

# Segmental Arterial Mediolytic in a Patient with Respiratory Syncytial Virus

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## Abstract

We report a case of segmental arterial mediolytic (SAM) in a 45-year-old man with respiratory syncytial virus (RSV). He presented with acute abdominal pain following a week of forceful coughing. Blood tests showed elevated inflammatory markers. CT angiography revealed arterial thrombi and a splenic infarct. Hypercoagulable studies were unrevealing and inconsistent with vasculitis or thrombophilia. The patient was diagnosed with SAM and started on apixaban, and a repeat CT showed improvement of arterial flow after one month. This case highlights a potential link between viral cough and the development of SAM and highlights the use of anticoagulation in treatment.

## Keywords

Segmental Arterial Mediolytic, Arterial Thrombosis, Anticoagulation, Dissection, RSV Complications

## 1. Background

Segmental arterial mediolytic (SAM) is a rare, idiopathic, non-atherosclerotic, and non-inflammatory vascular disease primarily affecting medium-sized abdominal arteries [1] [2]. SAM was identified in 1976 by Salvin and Gonzalez-Vitale, who described its histological features, including vacuolization and lysis of the arterial media. This lysis of arterial media can result in the formation of dissecting aneurysms and pseudoaneurysms [3]. SAM can affect patients of any age and gender, though it is most commonly observed in genetically male individuals in their fifth and sixth decades [1]. Radiological findings may resemble other conditions, including collagen vascular diseases, fibromuscular dysplasia, or inflammatory vasculitis; however, the key distinguishing features of SAM are the presence of aneurysms, stenoses, dissections, and occlusions in the splanchnic arteries, particularly

affecting the celiac, mesenteric, or renal arteries. Less commonly, ischemia and infarction occur with SAM [2]. Inflammatory markers, genetic tests for collagen vascular disorders, and hypercoagulability studies often return negative in the workup of SAM [1]. SAM is differentiated from vasculitis on pathology by the absence of true inflammation and the presence of mediolysis [4].

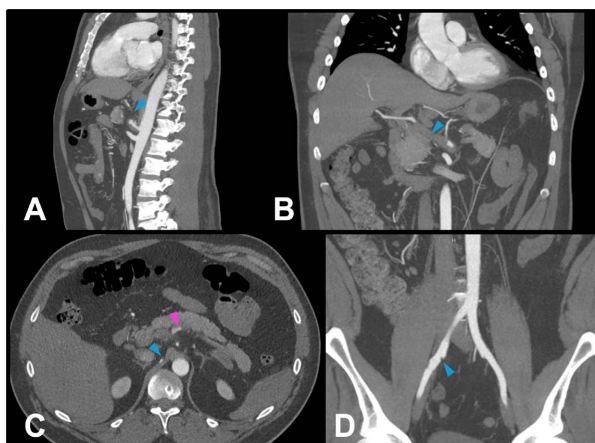
## 2. Objective

To present a rare case of SAM in a patient with RSV, highlighting the potential association between viral coughing and the development of SAM.

## 3. Case Presentation

A 45-year-old man presented to the emergency department with one day of acute, diffuse abdominal pain. Medical history included hyperlipidemia on atorvastatin. He reported nausea, without vomiting, which developed after a week of forceful coughing. He denied fever, chills, chest pain, hematuria, hematochezia, and epistaxis. The patient denied tobacco use. There was no personal or family history of autoimmune, bleeding, or clotting disorders. The physical exam was unremarkable, apart from a nonproductive cough. Although the patient reported abdominal pain, his abdomen was soft and nontender to palpation. On the day of admission, he received hydromorphone and underwent further workup for infection, dissection, acute coronary syndrome, etc. The CXR showed clear lungs. The infectious workup was positive for RSV.

### 3.1. Initial Findings



**Figure 1.** Contrast-enhanced computed tomography angiogram of the abdomen, chest, and pelvis, with blue arrows showing acute thrombosis in the celiac artery (A), common hepatic artery (B), and splenic artery (C); a short dissection of the right external iliac artery (D); and a splenic infarct (pink arrow) (C). An image of acute thrombosis in the proximal gastroduodenal artery is not included.

Abdomen and pelvis computed tomography (CT) revealed multiple acute thrombi in the distal celiac, common hepatic, proximal gastroduodenal, and splenic arteries,

along with a splenic infarct. Venous Doppler ultrasound of the upper and lower extremities showed no deep venous thrombosis. A contrast-enhanced CT angiogram of the chest, abdomen, and pelvis redemonstrated these arterial thrombi, the splenic infarct, and also revealed a short-segment dissection of the right external iliac artery (**Figure 1**). Vascular surgery was consulted, and no immediate surgical intervention was recommended. He was started on heparin.

### 3.2. Laboratory and Radiographic Discoveries

CBC, BMP, and lipid studies were unremarkable. Inflammatory markers were mildly elevated, with C-reactive protein (CRP) of 3.4 mg/dL and erythrocyte sedimentation rate (ESR) of 34 mm/hr. Broad hypercoagulability and vasculitis studies, outlined in **Table 1**, were performed and yielded unremarkable results. Hematology and rheumatology were consulted in the workup and management of this condition; both endorsed a strong suspicion of SAM. Four days after admission, the patient was started on apixaban at the standard dose for three months and discharged home. Apixaban was chosen due to ease of dosing. Repeat CT after one month showed improved flow throughout the celiac artery with contrast newly extending into the proximal splenic and common hepatic arteries, along with persistent distal celiac artery dissection and occlusion of the midsplenic artery. The non-flow-limiting focal dissection of the proximal right external iliac artery remained unchanged.

**Table 1.** Hypercoagulability and vasculitis studies.

| Test                                | Result                     |
|-------------------------------------|----------------------------|
| Cardiolipin antibodies, IgM and IgG | Not detected               |
| Factor 2 mutation                   | No mutation detected       |
| Factor V Leiden                     | No mutation detected       |
| Homocysteine                        | Wnl                        |
| Antithrombin 3 activity             | Wnl                        |
| Protein C activity                  | Wnl                        |
| DRVVT screen                        | Wnl                        |
| PTTLA ratio                         | Wnl                        |
| B2 glycoprotein                     | Wnl                        |
| JAK2 V617F mutation                 | Not detected               |
| ANA with reflex                     | Negative                   |
| ANCA IgG antibodies                 | Titer <1:20, not detected. |
| C3 and C4 complements               | Wnl                        |
| DNA double-standard antibody IgG    | Negative                   |
| Hepatitis panel with reflex         | Negative                   |
| ENA antibodies                      | Negative                   |
| Paroxysmal nocturnal hemoglobinuria | Not detected               |
| Alpha-fetoprotein tumor marker      | Wnl                        |

WNL = within normal limits.

## 4. Discussion

We present this case of a 45-year-old male, with a history of hyperlipidemia, di-

agnosed with presumed segmental arterial mediolysis in the setting of forceful, viral-associated coughing.

This presentation differs from previously reported cases of SAM and raises an interesting potential correlation between upper respiratory viral illness and the development of SAM, a noninflammatory arteriopathy more often associated with chronic arterial vasospasm [5]. His abdominal pain and nausea were likely related to the splenic infarct demonstrated on CT imaging, given rapid resolution and absence of further episodes. While segmental arterial mediolysis is typically associated with the formation of dissecting aneurysms, there were no aneurysms observed on this patient's CTA, further distinguishing his case.

A systematic review by Skeik *et al* found that SAM most commonly affects men in their sixth decade [6]. The main risk factors and conditions associated with SAM include hypertension, tobacco use, and hyperlipidemia. The most common presenting symptoms include abdominal pain and intra-abdominal bleeding. While our patient did not have hypertension or use tobacco, he had a history of hyperlipidemia treated with atorvastatin, with an LDL level of 109 mg/dL. No additional risk factors were identified, although subclinical vascular risks may have contributed. The most commonly affected vessels in SAM include the superior mesenteric, hepatic, celiac, renal, and splenic arteries, which often show aneurysms, dissections, or ruptures [5]. Our patient had thrombosis of the celiac, common hepatic, proximal gastroduodenal, and splenic arteries, along with dissection of the right external iliac artery and splenic infarct.

Multiple consulting physicians suspected that this patient's forceful coughing associated with RSV infection contributed to the condition's onset. A literature review reveals no known cases of SAM directly linked to coughing. However, there are many reports of coughing associated with arterial abnormalities. Studies have noted spontaneous rupture of the intercostal artery following severe coughing; vertebral artery dissection due to neck flexion during coughing; and internal artery dissection linked to coughing [7]-[9]. Our patient presented with a short-segment dissection of the right external iliac artery in the setting of forceful coughing, leading to thrombosis in multiple arterial vessels. One such possible connection between coughing and SAM could be barotrauma-induced arterial injury, although further research is needed [10] [11].

## 5. Summary

The diagnosis of SAM is based on clinical presentation, imaging findings, and the exclusion of other conditions. Inflammatory markers, genetic tests for collagen and vascular disorders, and hypercoagulability studies are typically negative in SAM, as was the case in our patient. His hypercoagulability and vasculitis tests were negative.

There is no gold standard treatment for SAM. Treatment depends on the severity of presentation. Optimization of risk factors is always recommended. Acute cases with aneurysmal rupture may require urgent vascular surgical or endovas-

cular intervention. Surgical options may include coil embolization or arterial repair. In the absence of such complications, SAM is often self-limiting and can be managed conservatively with appropriate blood pressure control and consideration of anticoagulation. The long-term prognosis of SAM is not well established, and follow-up imaging is advised. Anti-inflammatory agents and immunosuppressants are not effective for treating SAM. A recent study by Peng *et al.* illustrates that the most common long-term treatments for SAM are aspirin, antihypertensives in the form of beta-blocker therapy, and anticoagulation [12]. In our patient, hyperlipidemia was well managed with atorvastatin, and the blood pressure was normotensive. We elected to start a Factor Xa inhibitor given extensive arterial clotting. Our patient was started on apixaban and showed no signs of recurrence or disease progression at a one-month follow-up, confirmed by CT imaging. Apixaban was chosen over heparin or other antiplatelet therapy due to ease of dosing, lack of bridging and necessary lab monitoring, and prior studies demonstrating non-inferiority of apixaban versus heparin-based regimens for venous thromboembolism [13]. Unfortunately, data directly comparing apixaban to heparin-based regimens for treatment of arterial clots are lacking and present an area for future study.

### Limitations

Limitations of this case report include the lack of histopathologic confirmation. However, several guidelines accept image-based diagnoses when biopsy is impractical and/or unnecessary based on risk-benefit analysis, especially in hepatocellular carcinoma and other solid organ tumors [14].

## 6. Conclusions

In conclusion, we present an unusual case of SAM in a 45-year-old male with RSV. This case highlights the importance of considering SAM in patients with unexplained abdominal pain or arterial thrombosis, and documents a potential complication of coughing related to viral infection.

We also highlight the effect of medical management in SAM, rather than surgical management, which is supported by recent studies when patients present with SAM without complications of mesenteric ischemia or pseudoaneurysm rupture [15].

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### Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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