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# Acute on a Quiet Background: CT Angiographic Diagnosis of Spontaneous Isolated Superior Mesenteric Artery Dissection with Proximal False-Lumen Thrombosis

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**Abstract:** ***Spontaneous isolated dissection of the superior mesenteric artery (SISMAD) is the most frequently encountered visceral artery dissection, yet it remains an uncommon and often unsuspected cause of abdominal pain. Where it once surfaced mostly at autopsy, cross-sectional imaging now finds it in living, symptomatic patients, and the reported frequency has climbed accordingly[2,11]. We describe a 53-year-old man who presented with two months of insidious, food-aggravated periumbilical pain accompanied by anorexia and recent weight loss. Contrast-enhanced CT of the abdomen demonstrated an intimal flap arising 9.5 mm distal to the SMA origin and extending 7.5 cm caudally, with an eccentric, partially thrombosed false lumen of larger calibre compressing the true lumen by 70–80% to a residual diameter of roughly 3.8 mm. The dissection spared the ileocolic branches, although the mid-jejunal branches showed luminal irregularity and narrowing suggestive of hypoperfusion. No aortic dissection, frank bowel infarction, or aneurysmal rupture was present. The constellation corresponds to a Sakamoto type III lesion. Given haemodynamic stability and absence of transmural ischaemia, conservative management with blood-pressure control and antithrombotic therapy was pursued, with digital subtraction angiography and serial lactate held in reserve. This report sets out the CT angiographic signatures that secure the diagnosis and situates the imaging-based classification schemes that steer treatment.***

**Keywords:** Superior mesenteric artery dissection; SISMAD; CT angiography; intimal flap; false lumen thrombosis; Sakamoto classification; mesenteric ischaemia; visceral artery dissection

## 1. Introduction

Arterial dissection is overwhelmingly a disease of the aorta, but it spares no vessel. When it strikes a visceral artery in isolation- without any aortic component- the superior mesenteric artery (SMA) is the one most often hit, ranking behind only the renal, coronary, and intracranial vessels among isolated dissections overall[10,12]. The first description dates to Bauersfeld in 1947[1], and for decades the entity was a pathological curiosity: an autopsy series placed its frequency near 0.06%[6,10]. That figure now reads as a floor rather than a true prevalence. As multidetector CT angiography became the reflex first test for the acute or unexplained abdomen, lesions that would once have gone unrecorded began to declare themselves, and SISMAD shifted from rarity to recognised differential[2,11].

Two anatomical facts explain why the SMA is vulnerable. The artery transitions from a fixed retropancreatic segment to a mobile portion that pivots with the mesentery, and the shearing forces concentrated at this junction- typically 1.5 to 3 cm beyond the ostium, along the convex anterior wall — are thought to seed the intimal tear[17,7]. Proposed predispositions include atherosclerosis, cystic medial necrosis, fibromuscular dysplasia, connective-tissue disorders, segmental arterial mediolysis, and hypertension, though no single factor is consistently implicated and most patients are otherwise well[13,14]. A large follow-up cohort argued that hemodynamic stress at the curvature, rather than hypertension or hereditary arteriopathy, is the dominant mechanism[7].

Clinically the disease is a chameleon. Presentations span the asymptomatic incidental finding, the dull postprandial ache of chronic mesenteric angina, and the catastrophe of acute bowel infarction or aneurysmal rupture[2,14]. Middle-aged men predominate, generally in the fifth to seventh decades[18]. Because the symptom that brings these patients to attention — abdominal pain — is among the least specific in medicine, the diagnosis hinges on imaging, and on a radiologist willing to scrutinise the SMA when the more common explanations have been excluded[16,12]. The case below illustrates both the imaging hallmarks and the reasoning that converts an unexpected vascular finding into a defensible management plan.

## 2. Case Report

A 53-year-old man presented to Dr. D. Y. Patil Hospital, Navi Mumbai, late in the evening with periumbilical abdominal pain. The pain had begun roughly two months earlier and had been diffuse, non-radiating, insidious in onset, and progressive. It worsened reliably after meals and eased with medication. Over the same interval he had lost his appetite, and he reported

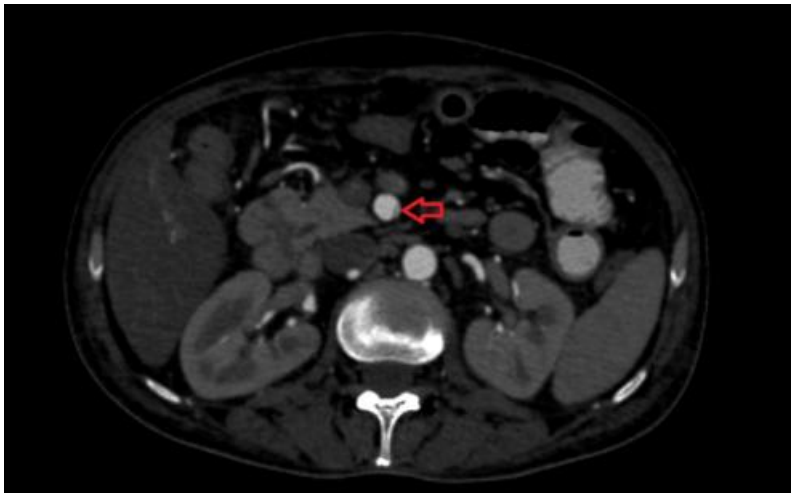
unintentional weight loss across the preceding fortnight. He was haemodynamically stable at presentation.

The postprandial character of the pain, set against weight loss and anorexia, raised the possibility of mesenteric vascular compromise and prompted contrast-enhanced CT of the abdomen with arterial-phase acquisition and multiplanar and maximum-intensity-projection (MIP) reformations, supplemented by three-dimensional volume-rendered reconstructions of the mesenteric vasculature.

## 3. Imaging Findings

***Primary dissection and false lumen***

Arterial-phase imaging resolved a curvilinear intimal flap within the proximal SMA, beginning approximately 9.5 mm distal to the vessel origin and extending caudally over a 7.5 cm segment. The flap separated a true lumen from a false lumen, the latter of larger calibre and lying eccentrically along the vessel. At the proximal entry the false lumen contained an eccentric hypodense component consistent with partial mural thrombosis. This thrombosed false lumen compressed the true lumen, producing 70–80% luminal narrowing with a residual true-lumen diameter of roughly 3.8 mm. Crucially, the dissection did not extend into the ileocolic branches, and there was no associated aortic dissection



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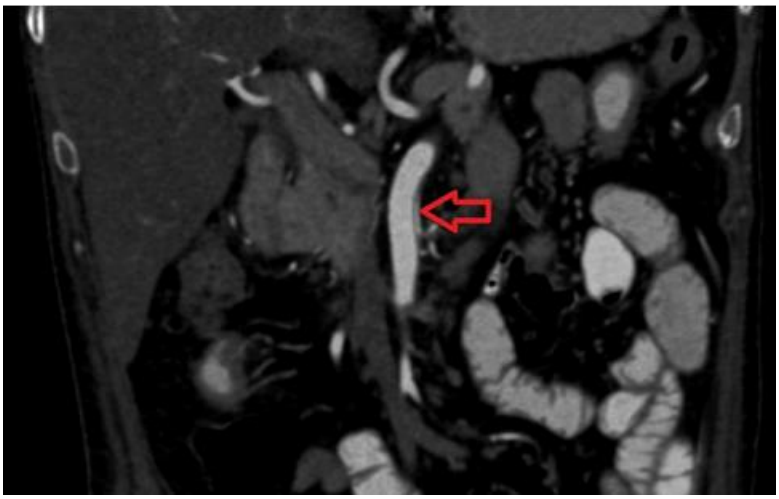

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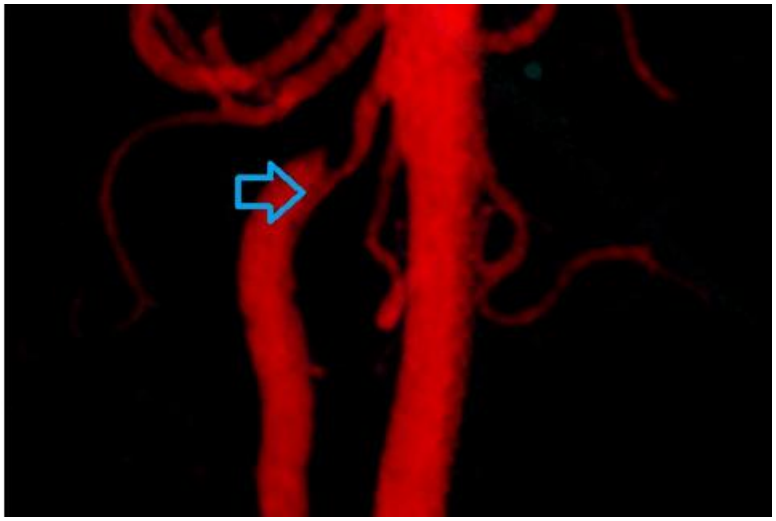

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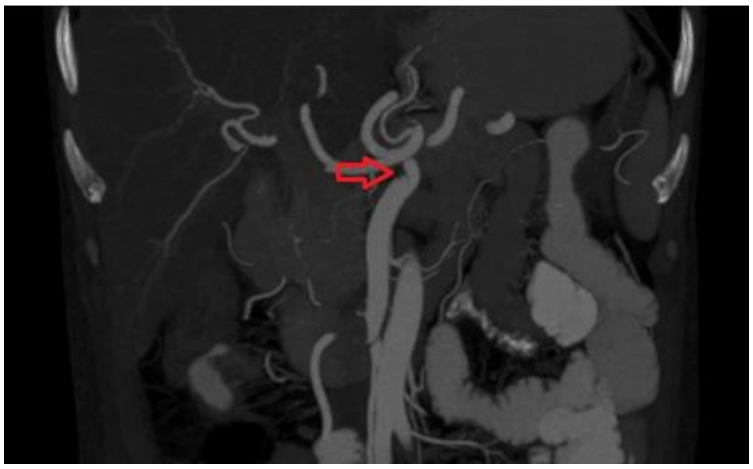
**Figure 1:** Intimal flap and false lumen of the proximal SMA

**(A)** Axial contrast-enhanced CT shows the proximal SMA with an intraluminal intimal flap separating true and false lumens (red arrow). **(B)** Coronal reformat demonstrates the longitudinal extent of the flap and the eccentric, larger-calibre false lumen (red arrow). **(C)** Sagittal three-dimensional volume-rendered reconstruction depicts the dissection entry along the proximal SMA (blue arrow), confirming origin sparing of the aorta. The flap commences ~9.5 mm distal to the SMA origin and extends ~7.5 cm caudally.

***Entry site, true-lumen compromise, and branch involvement***

The proximal entry tear and the degree of true-lumen stenosis were best appreciated on the coronal MIP and the oblique volume-rendered reconstruction. The MIP confirmed the focal narrowing of the contrast-opacified true lumen at the level of the thrombosed false lumen, while the three-dimensional reconstruction displayed the relationship of the dissection to the SMA origin and main trunk in a single projection- the kind of integrated overview that catheter angiography supplies only invasively[11]. The mid-jejunal branches showed luminal irregularity with narrowing, a pattern in keeping with hypoperfusion or evolving thrombo-occlusive change rather than established infarction.



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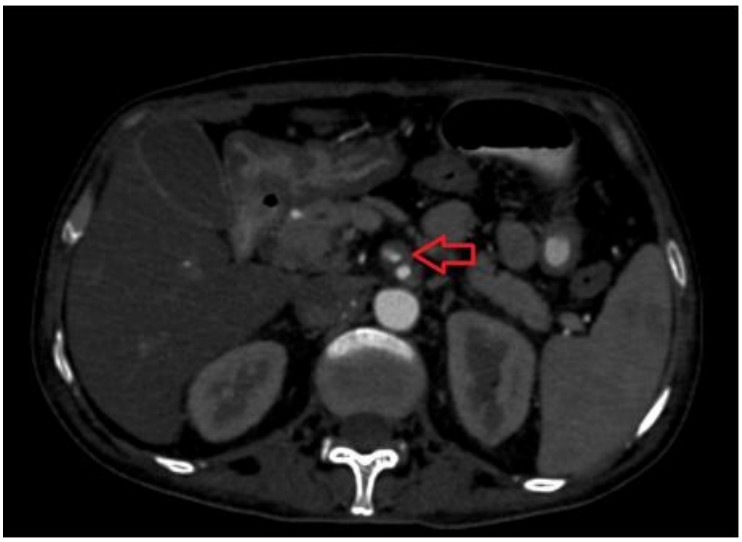

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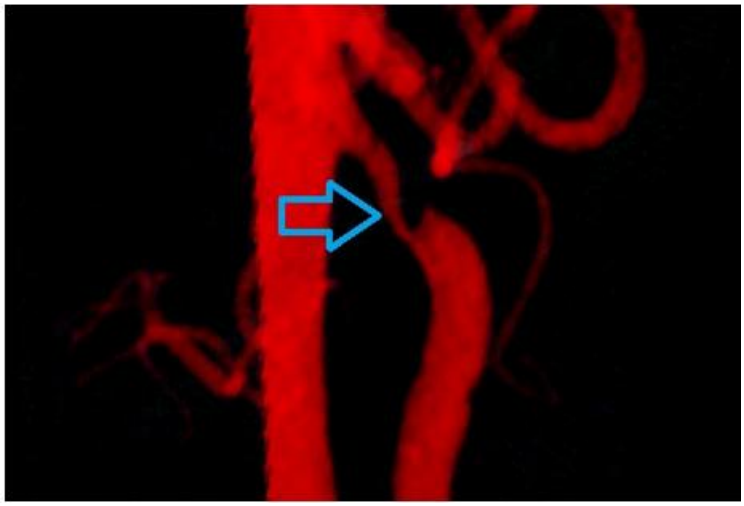

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**Figure 2:** Entry tear, true-lumen stenosis, and proximal false-lumen thrombosis
**(A)** Coronal MIP image localises the proximal entry and the eccentric thrombosed false lumen narrowing the true lumen to ~3.8 mm (red arrow). **(B)** Axial CE-CT at the entry level shows the eccentric hypodense partial thrombus within the false lumen and the resultant 70–80% true-lumen compromise (red arrow). **(C)** Coronal-oblique three-dimensional reconstruction profiles the dissection along the proximal SMA trunk (blue arrow), with the true lumen narrowed but patent distally.

***Distal mesenteric branches and ancillary findings***

Coronal MIP imaging of the mesenteric branches showed the jejunal branch with luminal irregularities and narrowing, again favouring hypoperfusion or thrombo-occlusive disease over completed ischaemia; no pneumatosis, mesenteric fat stranding diagnostic of infarction, or free intraperitoneal gas was identified. An incidental, unrelated grade III diffuse hepatic steatosis was noted. No CT sign of bowel-wall non-enhancement, mural thickening with target sign, or portomesenteric venous gas- the imaging markers that would mandate urgent intervention- was present[10,12].

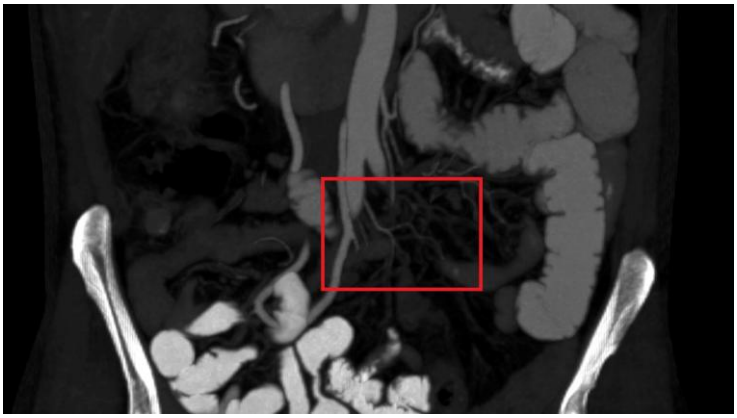

**Figure 3:** Distal mesenteric branch involvement

Coronal MIP image (boxed region) demonstrates luminal irregularity and narrowing of the mid-jejunal branches of the SMA, consistent with hypoperfusion or thrombo-occlusive change. No definite CT sign of established bowel infarction was present at the time of imaging.

***Imaging diagnosis***

The findings were interpreted as **spontaneous isolated superior mesenteric artery dissection with proximal partial thrombosis of the false lumen**, accompanied by long-segment mural thickening of the mid-jejunal loops. In view of the partially thrombosed false lumen with true-lumen compromise but without a patent re-entry, the lesion was categorised as a Sakamoto type III dissection. Because the bowel changes raised the question of subclinical ischaemia, digital subtraction angiography and serial serum lactate were advised to exclude evolving mesenteric ischaemia.

## 4. Discussion

***Why the SMA, and why proximally***

The location of this dissection is not incidental. Across reported series the entry tear clusters along the convex anterior wall of the SMA, a centimetre or two beyond the ostium, precisely where the fixed retropancreatic segment yields to the mobile mesenteric portion[13,7]. The mechanical argument is straightforward: repetitive flexion at a tethered-to-mobile junction concentrates wall stress, the intima tears, and blood tracks into the media. In the present case the flap began 9.5 mm distal to the origin- at the lower bound of the reported 1.5 to 3 cm range, but firmly within the vulnerable zone and sparing the ostium, which is the defining feature separating an isolated SMA dissection from aortic extension[2]. Histology, when available, has occasionally revealed segmental arterial mediolysis, suggesting that an intrinsic medial weakness can compound the haemodynamic insult[13].

***The CT angiographic signatures***

CT angiography carries the diagnosis. The cardinal sign is the intimal flap dividing true and false lumens, as seen here on axial and coronal images; the entry tear, the longitudinal extent, the degree of true-lumen stenosis, and any branch involvement follow from careful multiplanar and MIP review[11,2]. A thrombosed false lumen appears as crescentic low attenuation that fails to enhance- distinguishing it from the contrast-filled true lumen- and may be indistinguishable from an intramural haematoma when no intimal tear is resolved[10,17]. Ancillary features worth recording include aneurysmal dilatation (an outer diameter at least 1.5 times the adjacent normal vessel), periarterial fat stranding, and, most consequentially, signs of bowel ischaemia such as reduced or absent mural enhancement, bowel-wall thickening, mesenteric oedema, pneumatosis, or portomesenteric gas[10]. Three-dimensional reconstructions add little diagnostic information beyond the source images but communicate the anatomy to surgeons and interventionalists with a clarity that flat images rarely match[11]. In this patient the absence of any infarction sign, set beside jejunal branch narrowing, defined the central clinical question: hypoperfusion that will settle, or ischaemia that will progress.

***Classification and its bearing on treatment***

Imaging classification is not an academic exercise here; it maps onto management. Sakamoto's scheme, built on the appearance of the false lumen, remains the reference point: type I, a patent false lumen with both entry and re-entry; type II, a cul-de-sac false lumen without re-entry; type III, a thrombosed false lumen with an ulcer-like projection; and type IV, a completely thrombosed false lumen without such a projection[2]. The present lesion- a partially thrombosed false lumen compressing but not occluding the true lumen- sits at type III. Later authors refined the framework: Yun and colleagues added total SMA occlusion[3]; Zerbib and colleagues appended categories for dissecting aneurysm and for SMA thrombosis[4]; and Li and colleagues reorganised the types around true-lumen patency, which the earlier schemes neglected despite its prognostic weight[5,9]. The proliferation of systems reflects a genuine gap- no single classification commands consensus- but the shared lesson is consistent: the state of the true lumen and the presence of ischaemia, not the false lumen alone, drive decisions[6].

***Management rationale in this patient***

Contemporary practice, including the European Society for Vascular Surgery guidance, treats conservative management as first-line for the stable patient without bowel ischaemia or rupture[15]. The approach combines blood-pressure control, bowel rest, and antithrombotic therapy- antiplatelet agents or anticoagulation- with close clinical and imaging

surveillance[14,20]. Reported symptom resolution with this strategy is high, on the order of 90% or more, and the incidence of bowel ischaemia low[21]. The evidence for antithrombotics is observational rather than definitive, and one school of thought cautions that anticoagulation may delay protective false-lumen thrombosis[14,21]; the decision is therefore individualised. Endovascular stenting is reserved for persistent or recurrent symptoms, progressive true-lumen compromise, or enlarging dissecting aneurysm, and carries high technical success when the true lumen can be cannulated[8,20]. Open surgery- thrombectomy, intimectomy, aneurysmectomy, or bypass, with bowel resection where needed- is held for frank infarction, rupture, or failed endovascular rescue[5,19]. A dissection length of 50 mm or more has been flagged as a risk factor for conservative failure, a threshold this patient's 75 mm lesion exceeds, which justified the cautious posture of arranging digital subtraction angiography and lactate monitoring rather than discharging on reassurance alone[21]. The jejunal branch narrowing made that vigilance mandatory: subclinical ischaemia, if it declared itself, would convert a conservatively managed patient into a surgical one within hours.

## 5. Summary and Key Learning Points

- **Suspect it when the common causes fail.** SISMAD is the most frequent visceral artery dissection but a rare cause of abdominal pain; it deserves active consideration once the usual explanations for acute or postprandial pain have been excluded[16,12]
- **The intimal flap is the anchor sign.** Arterial-phase CT angiography with multiplanar and MIP reformations defines the entry tear, false-lumen status, true-lumen stenosis, and branch involvement; origin sparing distinguishes isolated SMA dissection from aortic extension[2,11]
- **Read the true lumen, not just the false one.** Classification by false-lumen morphology (Sakamoto and its modifications) orients the diagnosis, but true-lumen patency and ischaemia signs govern management[5,6]
- **Stable plus no ischaemia equals conservative.** Blood-pressure control, bowel rest, and antithrombotic therapy with surveillance resolve symptoms in most patients; intervention is reserved for persistent symptoms, progression, aneurysm, rupture, or infarction[15,21]
- **Branch narrowing changes the calculus.** Jejunal or distal branch hypoperfusion warrants serial lactate and a low threshold for angiography, since evolving ischaemia can outpace a reassuring initial scan; a long dissection segment (≥50 mm) further raises the risk of conservative failure[21,19]

## 6. Conclusion

This case captures spontaneous isolated SMA dissection in its most instructive form: a stable patient, a confident CT diagnosis, and a management decision that turned on the fine print of the imaging. The flap, the eccentric thrombosed false lumen, the 70–80% true-lumen compromise, and the spared ileocolic branches together placed the lesion as a Sakamoto type III and supported a conservative course. The jejunal branch narrowing kept that course honest, mandating angiographic and biochemical surveillance against the possibility of progression. CT angiography did more than detect the dissection- it characterised the false lumen, quantified the stenosis, surveyed the branches, and excluded infarction, delivering in one examination the information that dictates whether a patient is watched, stented, or operated upon[11,2]. For the practising radiologist the message is economical: when abdominal pain outlasts its usual explanations, look hard at the superior mesenteric artery, and let the true lumen and the bowel- not the flap alone- set the plan.

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