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# Management of a delayed, post-traumatic rupture of splenic artery pseudoaneurysm in a patient with life threatening co-morbidities: A treatment challenge

Meena Kumari, Masoom Parwez, Akash Jain, Bharati Pandya\*

Department of General Surgery, All India Institute of Medical Sciences, Bhopal, MP, India

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## ABSTRACT

**INTRODUCTION:** Splenic artery embolization (SAE) is an accepted intervention for patients with traumatic injury AAST III-IV in hemodynamically stable patients, splenic artery aneurysm and pseudoaneurysm (Brian and Charles, 2012). Unusual circumstances may pose different challenges in individual cases.

**CASE PRESENTATION:** A 52-year-old male on anticoagulants for past mitral valve replacement presented to us with history of blunt trauma sustained a month prior, was found to have grade IV splenic injury with delayed pseudo-aneurysmal rupture. In addition, his cardiac evaluation revealed an ejection fraction of 20%. A potential life threatening unstable cardiac status and hemodynamic irregularities accentuated due to the hemoperitoneum was an unusual challenge to deal with. After initial stabilization in ICU, the option of distal embolization of splenic artery was undertaken in a well-planned manner.

**DISCUSSION:** Unstable cardiac condition, anticoagulant therapy and delayed pseudo aneurysmal bleed led us into undertaking this procedure as a semi-emergency with calculated risks. We discuss this case due to the complexities and dilemmas on various aspects which we faced in his management.

**CONCLUSION:** Patient tolerated the procedure well and was discharged on the third day of embolization. Our experience taught us the judicious implementation of a viable and only lifesaving option for an otherwise inoperable patient due to multiple co-morbidities and would strongly recommend this interventional radiological, relatively innocuous procedure for salvaging such patients.

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## 1. Case report

A 52-year-old male, Indian male, businessman by occupation, presented in emergency department with an alleged history of road traffic accident one month prior, for which he underwent a CECT and was diagnosed to have grade III splenic injury with hemoperitoneum, cholelithiasis being an incidental finding. He was admitted for 5 days, kept on conservative non operative management (NOM) in a private hospital and subsequently discharged. His presenting complaints were progressive pain in the left upper abdomen for last 15 days and transient hematuria for 5 days, which followed catheterization.

Patient had no history of fever, vomiting or any bowel complaints. Hematuria present on admission subsequently cleared off.

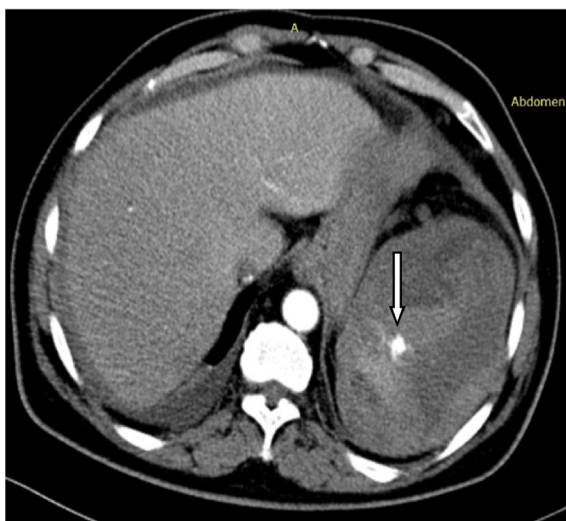
He was a known case of hypertension and atrial fibrillations; having undergone a prosthetic mitral valve replacement 12 years ago. Since the surgery he was on regular warfarin and beta-blockers.

He sustained head injury and had repeated seizures post injury in past 1 month and was on oral anti-epileptics for the same, as CT scan revealed a small left sided subdural hematoma. He had bilateral minimal pleural effusions with basal atelectasis on chest X-ray. Recent ECHO-cardiograph had shown dilated cardiomyopathy with a left ventricular ejection fraction of 20% but, with a normally functioning prosthetic valve. He had no known allergies or addictions.

On examination he was fully conscious and oriented, his heart rate was 57/min, Blood pressure of 140/80 mmHg, respiratory rate of 18/min and 96% SpO<sub>2</sub>. Abdomen was soft with tenderness in left hypochondrium. No rebound or guarding was noted. Foley's catheter was in place with mild hematuria. On the night of admission patient developed sudden fall in heart rate, blood pressure and saturation and required being shifted to ICU immediately where he was treated for arrhythmias and hemodynamic instability. Investigations revealed a hemoglobin of 6 g/dl, INR of 3.49, Urine R/M RBCs full field and no pus cells. Blood and fresh frozen plasma transfusion were given. After stabilization, CECT Abdomen was done which was suggestive of grade IV splenic injury involving 80% of the spleen with multiple, non-enhancing, devascularized areas noted scattered within and extending up till the hilum. Active contrast extravasation (contrast-blush) was noted from the splenic artery,

\* Corresponding author.

E-mail addresses: [meenahmc@gmail.com](mailto:meenahmc@gmail.com) (M. Kumari),[Masoomparwez92@gmail.com](mailto:Masoomparwez92@gmail.com) (M. Parwez), [Aksjain@gmail.com](mailto:Aksjain@gmail.com) (A. Jain),[bharati\\_pandya@rediffmail.com](mailto:bharati_pandya@rediffmail.com) (B. Pandya).<https://doi.org/10.1016/j.ijscr.2020.09.083>2210-2612/© 2020 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).



**Fig. 1.** CECT in delayed arterial phase showing contrast blush in traumatic spleen and hemoperitoneum.



**Fig. 2.** Selective angiography demonstrating the pseudoaneurysm in upper pole (distal splenic vessel).

the splenic parenchyma near the hilum along the superior pole; with moderate hemoperitoneum (Figs. 1 and 2). Bilateral pleural effusion with underlying segmental and subsegmental collapse and cardiomegaly were confirmed on CT scan.

After cardiology reference, warfarin was stopped and patient was shifted to heparin 1000 U/hour with serial monitoring of

hemoglobin, hematocrit, aPTT and PT/INR. Vitamin K was started. Further blood and fresh frozen plasma were transfused accordingly.

Interventional radiology consultant was consulted, and patient was planned for angiography and embolization. Consent was taken and prior arrangement for surgery made in case any unwarranted complication of angio-embolization was encountered. Pseudoaneurysm embolization was done by the interventional radiology consultant, under fluoroscopic guidance using 50:50 glue: lipiodol after the INR had come to 1.35 (Figs. 3–5). Procedure was successful and patient subsequently improved with lab-parameters and hemodynamic status returning to normal. Patient was discharged after shifting him back to oral anticoagulants and beta-blockers. On follow-up after 3 days, and 2 weeks, patient was asymptomatic with normal duplex sonography. Further telephonic queries made were satisfactory and patient was advised follow-up after 6 months and when necessary.

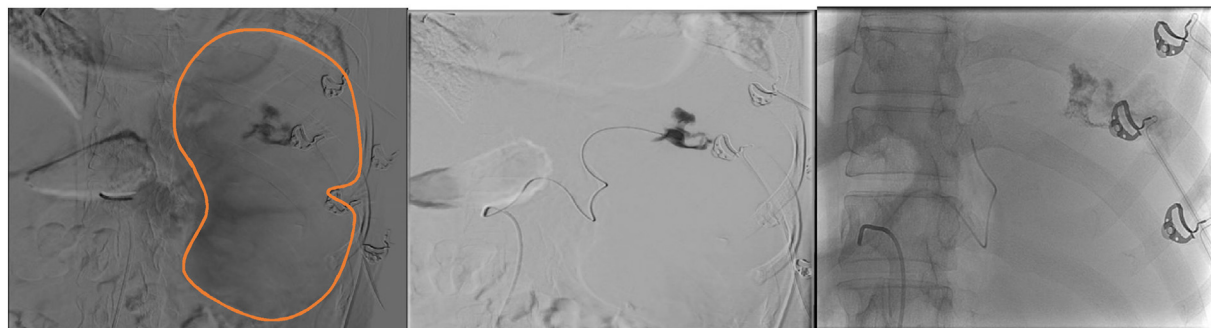
**2. Discussion**

The spleen tops the list of most injured organs in blunt abdominal trauma and if goes undetected or is not managed skillfully results in rapid exsanguination of the patient and hence accounts for immediate explorations in about 40% of cases [2–4]. The management of splenic trauma may be divided into non operative and operative. In the Non-operative management is included conservative management and interventional radiology. In operative management is included splenic preservation and splenectomy [1,3].

This approach to splenic trauma evolved due to the dreaded complication of extirpative surgery, OPSI (overwhelming post splenectomy infections), that was reported in 0.5% of traumatic splenectomies and up to 20% of non-traumatic causes for splenectomy. OPSI manifests in the first two years after surgery, but results in a lifetime risk of infections and carries a mortality of 50–80% [3]. Now, non-operative management of splenic trauma has become the standard of therapy in hemodynamically stable patients with 90% success in tertiary centers [5,6]. NOM of traumatic splenic injuries especially in AAST (American Association of Surgery for Trauma, Table 1) grades I to III has a high success rate, but, for injuries of grade IV, the success rates steeply drops to 33% and Grade V injuries in the majority, require operative management [2,5].

The advantages of NOM over surgical management are described as lower hospital costs, avoidance of non-therapeutic laparotomies, lower rates of intra-abdominal complications and blood transfusions, lower mortality and the maintenance of the immunological function, and the prevention of OPSI [5].

The conservative management with radiological intervention could be using various agents like, gel foam, particles, glue, or coils in isolation or together. It could be proximal or distal embolization or could be partial or complete [1]. We used glue in lipiodol 50:50



**Figs. 3–5.** Selective Catheterization of Splenic artery to reveal 3) The splenic outline with contrast blush in superior pole. 4) Pre-embolization picture and 5) Injection of glue.

**Table 1**  
Grading of splenic injuries as per AAST splenic injury scale.

Grading	Description
Grade 1	<ul style="list-style-type: none"> <li>• Hematoma, subcapsular, less than 10% surface area</li> <li>• Laceration, capsular tear, less than 1 cm parenchymal depth</li> </ul>
Grade 2	<ul style="list-style-type: none"> <li>• Hematoma, subcapsular, 10%–50% surface area</li> <li>• Intraparenchymal, less than 5 cm in diameter</li> <li>• Laceration, capsular tear, 1 cm–3 cm parenchyma depth that does not involve a trabecular vessel</li> </ul>
Grade 3	<ul style="list-style-type: none"> <li>• Hematoma, subcapsular, more than 50% surface area expanding; ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma 5 cm or greater and expanding</li> <li>• Laceration, greater than 3 cm parenchymal depth or involving trabecular vessels</li> </ul>
Grade 4	<ul style="list-style-type: none"> <li>• Laceration, laceration involving segmental or hilar vessels producing major devascularization (more than 25% of the spleen)</li> </ul>
Grade 5	<ul style="list-style-type: none"> <li>• Laceration, completely shattered spleen</li> <li>• Vascular, Hilar vascular injury that devascularizes the spleen</li> </ul>

for embolization of the distal splenic artery where the angiography had picked up the leaking pseudoaneurysm. The complications of NOM are hematoma, splenic pseudocyst, sepsis, delayed rupture, splenosis, and delayed exploration with increased morbidity and mortality [3]. SAE has known complications like Splenic infarct, bleeding, sepsis, followed by subsequent abscess, basal pneumonia, and atelectasis [1]. Revascularization though less common in distal embolization may occur and collaterals may also lead to rebleed [1,10]. Failure of the procedure may occur due to complications in the co-morbid elderly due to longer time required for distal embolization [1,10]. SAE was controversial in 2008 but has shown progressive reduction in failure rates of nonoperative management, from 25% to 10%, and an increase in splenic salvage rates from 79% to 100% [4]. SAE with coils for aneurysms, has a success rate from 67 to 92% in published data [9]. Risk of recanalization of 5% in (pseudo)aneurysms is reported and a 12.5% partial recanalization rate is described [9]. Recently distal embolization has reduced the rate of NOM failure even in patients with risk factors such as high-grade injury (IV-V, AAST), older age (> 55 years), disorders of consciousness, contrast blush on computed tomography (CT) and the presence of associated injuries, thus, expanding the indications for NOM. Nonetheless, the rates of delayed hemorrhage though rare, occur in 0–3.9% of patients. It is a life-threatening complication of NOM and often requires an emergency laparotomy and needs to be judiciously undertaken with explained risks [11].

Delayed splenic rupture, defined as the rupture of the spleen after 48 h of trauma with a previous asymptomatic period, was first described in 1902, by Baudet [7,8]. It is a rare but well-known complication of splenic trauma and is one of the causes of failure of NOM with an incidence of approximately 1%, and occurs due to expansion of a subcapsular hematoma, clot disruption, rupture of a pseudoaneurysm or splenic pseudocyst, between 4 and 8 days after injury. In majority, it occurs within a week, but cases that occur months after trauma have also been reported. In cases of delayed rupture mortality ranges from 5% to 15% [8].

Pseudo-aneurysm formation has been reported to occur in 10% of cases and may result in catastrophic hemorrhage if left untreated. These aneurysms can be detected as well-defined, hypo-echoic lesions on ultrasound. Color Doppler confirms the diagnosis by demonstrating hypervascularity and angiography is indicated in such patients [10]. Post-traumatic pseudoaneurysm as a cause for delayed hemorrhage in patients with NOM is seen in 2–27% [4].

Adults, especially above 50 years, with splenic trauma deserve special attention. Patients in this age group independently add to the morbidity associated with AAST grading [4]. A few studies found older age (above 55 years) to be a significant prognostic factor for NOM failure. These are owing to age related structural

and vascular changes. These patients show few findings initially, due to altered physiology with sudden, rapid deterioration later. Age related morbidity is accentuated by the medications they are on, more often anti-hypertensives like beta-blockers, or anticoagulants like aspirin, clopidogrel and warfarin [2], like in our case. These drugs may mask the hemodynamic changes and cause undue hemorrhage, leading to late presentations allowing limited management options [2].

Our patient was 52-year-old, with history of trauma a month ago, which means he had a delayed hemorrhage in a diagnosed grade IV splenic trauma and hence, an already failed NOM. He had 6 gm% hemoglobin to start with, a high INR due to warfarin, in addition to being a case of operated mitral valve with replacement prosthesis, whose ejection fraction was only 20%. Even in normal, minor surgical situations, this would carry an exceedingly high morbidity and mortality. In our patient, severe anaemia due to active bleed from pseudoaneurysm of the splenic artery and warfarin adding to a raised INR, cardiomegaly, atrial fibrillation, pulmonary edema and pleural effusion and a fluctuating hemodynamic state required a trying 5 day management in the ICU with delicate balance between fluid, plasma and blood replacements. Each problem he had, in itself was a major challenge. He was in a seesaw with equal risks involved in a surgical option as also in splenic preservation.

### 3. Conclusion

Splenic trauma, the most commonly encountered solid organ injury, can be successfully managed with NOM in most cases. However, higher age group and co-morbidities pose a challenge in both operative and non-operative management. With this patient successfully managed with distal SAE, we believe it is a feasible option worth implementing even for patients with otherwise limited options.

SCARE Check list adhered to [12].

### Declaration of Competing Interest

None.

### Funding

No Sponsors involved.

### Ethical approval

As the Institute is a teaching Institute run by Central government of India, Case reports require only patient's informed consent and are exempt from formal ethical approvals.

### Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

### Author's contribution

First author had admitted the patient and with the help of 2nd and 3rd authors was involved in active participation in the process of admission, procedural and management of the patient.

First author was also involved in concept of the design of paper, data interpretation and collection.

The corresponding author was in-charge of the overall patient care, writing and editing the paper, and follow-up care of the patient as well.

### Registration of research studies

1. Name of the registry: NA
2. Unique identifying number or registration ID: NA
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): NA for case reports in India.

### Guarantor

Corresponding author: Dr. Bharati Pandya.

### Provenance and peer review

Not commissioned, externally peer-reviewed.

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