Transcatheter Arterial Embolization for Impending Rupture of an Isolated Internal Iliac Artery Aneurysm Complicated with Disseminated Intravascular Coagulation

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A 90-year-old male, with impending rupture of an isolated internal iliac artery aneurysm (IIAA) complicated with disseminated intravascular coagulation (DIC) was successfully treated with transcatheter arterial embolization (TAE). After TAE, enlargement of the aneurysm was arrested and coagulation-fibrinolytic abnormalities induced by DIC improved without severe complications. Although IIAA is relatively rare, the post-operative mortality of patients with ruptures is reportedly high. We assessed the usefulness of this procedure for impending rupture of IIAA, especially for patients in high risk groups.

Key words: interventional radiology, abdominal artery aneurysm, complication

Introduction

In contrast to abdominal aortic aneurysms (AAA) and combined aortoiliac artery aneurysms, isolated iliac artery aneurysms are uncommon, only 0.9–1.9% (1–3). Thirty-four percent of patients with iliac artery aneurysms have multiple aneurysms and 10% of these are located in the internal iliac artery (1). Despite their rarity, several reports (3–6) have suggested that isolated iliac artery aneurysms have a high risk (50–75%) for rupture even in asymptomatic cases, and have an associated high post-operative mortality rate (48%) (3). Therefore, preventive treatment is recommended. In general, surgical options have included ligation, excision, or oblitative endoaneurysmorrhaphy (4). However, since objective cases of iliac artery aneurysms are usually complicated with a high age and arteriosclerotic disease, there are many contraindicated cases for direct operative approaches. Herein, we report a patient with impending rupture of an internal iliac artery aneurysm (IIAA) complicated with disseminated intravascular coagulation (DIC), who was treated successfully with transcatheter arterial embolization (TAE).

Case Report

A 90-year-old man was admitted to our hospital due to left lower abdominal pain on May 4, 1997. Both his past history and family history were unremarkable. On admission, vital signs were normal except for a slight fever. The physical examinations were normal except for mild tenderness on the left lower abdomen without peritoneal signs or pulsations.

Hematological data showed leucocytosis (2.69x10^4/mm^3). The coagulation test (Fibrinogen) was increased to the level of 53 mg/dl. Biochemistry data showed mild liver damage (alanine aminotransferase: ALT 191 IU/l), mild renal dysfunction (Creatinine 2.0 mg/dl) and an increase of C-reactive protein (20.1 mg/dl) (Table 1).

A scout film of the abdomen on the first hospital day showed no findings of intestinal gas in the pelvic cavity and a tumorous region was suspected. Plain abdominal computed tomography (CT) revealed a clear marginal round tumorous region on the bilateral internal side of the calcified iliac artery. The right tumor was about 8–9 cm and the left one was 4.5 cm in diameter. The right tumorous region showed inhomogenous internal density (Fig. 1A).

After administration of antibiotics from the first hospital...
Table 1. Laboratory Data on Admission

| Urinalysis:                           | Peripheral blood:                        | Chemistry:                                                     | Coagulation test:       |
|--------------------------------------|------------------------------------------|                                                              |                         |
| Glucose 1+                           | Total cholesterol 164 mg/dl               | Total protein 7.0 g/dl                                       | Fibrinogen 531 mg/dl    |
| Protein 1+                           | Serum amylase 54 IU/l                    | Albumin 4.1 g/dl                                             | HPT 108%                |
| Occult 1+                            | Na 137.1 mEq/l                           | A/G 1.4                                                      |                         |
| Urinary acid 1+                      | K 4.2 mEq/l                              | AST 91 IU/l                                                 |                         |
| Triglyceride 46 mg/dl                | Cl 100.7 mEq/l                           | ALT 191 IU/l                                                |                         |
| Total cholesterol 164 mg/dl          | CRP 20.1 mg/dl                           | LDH 393 IU/l                                                |                         |
| Creatinine 2.0 mg/dl                 | TTT 1.4                                  | ALP 163 IU/l                                                |                         |
| Occult 1+                            | ZTT 3.2 U                                | γ-GTP 13 IU/l                                               |                         |
| Urinary acid 1+                      | Coagulation test:                        | Total bilirubin 1.2 mg/dl                                   |                         |
| Triglyceride 46 mg/dl                |                                          | Direct bilirubin 0.3 mg/dl                                  |                         |
| Peripheral blood:                    |                                          | BUN 30.3 mg/dl                                              |                         |
| White blood cell 26,900/mm³           |                                          |                                                              |                         |
| Red blood cell 395×10⁶/mm³            |                                          |                                                              |                         |
| Hemoglobin 12.4 g/dl                 |                                          |                                                              |                         |
| Hematocrit 36.6%                     |                                          |                                                              |                         |
| Platelet 24.2×10⁹/mm³                |                                          |                                                              |                         |


Figure 1. Abdominal CT. A) Plain abdominal CT on the first hospital day. B) Enhanced abdominal CT on the 8th hospital day. C) Crescent-shaped enhanced area (arrows).
Figure 2. Abdominal aortography on the 10th hospital day. Note the vague and irregular enhancement toward the inside of the aneurysm (arrow).

In the early stage of hospitalization, we recognized rapid aggravation of anemia, thrombocytopenia and an increase in molecular DIC markers (D-dimer, thrombin-anti thrombin III complex; TAT) in contrast to the improvement of inflammatory changes. On the 8th hospital day, DIC was diagnosed and treatment was started. Enhanced abdominal CT on the same hospital day showed round intensified enhancement at the center of the right tumorous region, which was confirmed to be dilated IIAA (Fig. 1B). The high density regions on plain abdominal CT (Fig. 1A) and inhomogenous enhancement around the intensified enhanced region (Fig. 1B) suggested an internal hemorrhage. Furthermore, we observed a crescent-shaped enhanced area of the vascular wall on the upper-internal side (Fig. 1C), which was considered as a sign of intramural hematoma (7), and diagnosed it as an impending rupture of IIAA.

Abdominal aortography on the 10th hospital day showed marked arteriosclerotic changes in both the aorta and iliac artery (Fig. 2A). A hemorrhage was confirmed with the findings of totally dilated right IIAA from the bifurcation with vague and irregular enhancement toward the inside of the aneurysm (Fig. 2B) and TAE was selected for treatment (Fig. 3).

Discussion

As symptomatic cases of IIAA generally show a large aneurysmal diameter (3, 4), repair has been recommended when the diameter exceeds 3 cm even in asymptomatic patients (5). With the progress in interventional radiology, percutaneous treatment of aneurysms based on selective TAE provides alternative approaches (8-10). Recently, Cynamon et al (11) reported a patient with IIAA successfully treated with stented graft and embolization coils, a procedure which can obviate an extra-anatomic bypass. However, open femoral artery exposure is required. We chose selective TAE instead of direct operative approaches due to the emergent state with impending rupture, and complications of DIC, and his age. As materials for TAE, metallic coils and pieces of gel foam are recommended (12). In this case, it was difficult to embolize selectively at both the proximal and distal portions of the aneurysm because the aneurysm arose directly from the bifurcation of the internal iliac
artery and was spindle-shape.

Furthermore, there was a risk that the TAE materials would flow into the external iliac artery due to the massive blood flow from the markedly enlarged aneurysm. Therefore, we prepared a metallic guide wire (0.035 inch) which was pulled out of a wick and advanced 120 cm into the aneurysm. In addition, forty-four metallic coils were used. Then, complete interruption of blood flow was achieved.

Su et al (13) have reported a rare case of deep venous thrombosis with pulmonary embolus 4 months after successful TAE for IIAA. Although only a slight fever after TAE was observed in this case, the potential of long-term complications of venous thrombosis should be followed up.

After TAE, both arrest of aneurysm enlargement and improvement of the coagulation-fibrinolytic abnormality with DIC were achieved. The clinical course of DIC and impending rupture of IIAA developed in parallel, suggesting that secondary hyperfibrinolysis in IIAA contributed to the DIC state.

Recently, IIAA could be diagnosed by CT. However, IIAA is located in the pelvic cavity, which may show various clinical signs related to the adjacent organs including the ureter, common iliac vein, sigmoid colon, and the lumbosacral nerve (5). The symptoms include lower abdominal pain, intestinal hemorrhage, unilateral leg edema, constipation, hydronephrosis, and lumbosacral nerve pain, and should be recognized as differential diagnoses. We demonstrated here that selective TAE is effective, especially for patients with impending rupture of large IIAA at a high risk, as an alternative to direct operative approaches.

References