

Nodular Polyarteritis With Superior Mesenteric Artery Involvement: A Report of a Complex Case

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Abstract

Polyarteritis nodosa (PAN) is a chronic autoimmune vasculitis characterized by inflammation and structural damage to medium-sized blood vessels throughout the body. Its clinical presentation is heterogenous and often complex, with potential progression to disability. In this study, we report a case of a 73-year-old male with PAN who presented with acute abdominal pain. Computed tomography angiography (CTA) revealed a retroperitoneal hematoma and multiple aneurysms of the intestinal arteries. Emergency digital subtraction angiography (DSA) showed multiple aneurysms, stenoses, and dissections of the superior mesenteric artery (SMA). The diagnosis of PAN was confirmed. The patient experienced recurrent hemorrhage, which was managed by multiple arterial embolization procedures to achieve hemostasis. Subsequently, administration of methylprednisolone led to favorable clinical outcomes. This case underscores the importance of considering PAN in patients with acute abdominal pain and highlights the efficacy of staged endovascular and immunosuppressive therapy.

Key words: periarteritis nodosa; acute abdomen; superior mesenteric artery aneurysms; therapeutic embolization; endovascular procedures; case report

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Introduction

Polyarteritis nodosa (PAN) is a rare, systemic vasculitis primarily affecting medium- and small-sized arteries. It is an autoimmune disease with a global incidence ranging from 2.4 to 9.7 per million individuals (Kanecki et al, 2019; Wolff et al, 2023). PAN typically occurs in middle-aged and elderly populations, with incidence gradually increasing with age (Watts et al, 2000). The clinical manifestations of PAN are heterogeneous, commonly including fatigue, weight loss, and fever, whereas severe, acute abdominal pain occurs in a minority of cases (Enabi et al, 2024; Jennette et al, 2013). Abdominal involvement in PAN is characterized by systemic vascular pathology, with abdominal pain and gastrointestinal bleeding being the most frequent manifestations, both of which are closely associated with poor prognosis. In patients presenting with acute abdominal pain, especially those with concomitant systemic inflammation or vascular abnormalities, PAN should be strongly considered. Early diagnosis through imaging and laboratory evaluation is crucial for improving clinical outcomes (Karadag et al, 2024). Aneurysms involving the superior mesenteric artery (SMA) in PAN patients are infrequently reported (Kaczynski, 2012; Rohmer et al, 2023).

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In 1990, the American College of Rheumatology proposed ten classification criteria for PAN, primarily emphasizing clinical symptoms (Lightfoot et al, 1990). The 2012 Chapel Hill Consensus Conference highlighted the importance of imaging evidence in PAN diagnosis (Jennette et al, 2013). Currently, PAN diagnosis is primarily based on clinical presentation, imaging studies, and histopathological examination, while microbiological and genetic testing serve as complementary tools (Hočevar et al, 2021).

Management of PAN primarily involves systemic corticosteroids, with the addition of immunosuppressive agents, such as cyclophosphamide, for severe or refractory cases. Life-threatening complications, including aneurysm rupture, require prompt interventional or surgical hemostasis, followed by gradual initiation of immunosuppressive therapy once the patient is stabilized. Multidisciplinary collaboration and a staged treatment approach are especially crucial for complex cases (Hočevar et al, 2021; Lin et al, 2021).

The current case differs from previous reports of PAN in presenting with acute abdominal pain, multiple mesenteric artery aneurysms and dissections, recurrent massive hemorrhage, and the need for multiple interventional and surgical procedures. In this study, we report a PAN patient who presented primarily with acute abdominal pain, with digital subtraction angiography (DSA) revealing multiple SMA aneurysms, stenoses, and dissections. Despite the critical condition of the patient, effective control was achieved through exploratory laparotomy, endovascular embolization, and corticosteroid therapy, resulting in gradual clinical improvement. A detailed analysis of this case provides clinicians with a better understanding of the abdominal manifestations of PAN, broadening the diagnostic approach for patients with acute abdominal pain and highlighting PAN as a potential underlying etiology. We hope that the summary and analysis of this case can provide valuable reference and clinical experience for further research on the early diagnosis and treatment of PAN. This report was prepared following the CARE guidelines to ensure comprehensive and transparent case reporting, and the CARE checklist has been provided as Supplementary Material (Gagnier et al, 2013).

Case Report

Basic Information

A 73-year-old male presented with a 15-hour history of sudden-onset abdominal pain without identifiable precipitating factors. The pain progressively worsened over the subsequent 8 hours and remained unrelieved. An initial computed tomography (CT) scan performed at the referring hospital suggested intra-abdominal hemorrhage, prompting transfer to our facility for further evaluation and management. The patient had no significant past medical history, including hypertension, cardiovascular disease, diabetes mellitus, cerebrovascular disease, or other severe illnesses, and reported no known drug or food allergies. Born and residing in Jinhua, he was a farmer with a primary school education. He denied any history of smoking, alcohol consumption, or exposure to endemic water sources. He was married with three healthy children (two sons and one daughter). There was no known

family history of hereditary or similar disorders. On admission, he was alert and oriented, with a body temperature of 36.5 °C, pulse rate of 93 beats per minute, respiratory rate of 20 breaths per minute, and blood pressure of 110/71 mmHg.

Clinical Features

On the evening of 9 February 2024, the patient was admitted to the emergency department with a working diagnosis of "acute intra-abdominal hemorrhage". On admission, he was alert but appeared lethargic and had a pale complexion. Physical examination revealed tenderness and deep palpation pain in the upper abdomen, without rebound tenderness. Post-admission abdominal computed tomography angiography (CTA) identified a retroperitoneal hematoma and multiple mesenteric artery aneurysms (Fig. 1).

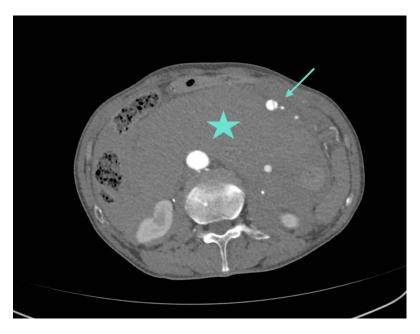


Fig. 1. Abdominal contrast-enhanced computed tomography scan. The asterisk indicates a retroperitoneal hematoma, and the arrow denotes a mesenteric artery dissection.

Laboratory Results

Upon admission, the patient exhibited marked inflammatory responses, accompanied by anemia. Inflammatory markers, including white blood cell count and high-sensitivity C-reactive protein (hs-CRP), were elevated, indicating acute systemic inflammation. Hemoglobin and red blood cell counts were low, suggesting anemia. Serum creatinine was mildly decreased. Overall, the patient presented with severe inflammation and systemic stress at the time of admission. The overall clinical condition at discharge had improved compared to that at admission. Due to the patient's stabilized condition, complement component C3 and C4, as well as lactate dehydrogenase, were not measured post-immunosuppressive therapy or on the day of discharge (Table 1).

Table 1. Laboratory test results upon admission, post-immunosuppressive regimen, and at discharge.

Parameter	Reference range	Admission (10 February 2024)	Post-immunosuppressive regimen (19 February 2024)	Discharge (19 March 2024)
White blood cell count (×10 ⁹ /L)	3.5–9.5	17.47	16.45	5.41
Neutrophils ($\times 10^9/L$)	40–75	16.92	97.6	90.2
Lymphocytes (×10 ⁹ /L)	20-50	0.24	0.2	8.7
High-sensitivity C-reactive protein (mg/L)	<3	40.83	45.17	< 0.5
Hepatitis B surface antibody	Negative	Positive	Positive	Positive
Hepatitis B surface antigen	Negative	Negative	Negative	Negative
Red blood cell count ($\times 10^{12}/L$)	4.3 - 5.8	3.61	2.21	3.51
Hemoglobin (g/L)	130–175	101	63	105
Hematocrit (%)	40–50	30.8	20.3	32.8
Complement C3 (g/L)	0.9 - 1.8	0.64		
Complement C4 (g/L)	0.1 - 0.4	0.1		
Serum creatinine (µmol/L)	40–135	31.6	44.1	32.1
Total bilirubin (μmol/L)	3–22	25.9	61.9	37.4
Lactate dehydrogenase (U/L)	120–246	362	2105	
Total protein (g/L)	63–82	56.6	57.5	74.9
Albumin (g/L)	35–50	28.1	34.4	32.6

Table 2. Inflammatory and coagulation markers during glucocorticoid therapy and a bleeding episode.

Parameter	Reference range	During bleeding episode (26 February 2024)
PCT (ng/mL)	< 0.05	0.07
IL-6 (pg/mL)	0–10	24.15
Prothrombin time (s)	9.4–12.5	13.20
International normalized ratio	0.85 - 1.15	1.14
Prothrombin time activity (%)	70.0-130.0	78.00
Activated partial thromboplastin time (s)	25.1–36.5	31.00
Thrombin time (s)	10.3–16.6	17.50
Fibrinogen (g/L)	2.38-4.98	1.37
D-dimer (μ g/L)	< 500	8588.00

IL-6, interleukin-6; PCT, procalcitonin.

Additional markers, including procalcitonin (PCT) and interleukin-6 (IL-6), were assessed around the time of gastrointestinal bleeding episodes to monitor inflammation further during glucocorticoid therapy. These markers provided insights into ongoing disease activity and potential infection risks (Table 2).

Imaging Diagnosis

Emergency contrast-enhanced three-dimensional (3D) CT angiography of the chest and abdominal aorta revealed a superior mesenteric artery (SMA) dissection, with distal branch dilation and localized expansion consistent with multiple aneurysmal extensions (Fig. 2). A large retroperitoneal hematoma was identified, accompanied by imaging features consistent with vascular wall inflammation, including irregular vessel wall thickening and enhancement, characteristic of systemic vasculitides such as polyarteritis nodosa (PAN) (Jennette et al., 2013).

The diagnosis of PAN was established based on clinical presentation, laboratory findings, and CTA features, consistent with the 2012 Revised International Chapel Hill Consensus Conference Nomenclature of Vasculitides (Jennette et al, 2013). According to the Chapel Hill criteria, PAN is defined as necrotizing vasculitis of medium or small arteries without glomerulonephritis or small-vessel involvement, often presenting with aneurysms and stenoses. In this case, the patient exhibited hallmark features of PAN, including multiple SMA aneurysms and dissections on CTA, acute abdominal pain indicative of visceral involvement, and elevated inflammatory markers (e.g., hs-CRP 40.83 mg/L, white blood cell count (WBC) 17.47×10^9 /L), consistent with systemic inflammation.

These findings satisfy the Chapel Hill diagnostic criteria for PAN, as CTA demonstrated necrotizing vasculitis of medium-sized arteries with aneurysms and dissections, without evidence of small-vessel vasculitis or glomerulonephritis. Notably, deviations from typical PAN presentations included the absence of involvement of other organs (e.g., kidneys, skin, or peripheral nerves) and lack of histopathological confirmation. Due to the patient's critical condition and recurrent life-threatening

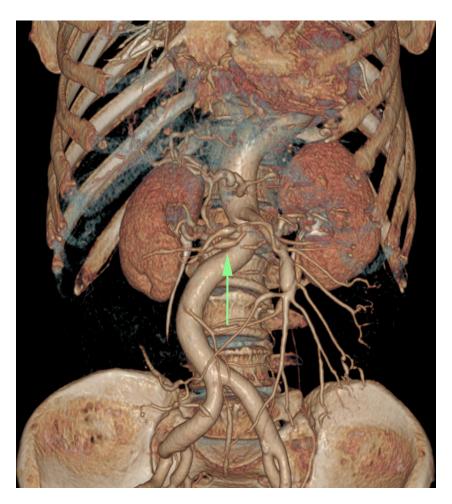


Fig. 2. Emergency contrast-enhanced three-dimensional (3D) CT demonstrating superior mesenteric artery (SMA) dissection with distal branch dilation, suggesting an aneurysm formation. The arrow indicates the bleeding vessel.

hemorrhages, tissue biopsy was considered clinically unsafe and was not performed. This approach aligns with expert consensus, which allows clinical and imaging-based diagnosis in such high-risk scenarios (Hočevar et al, 2021).

Follow-up CTA imaging post-treatment was not obtained because the patient refused further examinations after clinical stabilization, limiting direct visualization of vascular wall inflammation resolution. However, significant improvements in inflammatory markers and clinical symptoms provided indirect evidence of therapeutic efficacy (Table 1).

Treatment Process

Upon admission (9 February 2024), DSA revealed a SMA dissection with aneurysmal dilation of its branches, but no active bleeding. The patient underwent exploratory laparotomy on 10 February 2024, at 01:45, during which a 15×20 cm retroperitoneal hematoma was removed. Coil embolization of the mesenteric artery branches was performed, temporarily stabilizing the condition of the patient.

On the seventh postoperative day (16 February 2024, 20:20), the patient experienced a sudden exacerbation of abdominal pain. Repeat DSA revealed nodular changes in distal SMA vessels, accompanied by dissection and multiple aneurysms.

Rupture of a middle colic artery branch aneurysm resulted in bleeding (Fig. 3), which was successfully controlled with percutaneous embolization of the mesenteric artery using microcoils. The following day, due to worsening hemorrhagic shock, complete embolization of the middle colic artery was performed (Fig. 4), using microcoils and placement of a metal stent (Boston Scientific, $14 \text{ mm} \times 30 \text{ cm}$) within the true lumen of the SMA dissection to restore blood flow, while the false lumen was embolized.

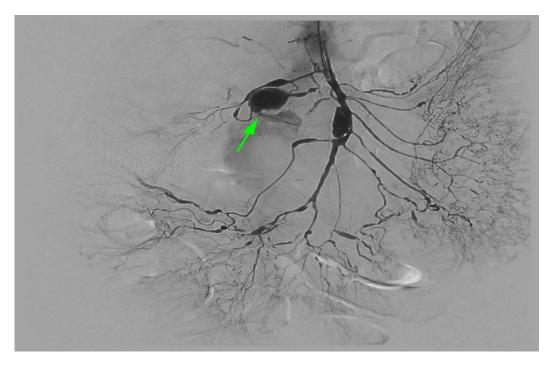


Fig. 3. Emergency digital subtraction angiography showing active bleeding at the ruptured middle colic artery branch aneurysm (the arrow).

On the ninth postoperative day (19 February 2024, 15:15), a second exploratory laparotomy confirmed restoration of colonic blood supply. Approximately 1200 mL of intraperitoneal blood and small bowel mesenteric hematoma were evacuated, and hemostasis was achieved in collaboration with the vascular and hepatobiliary surgical teams. Based on angiographic findings, clinical features, and laboratory results, the patient was diagnosed with PAN. Immunosuppressive therapy was initiated with intravenous methylprednisolone at 60 mg/d.

One week later (26 February 2024, 04:30), the patient developed recurrent abdominal pain with frequent melena. Laboratory evaluation showed elevated inflammatory markers, including interleukin-6 (IL-6) at 24.15 pg/mL (reference range 0–10 pg/mL), procalcitonin (PCT) at 0.07 ng/mL (reference range 0.00–0.50 ng/mL), prolonged prothrombin time of 13.2 seconds (reference range 9.4–12.5 s), low fibrinogen at 1.37 g/L (reference range 2.38–4.98 g/L), and markedly elevated D-dimer at 8588 μ g/L (reference range <500 μ g/L), indicating ongoing inflammation and active bleeding. Abdominal contrast-enhanced CT showed partial absorption of the previous hemoperitoneum but persistent aneurysmal dilations in the mesenteric and splenic arteries.

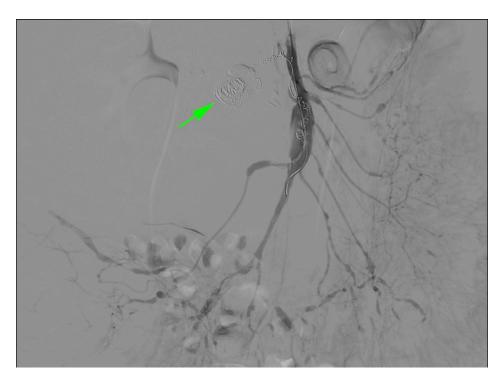


Fig. 4. Endovascular embolization of the middle colic artery. The arrow indicates the site of complete embolization.

Clinically, the patient experienced abdominal pain and had a history of upper gastrointestinal surgery. Emergent endoscopy on 27 February 2024 confirmed active bleeding at the distal esophagus, which was controlled with a titanium clip hemostasis. A subsequent endoscopy on 8 March 2024 revealed recurrent bleeding, which was again treated with clipping. No further bleeding occurred thereafter. The bleeding was attributed to a combination of glucocorticoid-induced gastrointestinal mucosal injury and vascular fragility secondary to disease activity. Methylprednisolone was temporarily discontinued; after supportive management with broad-spectrum antibiotics (piperacillin), proton pump inhibitors, and fibrinogen replacement, corticosteroid therapy was cautiously resumed at 40 mg/d. Biological disease-modifying antirheumatic drugs (bDMARDs) were not administered because of the patient's advanced age, high bleeding risk, and potential for infectious complications.

This comprehensive treatment approach successfully stabilized the patient without additional bleeding episodes. Post-treatment imaging follow-up demonstrated complete resolution of the retroperitoneal hematoma and disappearance of surrounding inflammatory changes (Fig. 5), confirming the effectiveness of combined interventional and immunosuppressive therapy. The condition of the patient gradually improved, and he was discharged in stable condition on 19 March 2024, at 11:49.

Prognosis

The patient underwent multiple interventional procedures, including arterial embolization and metal stent implantation. Post-treatment DSA and CTA revealed restored mesenteric blood flow, partial regression in some aneurysms, and no fur-



Fig. 5. Post-treatment abdominal CT (cross-sectional view). The arrow identifies the area that previously showed a retroperitoneal hematoma and surrounding inflammation, which follow-up imaging confirmed had been completely absorbed after treatment.

ther extension of the dissection, indicating significant therapeutic benefit. Following adjustment of immunosuppressive therapy, the clinical symptoms of the patient gradually improved, inflammatory markers declined significantly, and anemia showed notable recovery.

At the 3-month postoperative follow-up, contrast-enhanced CT revealed postoperative changes of the superior mesenteric artery dissection and aneurysms, with
mild distal branches dilatation but no evidence of new aneurysm formation or dissection progression (Fig. 6). These findings suggested relative vascular stability
after intervention. Laboratory evaluation at the same visit (Table 3) showed recovery of hemoglobin, normalization of coagulation indices, and stable renal and
hepatic function, consistent with clinical remission. During outpatient follow-up
at 6 months and 1 year, the patient reported no recurrence of abdominal pain or
bleeding episodes. However, no additional CT or laboratory examinations were
performed after the 3-month follow-up. Therefore, although short-term outcomes
appeared favorable and clinical status remained stable, the lack of long-term imaging and laboratory data limited assessment of vascular remodeling and the durability
of therapeutic efficacy.

Discussion

PAN typically involves the peripheral nerves, kidneys, and skin, and as the disease progresses, it may cause multiorgan injury (Pagnoux et al, 2005). The most common systemic symptoms include fatigue, weight loss, and fever (Hekali et al, 1991). However, cases of PAN presenting with acute abdominal pain accompanied by SMA dissection and multiple aneurysms are rarely documented. Several

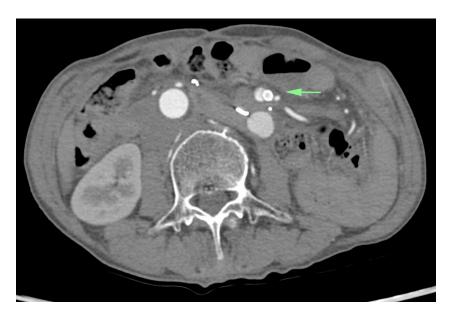


Fig. 6. Follow-up abdominal CT scan at 3 months postoperatively showing changes consistent with superior mesenteric artery dissection and aneurysms. Mild dilatation of distal SMA branches was observed (the arrow).

Table 3. Laboratory parameters at 3-month postoperative follow-up.

Parameter	Result	Reference range
Hemoglobin (g/L)	131	130–175
White blood cell count ($\times 10^9/L$)	5.11	3.5–9.5
Platelet count ($\times 10^9/L$)	116	125–350
Prothrombin time (s)	11.3	9.4–12.5
INR	0.98	0.85 - 1.15
Fibrinogen (g/L)	2.83	2.38-4.98
Serum creatinine (µmol/L)	45	40–135
ALT (U/L)	26.6	9–50
AST (U/L)	55.6	15–40

ALT, alanine aminotransferase; AST, aspartate aminotransferase; INR, international normalized ratio.

reports describe acute abdominal pain as an initial symptom of PAN. Hiraike et al (2013) reported a 73-year-old woman who presented with acute abdominal pain, intestinal wall thickening, and renal dysfunction, which was initially misdiagnosed as an acute abdominal condition. Despite treatment, her symptoms persisted, and PAN was eventually diagnosed after surgical resection of jejunal necrosis. Unfortunately, despite surgery and corticosteroid therapy, the patient died. Similarly, Gomes et al (2013) reported two cases of PAN presenting with acute abdominal colic, in which exploratory laparotomy revealed intestinal perforation and necrosis. Both patients ultimately died despite intensive therapy. In contrast, Asti et al (2015) described a 57-year-old male patient with PAN who presented with acute abdominal pain and improved after early corticosteroid therapy. The present case demonstrates successful disease control through early interventional management

with immunosuppressive strategies, resulting in a treatment outcome distinct from comparable reports in the literature and warrants further investigation.

The pathological mechanism of PAN primarily involves necrotizing inflammation of small- and medium-sized arteries, most often at arterial bifurcations. Disruption of the elastic lamina within the vessel wall leads to elevated proximal vessel pressure and dilation at stenotic sites, thereby promoting the formation of aneurysms (Ewald et al, 1987). The patient in this report was a middle-aged to elderly male. According to published evidence, risk factors such as infections, advanced age, and male gender are considered predisposing factors for PAN and are closely associated with both disease onset and prognosis (Wolff et al, 2023).

Aneurysm formation is a common manifestation of PAN, with reports of renal artery aneurysms (Ma et al, 2024; Tsukui and Kono, 2021), hepatic artery aneurysms (Li and Lao, 2021; Vera Cruz et al, 2024), and coronary artery aneurysms (Boppana et al, 2021; Huang et al, 2024). In contrast, SMA aneurysms in PAN are rarely reported. A 15-year-old male with PAN complicated by both hepatic and SMA aneurysms ultimately died of septicemia despite aggressive therapy (Kendirli et al, 2006). Conversely, a 4-month-old infant with PAN and SMA aneurysm achieved complete recovery following combined immunosuppressive and surgical intervention (Shimizu et al, 2019). These cases highlight that although SMA aneurysms are vascular lesions, the gastrointestinal complications they cause are of significant concern, as severe outcomes may be fatal.

The management of PAN requires multidisciplinary collaboration and continuous postoperative monitoring. Presence of aneurysms and rupture remain major therapeutic challenges, especially in elderly patients, in whom disease manifestations are often more complex. For instance, a 78-year-old male with PAN and multiple aneurysms in the pancreatic region initially improved with corticosteroid therapy but subsequently died due to acute pancreatitis complicated by septic shock and disseminated intravascular coagulation (DIC) (Takamatsu et al, 2022). In the present case, the patient exhibited significant SMA dissection and aneurysmal dilation with associated bleeding, indicating acute disease progression and increased arterial wall fragility. At admission, severe abdominal pain and retroperitoneal hemorrhage placed the patient in critical condition. Initial assessment confirmed SMA dissection with aneurysmal expansion and local bleeding. Given the severity, DSA was promptly performed to localize the lesion, followed by coil embolization and stent placement, which stabilized mesenteric blood flow and prevented further deterioration.

In recent years, non-surgical interventions for managing PAN have gained increasing attention, especially pharmacological therapies with corticosteroids, cyclophosphamide, and other immunosuppressive agents. These therapies have demonstrated efficacy in reducing aneurysm size and improving patient overall survival (Castelhano et al, 2021; Chung et al, 2021). Corticosteroids, particularly methylprednisolone, remain potent first-line drugs frequently employed to control rapidly progressive and severe forms of PAN (Castelhano et al, 2021). In the present case, given the critical condition of the patient at admission and recurrent life-threatening

hemorrhages, initial management prioritized endovascular intervention to control active bleeding.

Endovascular embolization is a well-established and effective technique for aneurysm management. For example, Kramann et al (1995) reported an 85% success rate using this technique. Additionally, embolization of ruptured aneurysms has been consistently shown to provide effective hemostasis and prevent further clinical deterioration (Park and Park, 2021; Takahashi et al, 2012). Nevertheless, Oomura et al (2006) described a PAN patient who experienced fatal aneurysm rupture despite intravenous corticosteroid therapy, underscoring the limitations of pharmacological treatment alone in acute hemorrhagic settings. These findings suggest that for PAN patients presenting with life-threatening hemorrhages, a phased therapeutic approach of "interventional hemostasis first, followed by gradual immunosuppressive therapy" may be optimal. In this approach, initial endovascular embolization achieves immediate hemostasis, stabilizes vital signs, and reduces shortterm mortality associated with bleeding complications. Subsequently, immunosuppressive therapy with corticosteroids and related agents is gradually introduced to suppress vascular inflammation, promote vascular healing, and prevent additional aneurysm formation, thus improving long-term survival and prognosis. In terms of nursing care, close monitoring of the patient's neurological status, vital signs, and abdominal findings was performed. Attention was given to detecting signs of hematemesis or melena, assessing wound and puncture site healing, and recording 24-hour urine output. Alongside this, abdominal drain care, basic nursing, and daily living care were provided, and any changes in the patient's condition were promptly assessed and reported for appropriate management. The efficacy and adverse effects of medications were carefully observed, while dynamic changes in imaging and laboratory results were continuously monitored. Anticipatory surveillance and management of potential complications were implemented, alongside psychological support and humanistic care, to ensure the effectiveness of comprehensive treatment.

This case adopted a phased treatment strategy of "interventional hemostasis and surgery in the acute phase, followed by gradual corticosteroid-based immunosuppressive therapy once the condition stabilizes". The advantages of this approach are as follows: For PAN patients presenting with acute abdominal pain and massive hemorrhage, the immediate priority is rapid hemostasis and stabilization of hemodynamics through interventional or surgical procedures. These measures create the conditions necessary for subsequent systemic immunotherapy, thereby effectively reducing the early risk of mortality. Following stabilization, timely initiation of corticosteroid therapy serves to suppress vascular inflammation, promote vascular repair, limit the formation of new lesions, and improve long-term prognosis. This strategy highlights the benefits of multidisciplinary collaboration and individualized treatment planning.

However, this approach has certain limitations. On one hand, repeated interventional and surgical procedures may heighten the risks of procedural trauma and secondary infection. On the other hand, immunosuppressive therapy introduces potential complications such as re-bleeding and opportunistic infection, which re-

quire careful adjustment of treatment timing and dosage. This is particularly critical in elderly patients or those with multiorgan dysfunction, where treatment tolerance must be thoroughly evaluated. Additionally, due to the critical condition of the patient, tissue biopsy was not performed, and the diagnosis relied primarily on imaging (DSA, CTA) and clinical presentations. This limitation may have reduced diagnostic accuracy and delayed differentiation from other vasculitic syndromes. Furthermore, this study lacks long-term follow-up data and records of hormone tapering, which limits the assessment of vascular remodeling, disease recurrence, and long-term therapeutic efficacy. Future investigations should include histopathological evidence to strengthen diagnostic certainty and to refine treatment decisions, especially the precision of immunosuppressive therapy.

This study also has limitations in follow-up and data collection. Disease activity markers, such as IL-6 and CRP, were not assessed during the 3-month and 1-year follow-up visits, and long-term records of corticosteroid tapering were lacking. Consequently, a comprehensive evaluation of vascular remodeling, disease recurrence, and long-term therapeutic efficacy was not possible. These limitations highlight the need for future studies to incorporate histopathological evidence, dynamic monitoring of inflammatory markers, and extended follow-up to enhance diagnostic accuracy and optimize immunosuppressive treatment strategies.

Conclusion

This case highlights the atypical presentation of PAN, with acute abdominal pain as the patient's initial symptom, underscoring the need to consider PAN in the differential diagnosis of acute abdominal pain. Additionally, mesenteric artery aneurysms, as a rare complication of PAN, emphasize the critical need for early identification and timely intervention of vascular lesions to optimize patient prognosis.

Learning Points

- This rare PAN case presented with acute abdominal pain and superior mesenteric artery (SMA) dissection with multiple aneurysms, a scarcely reported manifestation.
- It contributes to the literature by documenting successful management using initial endovascular embolization followed by methylprednisolone therapy.
- The case underscores the need to consider PAN in acute abdominal pain when vascular abnormalities are observed on imaging.
- Its clinical significance lies in demonstrating the effectiveness of multidisciplinary, staged interventions for complex PAN, thereby improving patient outcomes.
- Reporting SMA involvement in PAN adds valuable clinical evidence, highlighting the role of early imaging and intervention for improving prognosis.

Availability of Data and Materials

The data and materials in the current study are available from the corresponding author on reasonable request.

Author Contributions

YLN, XZ, and LL contributed to the study design. XZ conducted the literature search. YLN collected the clinical data and drafted the manuscript. LL revised the manuscript for important intellectual content. All authors contributed to revising the manuscript, read and approved the final version, and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This case report is in accordance with the ethical standards in the Declaration of Helsinki. The study was approved by the ethics committee of Jinhua Municipal Central Hospital (GA2025-06). Informed consent for publication of this case report and any accompanying images was obtained from the patient.

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Not applicable.

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Conflict of Interest

The authors declare no conflict of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://www.magonlinelibrary.com/doi/suppl/10.12968/hmed.202 5.0562.

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