

Vicarious Excretion Of Iv Contrast Through The Small Bowel In A Ct Abdominal Angiogram Study

Rithani KR^{1*}, Shiyam Sundaran¹, I Venkatraman¹, G Murugan¹

¹Department of Radiodiagnosis, Sree Balaji Medical College and Hospital, Chromepet, Chennai - 600044, Tamil Nadu, India.

*Corresponds to: Email: rithani21@gmail.com

ABSTRACT:

A 76-year-old male presented with hematemesis, epistaxis, pedal edema, and generalized weakness for one day and also with a history of peptic ulcer disease, coronary artery disease, and chronic alcohol use presented with hematemesis, hypotension, and renal impairment. A CT abdominal angiogram was performed to evaluate for vascular pathology. Laboratory findings indicated renal impairment with elevated urea (104 mg/dL) and creatinine (1.38 mg/dL). A CT abdominal angiogram revealed a partially thrombosed fusiform abdominal aortic aneurysm in the infrarenal segment measuring 4.7 cm in length and 3.7 × 3.7 cm in diameter, without evidence of rupture. Incidentally, hyperdense material was observed within the small bowel loops, despite no prior oral contrast administration, suggestive of vicarious excretion of intravenously administered contrast. Additional findings included bilateral pleural effusions, emphysematous lung changes, mild splenic infarcts, pancreatic atrophy, and diffuse atherosclerotic calcifications of the thoracoabdominal aorta and its branches. The presence of intestinal contrast excretion in this patient likely reflected impaired renal clearance secondary to renal dysfunction and systemic hypoperfusion. Vicarious excretion of contrast through the bowel is a rare but recognized compensatory mechanism in cases of renal insufficiency, shock, or circulatory compromise. This case underscores the significance of identifying vicarious excretion as an indirect radiologic indicator of renal dysfunction and systemic vascular compromise in elderly patients with atherosclerotic disease.

KEYWORDS: Vicarious excretion, Intravenous contrast, small bowel, Abdominal aortic aneurysm, Renal dysfunction, CT angiogram.

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INTRODUCTION:

Intravenous iodinated contrast agents are primarily eliminated via renal excretion through glomerular filtration. In certain circumstances, however, alternative pathways may be utilized, a phenomenon referred to as vicarious contrast excretion (1). This process eliminates contrast material through non-renal routes such as the hepatobiliary system, gastrointestinal tract, salivary glands, or respiratory mucosa. Although most frequently observed in patients with impaired renal function or delayed urinary excretion, vicarious excretion may occasionally occur even in individuals with preserved renal performance (2).

Contrast excretion through the small intestine is uncommon among the extra-renal routes (3). When present, it can be easily mistaken for intraluminal contrast from prior oral administration or gastrointestinal bleeding, potentially leading to diagnostic confusion (4). The exact mechanism remains unclear but is hypothesized to involve alterations in vascular permeability, mucosal transport, or compensatory diversion of the contrast medium through intestinal capillaries secondary to renal dysfunction or circulatory disturbances (5,6).

This case report describes a rare instance of vicarious excretion of intravenously administered contrast into

the small bowel identified on a CT abdominal angiogram.

CASE PRESENTATION:

A 76-year-old male presented with complaints of hematemesis and epistaxis for one day, along with pedal edema and generalized weakness. The patient had a history of peptic ulcer disease and coronary artery disease for which he had been on regular medication for the past eight months. He was a chronic smoker and alcohol consumer. On clinical examination, the patient was pale and hypotensive, with a blood pressure of 90/60 mmHg and peripheral oxygen saturation (SpO₂) of 90%. Laboratory investigations revealed elevated serum creatinine (1.38 mg/dL) and urea (104 mg/dL) levels, suggestive of renal impairment.

Given the suspicion of an upper gastrointestinal bleed and possible vascular pathology, a CT abdominal angiogram was performed. Imaging demonstrated focal fusiform dilatation of the abdominal aorta in the infrarenal segment measuring approximately 4.7 cm in length and 3.7 × 3.7 cm in diameter, containing intramural thrombus—findings consistent with a partially thrombosed fusiform abdominal aortic aneurysm. There was no evidence of rupture or periaortic fat stranding. The celiac, mesenteric, and renal arteries appeared normal.

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An unexpected finding in the study was the presence of hyperdense content within the small bowel loops, without prior oral contrast administration. This was interpreted as a vicarious excretion of intravenously administered contrast. Associated findings included bilateral pleural effusions (left > right), emphysematous and fibroatelectatic lung changes, and mild splenic hypodense areas suggestive of splenic infarction. The pancreas appeared mildly atrophic, and diffuse atherosclerotic calcifications were seen in the thoracoabdominal aorta and its branches.

The overall impression was of a partially thrombosed abdominal aortic aneurysm with vicarious excretion of IV contrast through the small bowel, likely secondary to renal dysfunction. Clinical correlation and further evaluation for renal and systemic vascular status were recommended.



Figure 1 shows a Coronal section of a CT Abdominal angiogram showing aneurysmal dilatation in the infrarenal segment of the abdominal aorta. The contrast-enhanced CT angiogram of the abdomen and pelvis (coronal reconstruction) shows normal opacification of the abdominal aorta and its major branches, including the celiac axis, superior mesenteric artery, renal arteries, and iliac arteries, without evidence of stenosis, aneurysm, or dissection. The visceral branches demonstrate normal course and caliber. The urinary bladder is markedly distended and demonstrates dense contrast opacification, indicative of vicarious excretion of intravenous contrast material. This finding suggests the presence of an alternative contrast elimination pathway, which can occur in the setting of delayed or impaired renal excretion. The visualized portions of the liver, spleen, and pancreas appear unremarkable.



Figure 2

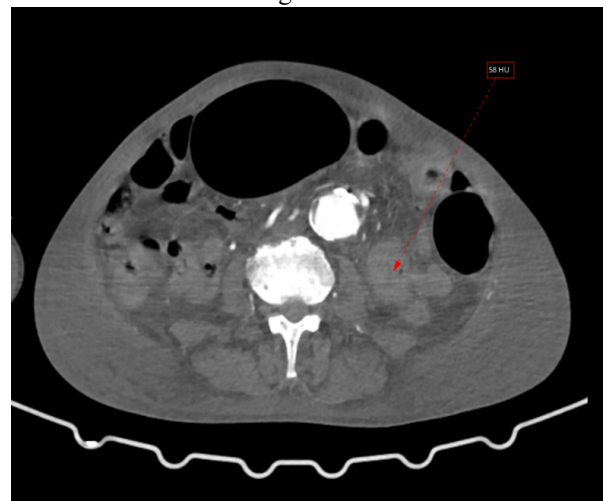


Figure 3

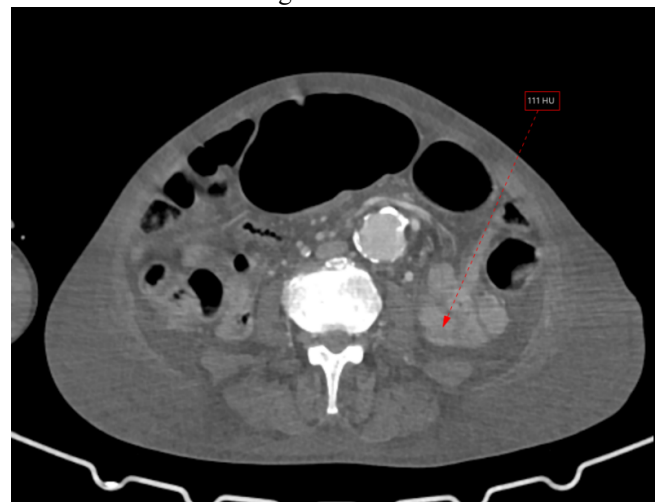


Figure 4

Figures 2, 3 and 4 shows Axial sections of CT Abdominal Angiograms (plain, arterial and venous phases respectively) showing hyper-dense content within the small bowel loops on contrast phases which shows increased attenuation as denoted by the HU values (48 HU in plain, 58 HU in arterial phase and 111 HU in venous phase), possibly denoting vicarious

Vicarious Excretion Of Iv Contrast Through The Small Bowel In A Ct Abdominal Angiogram Study

excretion of IV contrast into the small bowel. The surrounding mesenteric fat planes are preserved, and no abnormal collection or free fluid is noted. The bowel loops contain air and contrast, appearing nondilated with normal wall thickness, without features of obstruction or ischemia. The visualized osseous structures and paraspinal soft tissues are unremarkable.

DISCUSSION:

Vicarious excretion of intravenous (IV) contrast is an uncommon but recognized radiologic phenomenon in which contrast material, instead of being eliminated primarily through the kidneys, is excreted via alternative pathways such as the hepatobiliary system or gastrointestinal tract (1). Normally, iodinated contrast agents are filtered through the glomeruli and excreted in urine. However, in circumstances of impaired or delayed renal function, the body may resort to compensatory elimination routes through the liver, small bowel, or other mucosal surfaces. In the present case, the appearance of hyperdense content within the small bowel loops on CT, without prior oral contrast administration, represents such vicarious excretion.

Vicarious excretion through the bowel has been described in patients with renal insufficiency, dehydration, shock, congestive cardiac failure, or systemic hypotension conditions that reduce renal perfusion and impair glomerular filtration. In this case, the patient presented with hypotension and elevated serum urea and creatinine levels, indicating reduced renal clearance, which likely contributed to the abnormal excretory pathway (2). The intravenous contrast was eliminated through the intestinal mucosa, visualized as dense contrast opacification within small bowel loops on post-contrast images. The pathophysiology is believed to involve increased permeability of the intestinal mucosa, facilitated by mucosal congestion and transudation of contrast material into the bowel lumen.

The proposed mechanism involves both decreased renal clearance and increased permeability of the intestinal mucosa under hypoperfused states, allowing contrast to diffuse into the bowel lumen. Vicarious excretion may also occur through the gallbladder and biliary tract, appearing as opacification of the gallbladder and bile ducts on delayed imaging, but intestinal excretion is far less common. This finding should be differentiated from other causes of bowel hyperdensity, such as oral contrast administration, gastrointestinal bleeding, or retained barium, by correlating with the clinical history and timing of imaging.

The phenomenon is generally transient and resolves as renal function improves, though its recognition is important to avoid misinterpretation as oral contrast administration, gastrointestinal bleeding, or extravasation. In the context of the current case, vicarious excretion coincided with evidence of systemic atherosclerosis and a partially thrombosed

abdominal aortic aneurysm, both contributing to compromised vascular perfusion and renal dysfunction. Similar findings have been documented in literature, particularly in elderly patients with chronic vascular disease and renal impairment.

The similar study conducted by Lee et al. in the year 2025 reported vicarious excretion of contrast into the gallbladder and bowel in a patient with acute renal failure following coronary angiography, emphasizing that such excretion often indicates impaired renal function or delayed clearance (7). Another similar study by Yarmohammadi et al. in the year 2010 also documented cases of biliary and bowel excretion of IV contrast in patients with renal dysfunction and systemic hypotension, suggesting that the severity of renal impairment correlates with the degree of alternative excretion (8). Another study coincides with the study by Kanabolo et al. in the year 2010 described vicarious contrast excretion into both the bowel and urinary bladder in a patient with renal impairment, similar to the present case, highlighting that the phenomenon may coexist through multiple excretory routes (3).

In contrast, normal excretion patterns in patients with preserved renal function show prompt visualization of contrast within the renal collecting systems and urinary bladder, without significant hepatobiliary or intestinal opacification (9). Rarely, mild biliary excretion can occur even with normal renal function but is typically minimal and delayed, unlike the pronounced bowel opacification seen in patients with renal dysfunction.

From a diagnostic standpoint, the observation of vicarious contrast excretion should prompt correlation with renal function tests and hemodynamic status. It serves as an imaging clue to possible renal hypoperfusion or dysfunction (10). Furthermore, awareness of this finding prevents diagnostic errors during interpretation of abdominal CT scans, where hyperdense bowel content might otherwise be mistaken for gastrointestinal hemorrhage or oral contrast residue.

In the present case, the patient exhibited elevated serum urea and creatinine, systemic hypotension, and evidence of atherosclerotic vascular disease, all of which could contribute to reduced renal perfusion and clearance, leading to compensatory intestinal elimination. Recognition of this finding is crucial for radiologists, as vicarious excretion should not be misinterpreted as oral contrast residue or active gastrointestinal hemorrhage. Moreover, its presence may serve as an indirect radiologic marker of underlying renal compromise or circulatory insufficiency (11).

In summary, this case highlights vicarious excretion of intravenous contrast through the small bowel as a rare but significant imaging finding associated with renal impairment and systemic hypoperfusion. Recognition of this phenomenon is essential for accurate image interpretation and for drawing attention to underlying systemic or renal compromise.

CONCLUSION:

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Vicarious excretion of intravenous contrast through the small bowel represents a rare but clinically significant imaging finding, typically associated with renal dysfunction or systemic hypoperfusion. In the present case, intestinal excretion of contrast occurred secondary to impaired renal clearance and reduced perfusion, likely compounded by hypotension and atherosclerotic vascular disease. The presence of hyperdense contrast material within small bowel loops without prior oral contrast administration is a key diagnostic clue to this compensatory excretory mechanism. Recognition of this phenomenon is vital to prevent misinterpretation as gastrointestinal hemorrhage or oral contrast residue and should prompt evaluation of renal and hemodynamic status. Although transient, vicarious excretion serves as an important radiologic marker of renal compromise, reflecting the body's adaptive elimination of contrast through alternative pathways. Awareness and accurate identification of this finding enhance diagnostic confidence and contribute to appropriate patient management.

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