

AORTIC AND SUPERIOR MESENTERIC ARTERY THROMBOSIS IN ANTITHROMBIN III DEFICIENCY-DIAGNOSTIC AND THERAPEUTIC CHALLENGES IN CONSERVATIVE MANAGEMENT

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Abstract: Introduction: Thrombosis of large arterial vessels, such as the abdominal aorta and superior mesenteric artery, is a rare but serious condition that requires timely diagnosis and appropriate management. One of the risk factors is antithrombin III (AT III) deficiency, a rare coagulation disorder that increases the likelihood of thrombosis. While arterial thromboses are less common than venous ones, they can have significant clinical consequences.

Case report: We present the case of a 39-year-old woman hospitalized due to sudden abdominal pain. Diagnostic imaging, including a contrast-enhanced CT scan of the abdomen and pelvis and CT angiography, revealed thrombosis of the distal abdominal aorta and superior mesenteric artery. Laboratory testing confirmed low AT III levels, while tests for hereditary thrombophilias were negative, suggesting a likely acquired deficiency.

The patient was treated conservatively with AT III concentrate, low-molecular-weight heparin, and oral anticoagulation. Therapy was complemented with cardioprotective and gastroprotective medications, as well as physical rehabilitation. During hospitalization, the patient remained hemodynamically stable, and symptoms gradually resolved. Follow-up imaging after several months demonstrated complete recanalization of the affected vessels. Long-term monitoring over two years confirmed stable clinical status and absence of recurrent thrombosis.

Conclusion: This case highlights the importance of early diagnosis, identification of the underlying cause, and carefully implemented conservative management in patients with AT III deficiency. It demonstrates that even in extensive arterial thrombosis, conservative management can preserve organ function

and achieve a favorable outcome without the need for surgical intervention.

Keywords: abdominal aorta thrombosis, superior mesenteric artery thrombosis, antithrombin III deficiency, conservative treatment, anticoagulation therapy.

INTRODUCTION

Thrombosis of large arterial vessels, particularly the abdominal aorta and mesenteric arteries, represents a rare but potentially life-threatening condition that requires urgent diagnosis and a coordinated multidisciplinary therapeutic approach. In elderly patients, the most common causes include atherosclerosis, aneurysmal changes, or cardiogenic sources of embolism, whereas in younger individuals such manifestations are often associated with congenital or acquired coagulation disorders. Although rare, antithrombin III deficiency significantly increases the risk of thromboembolic events, with venous thromboses being far more common. Arterial thromboses, though less frequent, represent a serious clinical challenge with potentially severe outcomes. Timely recognition of such conditions, supported by detailed laboratory and radiological diagnostics, is crucial for therapeutic planning and prevention of complications (1, 2).

The presented case describes a young female patient with extensive thrombosis of the abdominal aorta and superior mesenteric artery. The aim of this report is to emphasize the importance of early diagnosis, precise determination of the etiology of thrombosis, and an effective conservative therapeutic approach, which in this case enabled preservation of vital organ function and achievement of a favorable long-term outcome.

CASE PRESENTATION

A 39-year-old female patient was admitted to the Department of General Surgery on April 10, 2022, due to the sudden onset of severe abdominal pain. Upon admission, she was conscious, communicative, afebrile, and hemodynamically stable, with normal vital parameters. Her medical history was unremarkable, with no chronic illnesses, previous surgeries, or similar symptoms reported.

A contrast-enhanced CT scan of the abdomen and pelvis revealed thrombotic material in the distal portion of the abdominal aorta extending into the superior mesenteric artery (SMA), with signs of reduced perfusion in certain segments of the small intestine but without evidence of perforation or ileus. No pathological changes were observed in the parenchymal abdominal organs (Figure 1 and 2).

CT angiography confirmed thrombosis of the distal abdominal aorta and superior mesenteric artery. According to the initial radiology report, subocclusive changes of several distal branches in the lower extremities were described. However, since the original images are unavailable, these findings could not be independently verified.

A follow-up abdominal CT scan performed on April 12, 2022, confirmed the presence of the abdominal thrombosis without progression or signs of acute complications. A moderate amount of free fluid was noted, without signs of peritonitis.

Laboratory analyses revealed a decreased level of antithrombin III, leading to the initiation of therapy with antithrombin concentrate (Kybernin). In the initial phase, anticoagulant therapy with low-molecular-weight heparin was administered, followed by a transition to oral anticoagulant therapy (rivaroxaban). The therapeutic protocol also included cardioprotective medication (acetylsalicylic acid), gastroprotective therapy, and symptomatic analgesia.

During hospitalization, the patient remained hemodynamically stable, without signs of progression of abdominal symptoms. She repeatedly complained of pain and weakness in the right foot; clinical examination confirmed limited dorsiflexion. Physical therapy was initiated to preserve muscle strength and function, resulting in gradual clinical improvement.

The patient was discharged on April 21, 2022, in good general condition, afebrile, and with a normal abdominal examination. Continued anticoagulant therapy, regular hematologic follow-up due to suspected coagulation disorder, and ongoing supervision by a vascular surgeon and physiatrist were recommended.

At a follow-up examination in September 2022, CT angiography demonstrated complete revasculari-



Figure 1. CT aortography demonstrating thrombotic occlusion of the abdominal aorta extending into the superior mesenteric artery (SMA). The arrow indicates an intraluminal filling defect corresponding to acute thrombus formation. Absence of contrast opacification distal to the thrombus is consistent with complete arterial occlusion.

(The image is from the authors' archive.)



Figure 2. Follow-up abdominal CT performed two days after admission demonstrates the presence of thrombotic masses, with no evidence of progression compared to the previous study and preserved viability of intra-abdominal organs.

(The image is from the authors' archive.)

zation. The patient still reported a subjective burning sensation in the right leg but had no new symptoms or signs of acute ischemia. Continuation of the current therapy for an additional two months was advised, followed by re-evaluation.

In addition to the above investigations, genetic testing for hereditary thrombophilias (Factor V Leiden, prothrombin G20210A mutation, MTHFR, and PAI-1) was performed, all yielding negative results.

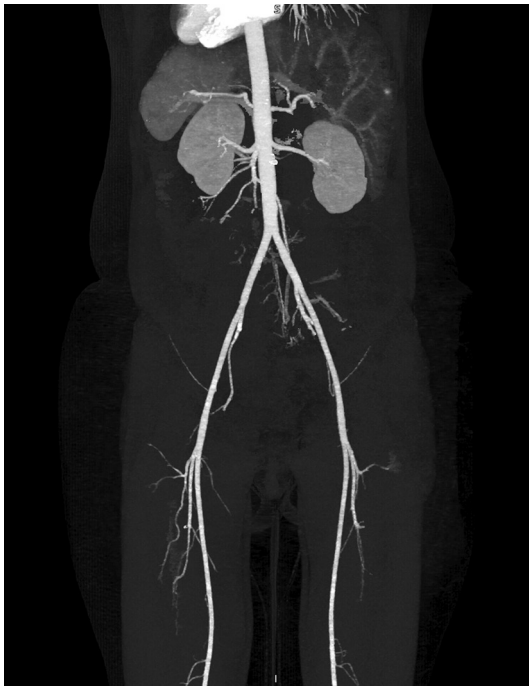


Figure 3. Follow-up CT aortoarteriography after two years demonstrates complete recanalization of all abdominal and lower extremity vessels. (The image is from the authors' archive)

The patient was subsequently followed regularly over the next two years, with normal laboratory findings, including antithrombin III levels, and without any recurrence of thromboembolic events (Figure 3).

DISCUSSION

Antithrombin III (AT III) deficiency is a rare but clinically significant disorder of hemostasis that increases the risk of thromboembolic events (3). Congenital forms usually manifest at a younger age, whereas acquired forms are more commonly observed in the context of acute thrombotic episodes, liver disease, nephrotic syndrome, or disseminated intravascular coagulation (DIC) (4, 5, 6).

In our patient, AT III deficiency was detected during an episode of acute thrombosis of the abdominal aorta and mesenteric artery. Genetic testing for hereditary thrombophilias (Factor V Leiden, prothrombin G20210A mutation, MTHFR, and PAI-1) was negative, suggesting a likely acquired rather than congenital form of the disorder. During a two-year follow-up period, AT III levels were regularly monitored and consistently within the reference range. These findings indicate that the condition was not a classical hereditary thrombophilia, suggesting either an acquired deficiency or a rare congenital form undetectable by standard genetic panels.

Regardless of etiology, the negative thrombophilia results and stable AT III levels highlight the impor-

tance of continuous hematologic monitoring and an individualized anticoagulant regimen, which reduced the risk of thrombotic recurrence and contributed to a favorable long-term outcome.

Arterial thrombosis of the abdominal aorta and mesenteric arteries is rare in younger individuals and is typically associated with atherosclerosis, cardioembolic sources, or coagulation disorders (5, 6, 7). In this case, the sudden onset of abdominal pain combined with findings from CT angiography enabled early diagnosis and initiation of targeted therapy. Despite extensive thrombotic involvement, the patient was managed conservatively with administration of antithrombin concentrate, low-molecular-weight heparin, and subsequent transition to rivaroxaban. Activated partial thromboplastin time, thrombin time, prothrombin time, anti-Xa activity, and D-dimer levels were regularly monitored.

This approach led to clinical stabilization, symptom regression, and complete vascular recanalization confirmed by follow-up CT angiography. The significance of this case lies in the successful conservative therapeutic approach, which effectively controlled the thrombotic process without the need for surgical intervention. The combination of antithrombin replacement, anticoagulant therapy, and close coagulation profile monitoring represents a standardized and safer treatment strategy for patients with AT III deficiency and multiple arterial thromboses, particularly in those with hemodynamic stability and no signs of acute ischemia of vital organs (1, 3, 6, 8). This approach allows gradual thrombus resolution, vascular lumen recanalization, and reduction of complications such as intestinal or limb infarction.

At the same time, multidisciplinary management involving a hematologist, vascular surgeon, and physiatrist ensures optimal coagulation control, neuromuscular function monitoring, and timely intervention in case of clinical deterioration. It is noteworthy that, despite initial neuromuscular symptoms affecting the right foot, the patient achieved good functional recovery with appropriate physical therapy. Long-term follow-up over two years demonstrated the absence of recurrent thrombosis and normal laboratory findings, confirming the efficacy of the conservative approach in this case.

This case highlights the importance of early diagnosis, individualized treatment planning, and multidisciplinary supervision in patients with suspected coagulation disorders. It also demonstrates that even extensive arterial thromboses can be successfully managed conservatively when therapy is carefully implemented and patients are closely monitored, thereby avoiding surgical intervention.

Table 1. *Conservative Therapy of Arterial Thrombosis in Antithrombin III Deficiency*

Therapy / Medication	Dosage / Administration	Duration	Notes / Purpose
Kybernin (Antithrombin III concentrate)	Individually adjusted dose according to AT III level	During the acute phase of hospitalization	Replacement of antithrombin III deficiency and stabilization of coagulation
Low-Molecular-Weight Heparin (LMWH)	Subcutaneous, therapeutic dose based on body weight	Initial phase of hospitalization	Rapid anticoagulation and prevention of thrombus progression
Oral anticoagulant	Initial dose titrated according to coagulation profile values	Long-term, per hematologic supervision	Maintenance of therapeutic dose and prevention of thrombotic recurrence
Acetylsalicylic Acid (ASA)	Standard cardioprotective dose (75–100 mg daily)	Long-term	Additional antithrombotic protection and cardioprotection
Gastroprotective therapy (PPI)	Standard dose	As needed with ASA / anticoagulants	Prevention of gastrointestinal complications
Analgesics	As needed	Symptomatic	Pain control
Physical therapy	Individual exercise program for the lower extremity	During and after hospitalization	Preservation of muscle strength and foot function, rehabilitation

Table 1 was created based on data synthesized from references (2, 3, 7, 9, 10, 11).

Table 2. *Surgical and Endovascular Treatment of Arterial Thrombosis: Indications and Methods*

Type of Intervention	Indications	Methods / Procedures	Notes / Objective
Conventional (Open) Surgery	- Acute ischemia of vital organs or limbs - Failure of conservative or endovascular therapy - Extensive or multiple thromboses - Anatomical obstacles preventing endovascular access	- Thrombectomy - Endarterectomy - Bypass procedures	Direct arterial recanalization, restoration of perfusion, and achievement of long-term vascular patency; associated with higher surgical risk and longer recovery period
Endovascular Procedures (Minimally Invasive)	- Subacute or acute thrombosis with preserved hemodynamic stability - High surgical risk - Partial thrombosis or segments accessible via catheter	- Catheterdirected thrombolysis - Percutaneous thrombectomy - Stent placement	Minimally invasive; reduce hospitalization time; often combined with anticoagulant therapy; suitable for patients with multiple or partially accessible arterial thromboses

Table 2 was created based on clinical concepts and therapeutic principles described in references (7, 8, 11).

Although conservative treatment was successful in this case, surgical management remains an important option for patients presenting with signs of acute ischemia of vital organs or limbs, progressive thrombosis, or failure of conservative therapy. Indications for surgery include acute intestinal infarction, limb gangrene, massive thrombosis compromising hemodynamic stability, or the presence of embolic fragments posing a risk of distal arterial occlusion. Surgical approaches may include thrombectomy, endarterectomy, bypass grafting, or other reconstructive vascular

procedures, depending on the localization and extent of thrombosis (9, 10). Decision-making regarding surgical intervention requires a multidisciplinary assessment involving vascular surgeons, hematologists, and radiologists to evaluate risk–benefit ratios and determine the optimal timing of surgery.

Literature reports indicate that, when feasible, an early combination of anticoagulant therapy and selective surgical intervention can improve outcomes and reduce morbidity, especially in patients with extensive or multiple arterial thromboses (Table 1).

In recent decades, endovascular procedures have emerged as an important alternative to open surgery in the treatment of arterial thromboses, particularly in patients with high surgical risk or limited resectable segments. Techniques such as catheter-directed thrombolysis, percutaneous thrombectomy, and stent placement enable direct thrombus dissolution or removal, restoration of perfusion, and reduction of long-term tissue damage. Endovascular approaches are typically used in subacute or acute thromboses where there is a vital threat to an organ or limb but hemodynamic stability can be maintained. These minimally invasive methods, often combined with systemic or local anticoagulant therapy, enhance treatment efficacy and reduce the need for open surgical procedures (1, 5, 9) (Table 2).

According to the literature, timely application of endovascular interventions can significantly improve outcomes, shorten hospitalization, and decrease morbidity in patients with extensive arterial thromboses (11).

CONCLUSION

The presented case demonstrates that antithrombin III deficiency, although rare, can lead to extensive arterial thrombosis and severe clinical manifestations even in younger patients. Timely diagnosis and implementation of a conservative therapeutic approach resulted in a favorable outcome without the need for surgical intervention. Long-term follow-up confirmed clinical stability and absence of recurrence, emphasizing

the importance of early recognition and appropriate management of such coagulation disorders.

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Note: Artificial intelligence was not utilized as a tool in this study.

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Sažetak

TROMBOZA AORTE I GORNJE MEZENTERIJALNE ARTERIJE U SKLOPU DEFICITA ANTITROMBINA III-DIJAGNOSTIČKI I TERAPIJSKI IZAZOVI KONZERVATIVNOG LEČENJA

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Uvod: Tromboza velikih arterijskih krvnih sudova, kao što su abdominalna aorta i gornja mezenterijalna arterija, predstavlja retko, ali ozbiljno stanje koje zahteva pravovremenu dijagnostiku i adekvatan terapijski pristup. Jedan od faktora rizika je deficit antitrombina III (AT III), redak poremećaj koagulacije koji povećava verovatnoću nastanka tromboza, pri čemu su arterijske tromboze ređe, ali potencijalno ozbiljnije.

Prikaz slučaja: Predstavljamo slučaj 39-godišnje žene koja je hospitalizovana zbog naglog abdominalnog bola. Dijagnostika, uključujući CT abdomena i

male karlice i CT angiografiju, otkrila je trombozu distalnog dela abdominalne aorte i gornje mezenterijalne arterije. Laboratorijskim testom potvrđen je nizak nivo AT III, dok su testovi na nasledne trombofilije bili negativni, sugerišući verovatnost stečenog oblika deficita.

Pacijentkinja je lečena konzervativno, uz primenu koncentrata antitrombina, niskomolekularnog heparina i oralnog antikoagulanta. Terapija je dopunjena kardioprotektivnim i gastroprotektivnim lekovima, kao i fizikalnom rehabilitacijom. Tokom hospitalizacije stanje pacijentkinje bilo je stabilno, a simptomi su se

postepeno povukli. Kontrolna dijagnostika nakon nekoliko meseci pokazala je potpunu rekanalizaciju zahvaćenih krvnih sudova, a dugoročno praćenje tokom dve godine potvrdilo je stabilnost kliničkog stanja i odsustvo recidiva tromboze.

Zaključak: Ovaj slučaj ističe važnost rane dijagnostike, identifikacije uzroka tromboze i pažljivo sprovedenog konzervativnog lečenja kod pacijenata

sa deficitom AT III. Pokazuje da, i kod opsežnih arterijskih tromboza, takav pristup može obezbediti očuvanje funkcije organa i povoljan ishod bez potrebe za hirurškom intervencijom.

Cljučne reči: tromboza abdominalne aorte, tromboza gornje mezenterijalne arterije, deficit antitrombina III, konzervativno lečenje, antikoagulantna terapija.

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