old male pedestrian was run over by a motorcycle. There was no significant medical history, and he was not on antithrombotic therapy. On admission, vital signs were as follows: Glasgow Coma Scale of E1V1M5, respiratory rate of 18/min, heart rate of

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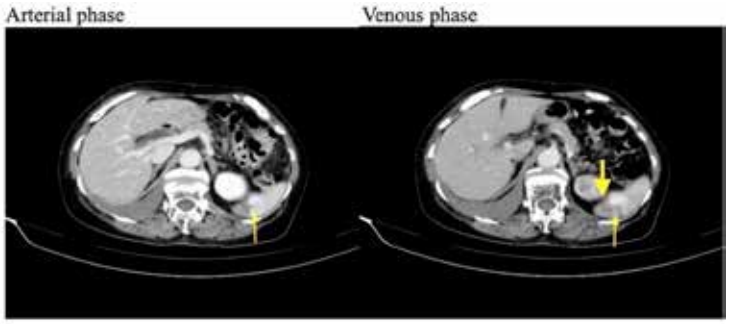

Fig. 1 In the arterial phase, a part that shows the local enhancement effect of the contrast medium in the lower part of the spleen was identified (CB; arrow).
In the venous phase, the washout of the region where the enhancement effect of the contrast medium was observed in the arterial phase was not identified (CB; arrow). A moderate subcapsular hematoma was identified (thick arrow), and the injury was classified as AAST grade II.

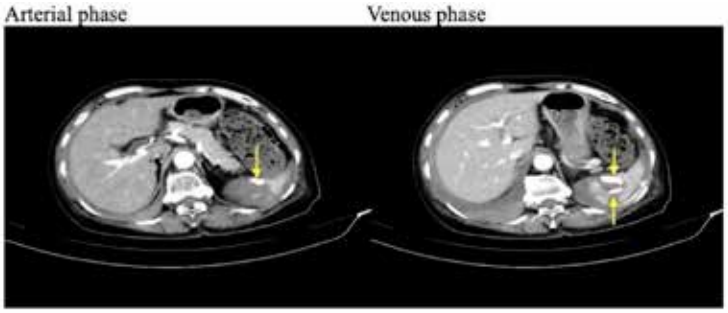

Fig. 2 Extravasation (arrow) of the contrast medium was identified in the lower part of the spleen in the arterial phase. In addition, fluid retention was observed around the liver and spleen.
In the venous phase, extravasation of the lower part of the spleen expanded slightly (arrow).

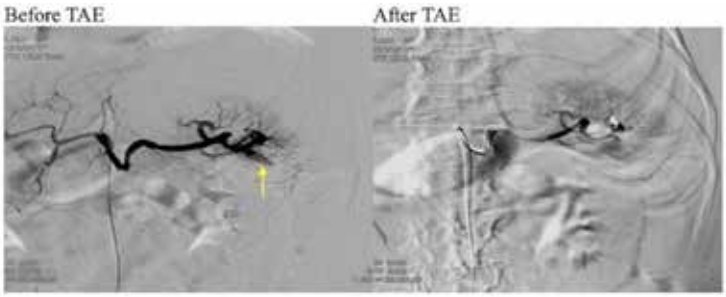

Fig. 3 Extravasation from the inferior terminal branch of the splenic artery was identified (arrow). After transcatheter arterial embolization with a microcoil at the inferior terminal branch of the splenic artery, extravasation disappeared.

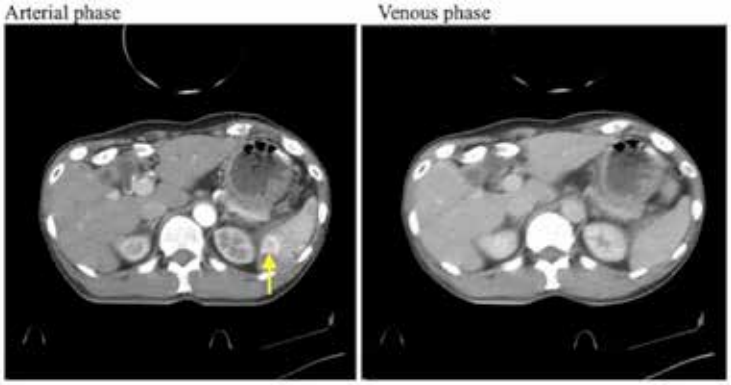

Fig. 4 In the arterial phase, local contrast media retention (CB; arrow) of 15-mm diameter was identified in the lower part of the spleen. In the venous phase, the regional contrast media retention of the splenic lower part recognized in the arterial phase disappeared.

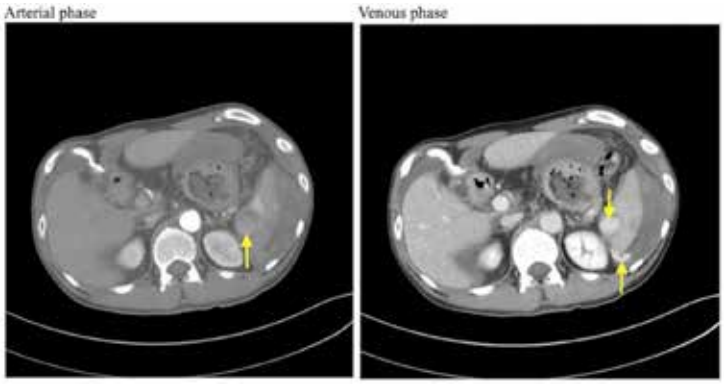

Fig. 5 In the lower part of the spleen, a site showing a circular shape of 15-mm diameter in the arterial phase (arrow) showed the retention of the contrast medium in the venous phase and extravasation (arrow) of the contrast medium from the same site.

Fig. 6 Angiography revealed a contrast medium retention (arrow) with a diameter of 15 mm in the splenic parenchyma. After transcatheter arterial embolization was performed using a microcoil in the inferior terminal branch of the spleen, the contrast medium retention disappeared.

78/min, blood pressure of 130 mmHg as observed by the manual method, and oxygen saturation of 97% (on 10-L O_2 reservoir mask). At the hospital, imaging studies were performed, and a diagnosis of traumatic subarachnoid hemorrhage, splenic injury, and left open ulna fracture [ISS 27] was made. Regarding the splenic injury, contrast-enhanced CT showed a high-density area in the arterial phase with a diameter of 15 mm in the lower part of the spleen, whereas in the venous phase, there was an area of lesions with the same level of contrast as that of the surrounding tissues. There was an unclear presence of laceration and subcapsular hematoma (Fig. 4). Conservative treatment was performed. However, the patient deteriorated and experienced shock with a systolic pressure of 50/- mmHg on the third day of hospitalization. Because the blood pressure was recovered due to fluid resuscitation, CT was performed. In the lower part of the spleen, a site with a 15-mm circular shape in the arterial phase showed contrast medium retention in the venous phase and contrast medium extravasation from the site (Fig. 5), suggesting pseudoaneurysm rupture. Furthermore, no obvious blood coagulation disorder was observed. SBP was elevated in response to rapid fluid therapy, we decided to perform hemostasis with TAE. Angiography revealed a pseudoaneurysm of 15 mm in diameter in the inferior terminal branch of the splenic artery with extravasation from the same site. Therefore, TAE was performed for the inferior terminal branch of the splenic artery using a microcoil (Fig. 6). Subsequently, the vital signs stabilized, and the progress was good; thus, the patient was transferred for rehabilitation.

DISCUSSION

In general, in hemodynamically stable blunt splenic injury, contrast-enhanced CT is performed to choose a treatment strategy based on injury morphology, vital signs, intraperitoneal bleeding amount, CB presence, and active bleeding [3, 4]. The injury scale (Table 1) advocated by the American Association for the Surgery of Trauma (AAST) is predominantly accepted worldwide for the evaluation of the degree of splenic injury morphology. The spleen organ injury scale of the AAST was revised in 2018, and the most important change in the revision was the inclusion of CT diagnosed vascular injury into the organ injury scale [5]. In this article, we have adopted the spleen organ injury scale of the AAST before the revision because the current main guidelines [3, 4] for spleen trauma refer to it, and there is no guideline referring to the revised scale yet.

The fatality of delayed splenic rupture ranges from 5% to 15%, which is reported to be higher than that of acute splenic rupture (1%) [6, 7]. Although the pathophysiology of delayed splenic rupture remains largely unknown, it is currently suggested that it involves any of the following three pathologies: 1. the splenic capsule breaks at the time of injury, hematomas form around the injured area, and temporary hemostasis is achieved by the tamponade effect, but the tamponade

Table 1 Spleen Organ Scale of the American Association for the Surgery of Trauma (AAST) (before the 2018 revision)

Grade	Injury type	Description of injury
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1 cm Parenchymal depth
II	Hematoma	Subcapsular, 10%–50% surface area Intraparenchymal, 5 cm in diameter
	Laceration	Capsular tear, 1–3 cm parenchymal depth that does not involve a trabecular vessel
III	Hematoma	Subcapsular, >50% surface area or expanding; ruptured Subcapsular or parenchymal hematoma; intraparenchymal hematoma > 5 cm or expanding
	Laceration	>3-cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (>25% of the spleen)
V	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury with devascularized spleen

effect weakens over time, leading to splenic rupture [7]; 2. subcapsular hematoma is formed at the time of injury, subsequently leading to intraperitoneal hemorrhage with the increase in internal pressure [6, 8–10]; and 3. a pseudoaneurysm is formed in the spleen due to blunt trauma and causes delayed rupture [11]. The case 1 patient was considered to have AAST grade II injury morphology because contrast-enhanced CT on the third day after the injury showed no obvious perisplenic hematoma, intraperitoneal hemorrhage was suspected, and capsular damage in addition to a mild sub-splenic hematoma was identified (Fig. 1). In addition, there were no clear findings suggestive of pseudoaneurysm, even on performing angiography at the time of delayed splenic rupture. From the above, the possibility of damage to the inferior terminal branch of the splenic artery was considered; thus, case 1 seemed to correspond to pathology "2" mentioned above. In case 2, contrast-enhanced CT showed no obvious intra-abdominal bleeding or extravasation at the time of admission, but a lesion of 15-mm diameter was observed that was densely dyed in the arterial phase in the lower part of the spleen and had isodense areas in the surrounding splenic tissue in the venous phase (Fig. 4). The vital signs were stable, there was no active intra-abdominal bleeding, and the injury morphology corresponded to AAST grade I. Angiography performed at the time of delayed splenic rupture revealed a pseudoaneurysm of approximately 15 mm in diameter in the inferior terminal branch of the splenic artery and extravasation from the same site (Fig. 6), which was considered to correspond to pathology "3" mentioned above.

Regarding splenic injury morphology, Peitzman and colleagues reported that the success rate of NOM for AAST grade III injures is approximately 50%, and the success rate of NOM decreases as the injury morphology rises to grades IV and V [12]. In addition, Crichton *et al.* reported in AAST grade that TAE should not be performed routinely for AAST grades I–III due to the possibility of various complications such as splenic abscesses. Moreover, the splenic injury morphology of AAST grades IV and V contributes to the success of NOM [13]. It is thus crucial to make a cautious decision on whether or not to perform TAE for mild splenic injury.

Regarding CB, Schurr *et al.* reported that the presence of CB may be a factor responsible for NOM failure [1]. Kaneko *et al.* reported a case in which CB appeared during blunt splenic injury treatment, leading to a delayed splenic rupture; they also reported that urgent angiographic examination should be performed if CB is recognized [14]. In addition, the EAST guidelines state that the presence of CB alone should not be the indication for TAE, and it is necessary to evaluate vascular injury via angiography and comprehensively examine the indications for TAE when CB is recognized [3]. Also, according to WSES classification of splenic trauma and the management guidelines, there is no absolute indication for TAE against the minor splenic injury with CB like case 1 or case 2 [4].

In this study, the case 1 patient was old, and the case 2 patient consumed alcohol and had a head injury, which might prevent them from resting. As a result, blood pressure rose, and force was applied to sub-splenic hematoma in case 1 and pseudoaneurysm in case 2 and splenic ruptures were occurred. In addition, both case 1 (ISS 34) and 2 (ISS 27) patients sustained multiple injuries. Olthof *et al.* reported ISS of ≥25 as predictors of NOM failure [15]. Therefore, the inability to rest and multiple injuries seemed to be a risk factor for NOM failure.

Regarding the time of delayed splenic rupture, it occurred on the tenth day in case 1 and on the third day in case 2 after the injury. In case 1, the pathophysiology was believed to be the rupture of sub-splenic

hematoma, whereas in case 2, the pseudoaneurysm was ruptured. Therefore, in cases wherein a pseudoaneurysm is likely to occur, TAE should be prioritized and performed urgently.

To summarize, there are some cases in which TAE should be performed in the early stage of CB, as CB could indicate vascular injury, even if morphological CT findings just after injury are minor. We recommend that TAE should be performed urgently in such cases in which the presence of a pseudoaneurysm is very probable and factors such as multiple injuries and the inability to get rest are involved.

CONFLICT OF INTEREST

There are no conflicts of interest to be disclosed.

REFERENCES

1) Schurr MJ, Fabian TC, Gavant M, Croce MA, Kudsk KA, Minard G, *et al*. Management of blunt splenic trauma computed tomographic contrast blush predicts failure of nonoperative management. J Trauma 1995; 39: 507–13.
2) Requarth JA, D'Agostino Jr RB, Miller PR. Nonoperative management of adult blunt splenic injury with and without splenic artery embolotherapy: a meta-analysis. J Trauma 2011; 71: 898–903.
3) Stassen NA, Bhullar I, Cheng JD, Crandall ML, Friese RS, Guillamondegui OD, *et al*. Selective nonoperative management of blunt splenic injury: an Eastern Association for the Surgery of Trauma practice management guideline. J Trauma 2012; 73: 294–300.
4) Coccolini F, Montori G, Catena F, Kluger Y, Biff W, Moore EE, *et al*. Splenic trauma: WSES classification and guidelines for adult and pediatric patients. World J Emerg Surg 2017; 12: 1–26.
5) Kozar RA, Crandall M, Shanmuganathan K, Zarzaur BL, Coburn M, Cribari C, *et al*. Organ injury scaling 2018 update: Spleen, liver, and kidney. J Trauma Acute Care Surg 2018; 85: 1119–22.
6) Kluger Y, Paul DB, Raves JJ, Fonda M, Young JC, Townsend RN, *et al*. Delayed rupture of the spleen—myths, facts, and their importance: case reports and literature review. J Trauma 1994; 36: 568–71.
7) Kodikara S, Sivasubramanium M. Mechanisms of delayed splenic rupture: a new hypothesis. Leg Med 2009; 11: 515–7.
8) Davis JJ, Cohn I Jr, Navice FC. Diagnosis and management of blunt abdominal trauma. Ann Surg 1976; 183: 672–8.
9) Foster RP. Delayed haemorrhage from the ruptured spleen. Br J Surg 1970; 57: 189.
10) Allen TL, Greenlee RR, Price RR. Delayed splenic rupture presenting as unstable angina pectoris: case report and review of the literature. J Emerg Med 2002; 23: 165–9.
11) Muroya T, Ogura H, Shimizu K, Tasaki O, Kuwagata Y, Fuse T, *et al*. Delayed formation of splenic pseudoaneurysm following nonoperative management in blunt splenic injury: multi-institutional study in Osaka, Japan. J Trauma 2013; 75: 417–20.
12) Peitzman AB, Heil B, Rivera L, Federle MB, Harbrecht BG, Clancy KD, *et al*. Blunt splenic injury in adults: multi- institutional Study of the Eastern Association for the Surgery of Trauma. J Trauma.2000; 49: 177–87.
13) Crichton JCI, Naidoo K, Yet N, Brundage SI, Perkins Z. The role of splenic angioembolization as an adjunct to nonoperative management of blunt splenic injuries. A systematic review and meta-analysis. J Trauma 2017; 83: 934–43.
14) Kaneko N, Yanagawa Y. Case report of delayed splenic rupture: significance of "contrast blush" on follow-up contrast-enhanced CT scans. J Abdom Emerg Med 2007; 27: 541–5 (In Japanese, Abstract in English).
15) Olthof DC, Joosse P, van der Vlies CH, de Haan RJ, Goslings JC. Prognostic factors for failure of nonoperative management in adults with blunt splenic injury: a systematic review. J Trauma 2013; 74: 546–57.