Non-Traumatic Arterial Hemorrhage: Efficacy of Transcatheter Artery Embolization

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Aims and objectives

Arterial hemorrhage of the trunk occurs on the ground of traffic accident, accident at work, or iatrogenic etiology in many cases and it sometimes results in fatal bleeding.

In contrast, non-traumatic arterial hemorrhage (NAH) occurring along with a coagulation disorder is sometimes experienced and results in fatal bleeding.

Such cases of NAH treated by surgical drainage and transcatheter arterial embolization (TAE) are reported [1-3]; however, there are a few well-organized studies regarding the incidence and the treatment of NAH occurring along with a coagulation disorder.

The purpose of this study is to determine the efficacy of TAE for NAH.
Methods and materials

From the database of our hospital, National Center for Global Health and Medicine, and the two affiliated hospitals, we extracted the 256 patients in the abdomino-pelvic region whom urgent TAE was performed.

The investigation periods and the studied institutions were as follows:

- From January 1995 to July 2010 (Kyushu University Hospital) (n = 191)
- From April 2010 to April 2011 (Tokyo Women’s Medical University Hospital) (n = 30)
- From April 2013 to December 2014 (National Center for Global Health and Medicine) (n = 35)

Among them, patients who showed abnormal values on coagulation test at the onset of NAH were searched; cases with disseminated intravascular coagulation (DIC) due to chronic bleeding were excluded from the study group.

The frequency and the cause of NAH, presence of coagulopathy, findings of CT and angiography, methods of TAE and the technical and clinical successes of TAE were analyzed.
Results

Summary of the Results

The causes and frequency of the arterial hemorrhage in the 256 cases were shown in the Fig. 1 on page 9.

1. We found nine patients with NAH (four men and three women; 25-88 years old with median age of 66 year-old) (3.5%) among the 256 patients that were treated by emergency TAE. The detail of the patient profile was shown in Fig. 2 on page 9.

2. All patients had coagulopathy. The causes of coagulopathy were as follows: Anticoagulant therapy 4 (Warfarin 2, Caprocin 2); Hemophilia B 1; Antiphospholipid syndrome 1; Factor VIII inhibitor 1; antiplatelet therapy 1, and unknown cause 1.

3. The bleeding sites were as follows: iliopsoas muscle 3 (Fig. 4 on page 11); hepatic subcapsular region 1 (Fig. 5 on page 12); rectus abdominis muscle 2; splenic parenchyma 1; small intestine 1; and gluteal muscle 1.

4. The primary diseases of the four cases complicated with anticoagulant therapy or antiplatelet therapy were as follows: status of post cardiac valve replacement 2; chronic atrial fibrillation 1; deep vein thrombosis 1; and status of post stenting for coronary artery stenosis 1.

5. Blood findings (11 sessions, n = 9):

   - Hb level: < 9 g/dL in all sessions (mean 7.3)
   - Platelet level: 5.8-34.2 x 10^4/µL (mean 11.9)
   - Activated partial thromboplastin time (APTT) at onset showed moderate to high values in all cases but one (8/9 cases; 9/11 sessions).
   - PT%: 13.0-106.0% (mean 64.5)
   - In all four cases with anticoagulant use, PT% showed < 50% and PT-INR has been beyond the management criteria (2.0-3.0) of the oral anticoagulation in oral anticoagulation guidelines [4].

The detail of findings of CT and angiography and the TAE was shown in Fig. 3 on page 10.

1. Prognosis: All patients except two survived without rebleeding after the primary hemostasis. Two patients recurred in the contralateral region (Fig. 6 on page 13, Fig. 7 on page 14, Fig. 8 on page 15, Fig. 9 on page 16); one patient survived and another died of sudden hypotension in 5 days after re-TAE (Fig. 8 on page 15, Fig. 9 on page 16)

2. Angiographic findings:
• Extravasation / pooling: 10/11 sessions
• Arteriovenous shunt: 1/11 sessions
• Number of the involved blood vessels: one, 5/11 sessions; two, 2/11 sessions; and three, 4/11 sessions.

The detail of angiographic techniques and the prognosis was shown in Fig. 3 on page 10.

1. Embolic methods: for all cases, TAE was performed via the microcatheter inserted superselectively using gelatin sponge particles (GSP).
2. For the case with splenic hemorrhage, partial splenic embolization was performed using GSP mixed with antibiotics.
3. Technical success: 11/11 sessions
4. Complications: 0/11 sessions
5. Prognosis: All patients but one survived without rebleeding more than one month. However, two patients recurred in the contralateral side of the muscles (gluteal muscle and iliopsoas muscle) in two days (Fig. 6 on page 13, Fig. 7 on page 14) and two months later (Fig. 8 on page 15, Fig. 9 on page 16), respectively. One of the two patients died due to the hypovolemic shock on the third day after the second TAE (Fig. 9 on page 16).

Concepts and Characteristics of NAH

Spontaneous NAH associated with coagulopathy is an uncommon and often misdiagnosed cause of abdominal pain. As for clinical symptoms, it occurs as sudden stomachache. Because a symptom is strong and is often accompanied by peritoneal irritation sign, so the patients may be diagnosed acute abdomen, often leading to abdominal operation.

It is considered that most of the patients have some kinds of underlying disease. Especially, NAH in the rectus abdominis muscle is considered as a condition in which haematoma is formed within the Sarcolemma by the rupture of the epigastric artery due to the sudden constriction of the rectus abdominis muscle [1-3].

Clinical conditions that may cause NAH are classified into direct cause (anticoagulant therapy) and non-direct cause such as 1) underlying diseases such as arterial sclerosis and hypertension; 2) poor diabetes control (peripheral circulatory failure); 3) sepsis; and 4) haemodialysis. However, there was a few well-organized reports regarding the underlying disease.

NAH associated with Coagulopathy: Literature Review
The soft tissues are the commonest sites of anticoagulant-related haematoma, reported in 21-31% of patients on anticoagulants, retroperitoneal haematoma, iliopsoas haematoma and rectus-sheath haematoma, being the most frequent sites [5].

In our study, all patients had coagulopathy: anticoagulant therapy (4/9: 44%) and prolongation of APTT level (8/9: 89%), antiphospholipid syndrome (1/9: 11%), inactivity of anti-hemophilic factor B (1/9: 11%) and presence of factor VIII inhibitor (1/9: 11%).

In our study, most of the patients showed the acute onset style of 1-2 days; chief symptoms were the anemia and the dull pain of abdomen, back or buttock. In 6 of the 9 (67%) patients the bleeding site was within the Sarcolemma of the muscles such as rectus abdominis muscle, iliopsoas muscle and gluteal muscle.

In our series (n = 9), five women (mean age 51 years) with rectus sheath haematoma (n = 2), hepatic subcapsular haematoma (n = 1), iliopsoas haematoma (n = 1), and gluteal haematoma (n = 1), and four men (mean age 72 years) with iliopsoas haematoma (n = 2), hemorrhage in the small intestine (n = 1), and splenic parenchyma (n = 1), which were successfully treated by 11 sessions of TAE of inferior phrenic artery (n = 2), hepatic artery branches (n = 1), splenic artery (n = 1), ileal artery (n = 1), inferior epigastric artery (n = 1), gluteal artery (n = 2), lumbar artery (n = 4) and iliolumbar artery (n = 2).

Haematomas of the unusual sites such as the small intestine, the small intestine, hepatic subcapsular region and splenic parenchyma were included in 3 of 9 patients (33%). In the case that resulted in haematoma of the hepatic subcapsular region, HELLP syndrome named for 3 features of the disease (hemolysis, elevated liver enzyme levels, and low platelet levels), a life-threatening condition that can potentially complicate pregnancy, cannot be excluded. In that case, however, there was no sign of severe preeclampsia or eclampsia’s seen in HELLP syndrome.

**Image Findings of NAH**

Many authors described that the gold standard for diagnosis of NAH is computed tomography [5-9], and ultrasonography can be used in follow-up.

Zissin R, et al. [5] described the computed tomography (CT) and angiographic findings of life-threatening extraperitoneal haemorrhage complicating anticoagulant therapy treated with transcatheter arterial embolization (TAE) in four consecutive patients with large, extraperitoneal anticoagulant-related haematomas, reporting that extraperitoneal anticoagulant-related haematomas demonstrated on CT as extended rectus sheath
haematoma in three and expanding iliopsoas haematoma in one, were successfully treated by TAE of the inferior epigastric (n = 3) and lumbar artery (n = 1).

In our series, unenhanced CT demonstrated the haematomas in all the patients (100%), and contrast-enhanced CT diagnosed as having active arterial bleeding within the haematoma requiring TAE in 8 of 9 patients (89%). The remaining one having splenic haematoma was referred to angiography because of haemodynamic instability. Unenhanced CT has proved an excellent modality for the diagnosis of coagulopathy-related haematomas.

Contrast-enhanced CT has an important role in detecting active bleeding that provides an indication for angiographic therapy and the pre-interventional planning with determination of the targeted vessels and extent of active bleeding. We suggest that unenhanced CT and contrast-enhanced dynamic scan should be performed concurrently, unless contraindicated.

**Treatment for NAH with Coagulopathy**

The treatment of choice is nonsurgical therapy because RSH is a self-limited condition. It is reported that the conservative treatment such as rest, ice application, and withdrawal of the anticoagulant therapy successfully stopped the bleeding [6].

Surgical intervention should be reserved for cases with hemodynamic instability. Recently arteriography with selective embolization of the epigastric arteries is the first therapeutic option. In our study, we were able to stop bleeding by the superselective embolization using the combination of gelatine sponge: there were no cases treated by NBCA. This is because the bleeding was slow-flow and small, and the sites of extravasation was relatively peripheral arterial branches. As far as we searched, there is no report of case with NAH treated with use of N-butyl cyanoacrylate (NBCA).

In our series, two of the nine patients recurred in the contralateral sites of muscles which arose asynchronously. In view of the clinical scenarios, we performed a diagnostic and therapeutic angiography of the bleeding vessels by TAE. To the best of our knowledge, these two cases are the first cases concurrently arose. Murena L, et al. [7] reported bilateral iliopsoas haematomas that concurrently arose during anticoagulant therapy, especially with heparin and warfarin and were successfully treated by TAE for bilateral fourth right lumbar artery trunk. In patients with coagulopathy, it is suggested that the NAH can repeat if the hemorrhagic condition is not improved.
Ierardi AM, et al. [6] reported technical success, clinical success and mortality were 100%, 85%, and 0%, respectively. Smithson A, et al. [8] reviewed 24 patients with spontaneous rectus sheath haematoma, and showed 19 cases (79.1%) responded to conservative management while 5 (20.8%) required interventional treatment, which consisted in an arteriography with selective embolization of the epigastric arteries in all cases. One of 19 patients who received conservative treatment died while none of 5 patients who received interventional treatment died (statistically not significant).

In our series, all patients (9 of 9 patients; 11 of 11 sessions) responded to TAE and become hemodynamically stable, however, in one of nine patients (1 of 11 sessions: 9%) developed a sudden hypovolemic shock and died three days later.

In summary, awareness of this treatment improve the outcome of patients in NAH and early application of the TAE as appropriate according to the patients' conditions is important.
## Images for this section:

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</tr>
</thead>
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<tr>
<td>Traumatic</td>
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<td>18.0%</td>
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<tr>
<td>Neoplastic</td>
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<td>16.8%</td>
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<td>13.3%</td>
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<tr>
<td>Inflammatory disease</td>
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<td>Arteriovenous malformation</td>
<td>13</td>
<td>5.1%</td>
</tr>
<tr>
<td>Others (Collagen disease, SAM)</td>
<td>18</td>
<td>7.0%</td>
</tr>
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<td>NAH associated with coagulopathy</td>
<td>9</td>
<td>3.5%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>256</strong></td>
<td><strong>100%</strong></td>
</tr>
</tbody>
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Note: Number indicate the number of cases. SAM, segmental arterial mediolysis.

**Fig. 1:** Profiles of patients who underwent emergency TAE

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| Case | Age/Gender | Underlying conditions | Symptoms | Types of Coagulopathy | ACT | CT | Involved organ | PT% (N ≥ 70) | APTT(s) (N <40) | Platelet Count (x10^4/μL) | Changes in Hb level (g/dL) |
|------|------------|-----------------------|----------|-----------------------|-----|----|---------------|-------------|---------------|----------------|----------------|----------------|
| 1    | 60/F       | After CVR             | Anemia; Subcutaneous hematoma | In ACT | Y (Caprocin) | Hematoma; CE | Rectus-sheath | 46.2       | >200          | 34.2           | 12.2→6.9       |
| 2    | 25/F       | DVT; Pregnant         | Nausea; Chest/Abdominal pain | In ACT | Y (Caprocin) | Hematoma; CE | Hepatic subcapsular region | 49.9       | >150          | 6              | 11.8→6.9       |
| 3    | 84/M       | Chronic AF; Cholangitis | Anemia; Epigastralgia; Icterus; Fever | In ACT | Y (Warfarin) | Hematoma; CE | Iliopsoas muscle (R) | 30.8      | 80.4          | 9.7            | 12.9→8.0       |
| 4    | 83/M       | After CVR             | Anemia; Melena | In ACT | Y (Warfarin) | CE | Small intestine | 13.0       | 97.5          | 7.5            | 7.6→4.9        |
| 5    | 34/M       | Hemophilia B; Chronic hepatitis C | Anemia; Epigastralgia | Inactivity of anti-hemophilic factor B | NA | Hematoma; Splenomegaly; No CE | Splenic parenchyma | 104.0   | 92.2          | 23             | 15.0→8.0       |
| 6    | 32/F       | AS                    | Anemia; Lumbago | AS | NA | Hematoma; CE | Rectus-sheath | 87.0       | 91.2          | 9.2            | 13.6→7.5       |
| 7    | 66/F       | Panniculitis of lower limbs | Subcutaneous hematoma; Shock Factor VIII inhibitor | NA | Hematoma; CE | Iliopsoas muscle (R) | 83.0      | 93.9          | 7.8            | 9.1→6.1        |
| 8    | 72/F       | MI                    | Anemia; Buttock pain | AT | NA | Hematoma; CE | Gluteal muscle (L) | 102.5     | 17.3          | 22.0           | 11.2→8.7       |
| 9    | 88/M       | MI                    | Anemia; Buttock pain | AT | NA | Hematoma; CE | Gluteal muscle (R) | 106.0     | 22.1          | 29.8           | 10.8→8.1       |
| 9    | 88/M       | Esophageal cancer     | Anemia; Lumbago | NA | NA | Hematoma; CE | Iliopsoas muscle (L) | 41.3      | 51.5          | 9.2            | 11.8→6.9       |

Note: CVR, cardiac valve replacement; DVT, deep vein thrombosis; AF, atrial flutter; AS, Antiphospholipid Syndrome; Y, yes; NA, not applicable; ACT, anticoagulant therapy; CE, contrast extravasation; R, right; L, left; MI, myocardial infarction; AT, antiplatelet therapy.

**Fig. 2:** Profiles of patients with NAH

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<table>
<thead>
<tr>
<th>Case</th>
<th>Injured Arteries</th>
<th>Number of Injured Arteries</th>
<th>Embolic Methods*</th>
<th>Embolic Materials</th>
<th>Technical Success</th>
<th>Recurrence</th>
<th>Prognosis</th>
<th>Treatment Performed in Concordance with IR</th>
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<tr>
<td>1</td>
<td>Right inferior phrenic artery</td>
<td>1</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Transfusion of packed red blood cell</td>
</tr>
<tr>
<td>2</td>
<td>Hepatic artery branches (A6, A8), Right inferior phrenic artery</td>
<td>3</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Urgent cesarean section; Gauze packing; and Administration of platelet, FFP and Viita factor</td>
</tr>
<tr>
<td>3</td>
<td>Right 2nd, 3rd and 4th lumbar arteries</td>
<td>3</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Antibiotics; and Packed red blood cell transfusion</td>
</tr>
<tr>
<td>4</td>
<td>3rd ileal artery</td>
<td>1</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Administration of FFP; Transfusion of packed red blood cell</td>
</tr>
<tr>
<td>5</td>
<td>Upper branch of splenic artery</td>
<td>3</td>
<td>PE</td>
<td>GSP + CEZ</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Administration of IX factor</td>
</tr>
<tr>
<td>6</td>
<td>Lt. inferior epigastric artery</td>
<td>1</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Transfusion of packed red blood cell</td>
</tr>
<tr>
<td>7</td>
<td>Right 2nd, 3rd and 4th lumbar arteries</td>
<td>3</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Administration of VIII factor; Steroid pulse therapy; and Immunosuppressive therapy using cyclosporine and Rituximab</td>
</tr>
<tr>
<td>8</td>
<td>Left superior gluteal artery</td>
<td>1</td>
<td>PE</td>
<td>GSP</td>
<td>Y (Recurred 2 months later)</td>
<td>Alive</td>
<td>Surgical removal of hematoma</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right superior gluteal artery</td>
<td>1</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Alive</td>
<td>Surgical removal of hematoma</td>
</tr>
<tr>
<td>9</td>
<td>Right 4th lumbar artery, Right iliolumbar artery</td>
<td>2</td>
<td>PE</td>
<td>GSP</td>
<td>Y (Recurred 2 days later)</td>
<td>Alive</td>
<td>NA</td>
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<tr>
<td></td>
<td>Left 4th lumbar artery, Left iliolumbar artery</td>
<td>2</td>
<td>PE</td>
<td>GSP</td>
<td>Y</td>
<td>N</td>
<td>Dead 5 days later</td>
<td>NA</td>
</tr>
</tbody>
</table>

GSP: gelatin sponge particle; Y: yes; N: no; NA: not applicable; PE: Proximal embolization; CEZ, Cefazolin. *TAE performed superselectively.

**Fig. 3:** Angiographic findings and effects of TAE in patients with NAH

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**Fig. 4:** Case 1. An 84 year-old male with retroperitoneal haematoma due to chronic intake of Warfarin for arterial fibrillation. A: Contrast-enhanced CT shows haematoma within the right iliopsoas muscle. Active arterial bleeding were also seen (arrows). B: Aortography shows multiple pooling (arrows). C: Selective angiography shows contrast extravasation from the 4th lumbar artery. Subsequent transcatheter embolization via the right 2nd, 3rd and 4th lumbar artery shows disappearance of the extravasation (not shown).

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Fig. 5: Case 2. A 25 year-old pregnant female with haematomas in the subcapsular region of the liver. A: CT following the emergent Caesarean section and removal of the subcapsular haematoma of the liver shows active arterial bleeding near the hepatic capsule. B: Selective angiography via the posterior branch of right inferior phrenic artery shows extravasation and pooling. C: Right hepatic arteriography also shows the contrast extravasation and pooling in the subcapsular region. Subsequent embolization using GSP was performed. Arrowhead shows the tip of the microcatheter. There is no recurrence of bleeding.

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Fig. 6: Case 3. A 70 year-old female within the left gluteal muscle with medical history of internal use of two antiplatelet drugs use of bleeding. She developed a sudden buttock pain and was transported to our institute. She has a past history of silicon infusion in the bilateral buttocks more than 20 years ago. A-B: Axial and coronal images of contrast-enhanced CT show a large haematoma within left gluteal muscle. Extravasation was noted (arrow). C-D: Angiography of the left common iliac artery shows a tiny pooling in the territory of left superior gluteal artery only in the capillary phase (D). Subsequent TAE using GSP was successfully performed.

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Fig. 7: Case 3. (Continued) Recurrent bleeding in the contralateral gluteal muscle in the long term seen in a 70 year-old female. After the treatment of the haematoma in the left gluteal muscle, internal use of two antiplatelet drugs was restarted nine days before. A 20-cm-haematoma with a right sudden buttock pain developed two months after the initial TAE (Fig.6), and she was readmitted to our hospital. A: Contrast-enhanced CT show a new haematoma within the right gluteal muscle and an extravasation was also seen within the haematoma (arrow). Note that the haematoma in the left gluteal muscle is resolving. B-C: Arterial (B) and capillary (C) phases of arteriogram via the right superior gluteal artery shows several extravasation (arrows). TAE via the right superior gluteal artery using GSP was performed. D: Angiogram following the TAE shows disappearance of the extravasation was shown. Rapid pain relief was obtained.
Fig. 8: Case 4. An 88 year-old male within the iliopsoas muscle without definitive cause of bleeding. A-B: Axial and coronal images of contrast-enhanced CT show multiple haematomas within the right iliopsoas muscle. Extravasation was also seen (arrows). Note that the left iliopsoas muscle is preserved. C: Arteriogram via the right 3rd lumbar artery shows multiple tiny pooling (arrows). D: TAE via the right 3rd lumbar artery using GSP was performed.

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Fig. 9: Case 4. (Continued) Recurrent bleeding in the contralateral iliopsoas muscle in the short term seen in an 88 year-old male. On the day after the next day after the previous TAE (Fig. 8), the vital sign fell down and contrast-enhanced CT was performed. A-B: Axial and coronal images of contrast-enhanced CT show a new haematoma within the left psoas major muscle and an extravasation was also seen within the haematoma (arrows). Note that the haematoma in the right iliopsoas muscle is resolving. C: Arteriogram via the left 4th lumbar artery does not show an overt extravasation but a tiny pooling (arrows). D: TAE via the left 4th lumbar artery using GSP was performed and disappearance of the stain was shown. The vital signs were restored normal after the 2nd TAE, but he died of sudden hypotension three days later.

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Conclusion

• It is highly important that most of the patients with NAH have a coagulopathy associated with variable causative factors.

• Superselective transcatheater embolization using gelatine sponge has been shown to be an effective and safe method for managing such haematomas when conservative treatment is insufficient, especially in patients with haemodynamic instability or with active bleeding on contrast-enhanced CT.
References