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SUPERIOR SAGITTAL SINUS FILLING DEFECT IN METASTATIC LUNG CANCER: TUMOUR THROMBUS, BLAND THROMBUS OR BOTH?

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ABSTRACT

Background: Cerebral venous sinus thrombosis is an uncommon but potentially life-threatening cause of stroke. In patients with active malignancy, dural venous sinus filling defects present a diagnostic challenge, as they may represent bland thrombosis, tumour thrombus or benign anatomic variants such as arachnoid granulations. Although anticoagulation is recommended for confirmed cerebral venous sinus thrombosis, guidance is limited when imaging findings are equivocal, or tumour involvement is suspected. Diagnostic misclassification may result in unnecessary anticoagulation or failure to treat thrombosis.

Case description: A 66-year-old woman with a history of subtotal colectomy complicated by chronic malabsorption and newly diagnosed metastatic lung cancer underwent brain magnetic resonance imaging for staging. Imaging revealed multiple intracranial metastases and a heterogeneous filling defect within the posterior superior sagittal sinus, with mild sinus expansion and internal flow voids, raising concern for cerebral venous sinus thrombosis or tumour thrombus. Anticoagulation was initiated despite the patient remaining neurologically intact. Subsequent computed tomography angiography and venography demonstrated preserved venous flow and findings consistent with arachnoid granulations rather than thrombosis or tumour invasion, allowing anticoagulation to be discontinued. The patient's altered gastrointestinal anatomy was clinically relevant, as it would have limited the reliability of direct oral anticoagulant therapy.

Conclusions: This case demonstrates that magnetic resonance imaging alone may be insufficient to reliably differentiate cerebral venous sinus thrombosis, tumour thrombus and benign anatomic variants. In patients with active malignancy, confirmatory vascular imaging should be obtained before committing to prolonged anticoagulation. A structured, multimodal diagnostic approach can reduce misdirected therapy and unnecessary risk.

KEYWORDS

Cerebral venous sinus thrombosis, superior sagittal sinus, tumour thrombus, arachnoid granulations, stroke mimics, neuroimaging, anticoagulation

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LEARNING POINTS

- In patients with active malignancy, dural venous sinus filling defects have a broad differential that includes bland cerebral venous sinus thrombosis, tumour thrombus and benign anatomic variants, each with distinct management implications.
- Magnetic resonance imaging findings may be indeterminate; confirmatory vascular imaging with computed tomography angiography or computed tomography venography is often necessary before initiating or continuing systemic anticoagulation.
- Anticoagulation decisions should be individualised, particularly in patients with altered gastrointestinal anatomy or malabsorption, where direct oral anticoagulant efficacy may be unreliable.

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) is an uncommon but clinically significant cause of stroke, accounting for approximately 0.5–3% of cerebrovascular events[1]. Although malignancy is a well-recognised prothrombotic state and cancer-associated CVST is reported across multiple solid tumours[2], an important and unresolved diagnostic challenge arises when a dural venous sinus filling defect is encountered in a patient with active cancer: is the lesion a bland thrombus, a tumour thrombus or a benign anatomic mimic, and how should this distinction guide anticoagulation? Current guidelines uniformly recommend therapeutic anticoagulation for CVST, even in the presence of intracranial haemorrhage[3], yet these recommendations do not address situations where the filling defect may represent tumour infiltration or a non-thrombotic variant such as arachnoid (Pacchionian) granulations. This creates a clinically consequential gap in decision-making, as misclassification can expose patients either to unnecessary bleeding risk from empiric anticoagulation or to the dangers of withholding anticoagulation in true CVST.

Imaging features such as sinus expansion, internal enhancement and intralesional vascularity may raise suspicion for tumour thrombus, but no modality reliably distinguishes tumour thrombus from bland thrombus or benign variants in the cerebral venous sinuses[4,5]. This uncertainty is amplified in patients with cancer, where both tumour invasion and bland thrombosis are plausible, and where the risks of inappropriate treatment are high.

An additional layer of complexity arises when anticoagulation is required in patients with altered gastrointestinal anatomy or malabsorption syndromes. Direct oral anticoagulants (DOACs), including apixaban, rely predominantly on proximal small-bowel absorption, and drug exposure may be unpredictable in patients with subtotal colectomy, short bowel or rapid intestinal transit[6]. In such cases, selecting an anticoagulant with reliable pharmacokinetics becomes an essential component of management.

Here, we present a patient with metastatic lung cancer and a superior sagittal sinus filling defect that was initially concerning for tumour or bland thrombus but ultimately represented arachnoid granulations. This case illustrates the diagnostic ambiguity inherent in venous sinus filling defects, the importance of multimodal imaging before committing to prolonged anticoagulation and the therapeutic challenges encountered when bowel anatomy complicates anticoagulant selection.

CASE DESCRIPTION

A 66-year-old woman with a history of subtotal colectomy complicated by chronic diarrhoea and malabsorption, longstanding tobacco use (~50 pack-years) and a prior episode of portal vein thrombosis, presented with several weeks of progressive right-sided abdominal discomfort, early satiety, fatigue and functional decline. Her abdominal pain began in mid-October 2025 and was described as a dull, postprandial right upper quadrant pressure associated with markedly reduced oral intake. She denied headache, visual changes, focal neurologic deficits or pulmonary symptoms.

Initial laboratory testing was largely unremarkable: haemoglobin 13.8 g/dl, white blood cell count 7.2 $\times 10^9$/l, platelets 258 $\times 10^9$/l, sodium 137 mmol/l, creatinine 0.9 mg/dl, aspartate aminotransferase (AST) 32 U/l, alanine aminotransferase (ALT) 28 U/l, alkaline phosphatase 110 U/l, total bilirubin 0.7 mg/dl and international normalised ratio (INR) 1.0. Lactate dehydrogenase (LDH) was markedly elevated at 2,488 U/l, consistent with high tumour burden and rapid cellular turnover. The absence of leucocytosis, electrolyte derangements or coagulopathy suggested that her presentation was unlikely due to infection, acute hepatic dysfunction or metabolic instability.

Initial staging imaging included a low-dose computed tomography (CT) of the chest that demonstrated a left hilar and infrahilar mass extending into and encasing the left mainstem bronchus, with bulky mediastinal and hilar adenopathy. A CT of the abdomen/pelvis revealed numerous hepatic lesions with marked hepatomegaly (approximately 26 cm), consistent with suspected metastatic lung cancer. Given this constellation of findings, the patient was referred for further oncologic evaluation.

Magnetic resonance imaging (MRI) of the brain with and without contrast revealed approximately 9–10 enhancing metastatic lesions distributed throughout the cerebrum and cerebellum. In addition, MRI demonstrated a heterogeneous filling defect within the posterior superior sagittal sinus measuring approximately 32 × 15 mm *(Fig. 1)*. The lesion was associated with mild sinus expansion and contained multiple internal linear flow voids, raising concern for possible

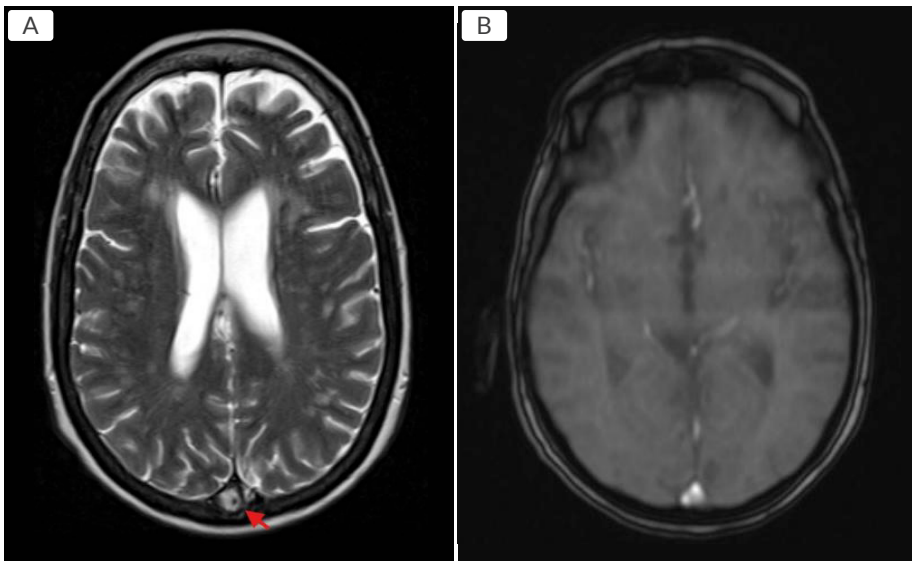


Figure 1. MRI of the brain demonstrating a posterior superior sagittal sinus filling defect. A) Axial T2-weighted MRI showing a well-defined lobulated filling defect at the posterior superior sagittal sinus (red arrow), consistent with prominent arachnoid granulations. B) Axial T1 post-contrast MRI demonstrating the corresponding lesion without evidence of sinus expansion or intraluminal enhancement.

venous sinus thrombosis although not sure whether bland or tumour thrombus, and prompting the recommendation to initiate intravenous unfractionated heparin. Diffusion-weighted imaging was limited by motion artefact but did not clearly demonstrate restricted diffusion within the filling defect. The sinus appeared patent proximal and distal to the lesion.

Because of concern for CVST in the setting of metastatic disease, the patient was transferred to a tertiary centre for neurosurgical and neurocritical care evaluation. On arrival, she remained neurologically intact with no signs of increased intracranial pressure. A computed tomography angiography (CTA)/computed tomography venography (CTV) of the head was obtained to better characterise the lesion. The CTA demonstrated that the filling defect corresponded to prominent arachnoid (Pacchionian) granulations rather than true thrombus, with preserved flow through the superior sagittal sinus and no evidence of occlusion. Based on these findings, systemic anticoagulation was safely discontinued.

The patient's history of subtotal colectomy and chronic malabsorption was clinically relevant because it limited the suitability of direct oral anticoagulants (DOACs), and warfarin or low–molecular-weight heparin (LMWH) would have been required if true thrombosis had been confirmed. She was concurrently undergoing evaluation for tissue diagnosis and CT-guided liver biopsy was planned to establish histology.

DISCUSSION

This case highlights a clinically significant and under-addressed diagnostic dilemma: distinguishing tumour thrombus from bland thrombus or an anatomic mimic within the cerebral venous sinuses and determining how anticoagulation decisions should be made when imaging findings are ambiguous[6]. In patients with active malignancy, a superior sagittal sinus filling defect may represent several possibilities, including bland CVST, tumour thrombus from direct venous invasion or intraluminal metastasis, mixed tumour–bland thrombus, arachnoid (Pacchionian) granulations or congenital sinus variants. Although this

Feature	Bland CVST	Tumour thrombus	Arachnoid granulations
Definition	Thrombus within a dural venous sinus	Intraluminal extension of tumour cells	Benign protrusions of arachnoid villi
MRI findings	Intrinsic filling defect; may show sinus congestion	Heterogeneous filling defect; may show sinus expansion or internal flow	Well-defined lobulated defect; no expansion
CTA/CTV findings	Filling defect with partial or complete sinus obstruction; may show reduced flow	Enhancing intraluminal mass; sinus expansion may be present	Non-obstructive filling defect with preserved venous flow; no enhancement
Typical associations	Hypercoagulable states, including cancer	Advanced or metastatic malignancy	Incidental anatomical variant
Treatment	Anticoagulation is recommended because thrombosis at this location within the cerebral venous sinuses can propagate and lead to venous infarction and intracranial hypertension	Treat underlying malignancy; anticoagulation is not routinely required	No treatment

Table 1. Key differences between bland CVST, tumour thrombus and arachnoid granulations.

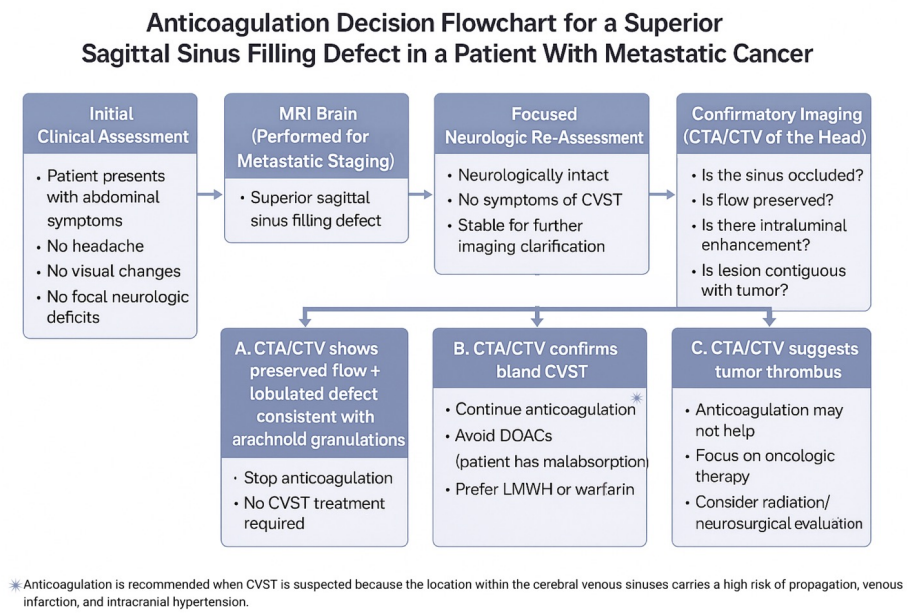


Figure 2. Anticoagulation decision flowchart for a superior sagittal sinus filling defect. A stepwise clinical pathway outlining the evaluation of a superior sagittal sinus filling defect in a patient with metastatic lung cancer, distinguishing arachnoid granulations, bland CVST and tumour thrombus.

differential is broad, it is critical to evaluate systematically because each entity carries different implications for management. For example, bland CVST is the most immediately dangerous if untreated, whereas arachnoid granulations are benign and require no therapy. A concise comparison of the most relevant differential diagnoses is provided in *Table 1*.

Imaging features may offer clues, but none are definitive. Early vascular ultrasound studies suggested that tumour thrombi often contain internal neovascularity, with one Doppler study demonstrating intrathrombus vascular flow in six of seven tumour thrombi, compared with an absence of flow in bland thrombi[7]. However, such findings have not been validated in the cerebral venous system where sinus expansion, internal linear flow voids or enhancement patterns can also be seen in benign variants such as arachnoid granulations. This limitation is especially important because treating a true CVST as tumour thrombus and withholding anticoagulation can result in catastrophic venous infarction, whereas anticoagulating an anatomic mimic exposes the patient to unnecessary bleeding risk.

Current CVST guidelines from the American Heart Association/American Stroke Association and the European Stroke Organisation[3,4] universally recommend therapeutic anticoagulation even in the presence of intracranial haemorrhage because propagation of a bland thrombus may lead to malignant intracranial hypertension, herniation and death[8]. However, these guidelines do not address situations in which tumour thrombus is suspected or when imaging findings cannot reliably distinguish between tumour, bland thrombus and benign variants. This gap leaves clinicians to extrapolate from limited or indirect data. Our case illustrates this problem clearly: MRI demonstrated a heterogeneous filling defect with sinus expansion and internal flow voids, features that could suggest tumour thrombus, yet CTA ultimately confirmed that the finding represented prominent arachnoid granulations with preserved venous patency. Without confirmatory multimodal imaging, the patient would have remained on unnecessary anticoagulation. This highlights the importance of obtaining CTA or CTV before committing to prolonged anticoagulation in cases where MRI findings are equivocal.

In addition to the diagnostic uncertainty, our patient had a unique therapeutic consideration related to her history of subtotal colectomy and chronic malabsorption. Although this nuance is uncommon, it has important implications for anticoagulation if true CVST is confirmed. Apixaban and other DOACs rely primarily on proximal small-bowel absorption, with sharply reduced uptake distally[9,10]. In patients with altered gastrointestinal anatomy or rapid transit times, DOAC exposure may be unpredictable and potentially subtherapeutic, increasing the risk of treatment failure[11]. For such patients, LMWH or warfarin, agents with more predictable pharmacokinetics or measurable effect, may be safer options. In this case, once CTA demonstrated that the lesion represented Pacchionian granulations rather than thrombus, anticoagulation could be safely discontinued, but if therapy had been required, DOAC malabsorption would have influenced anticoagulant selection.

Overall, this case underscores the absence of standardised guidance for evaluating suspected cerebral venous tumour thrombus and demonstrates the limitations of relying on MRI alone for differentiation among tumour thrombus, bland thrombus and benign anatomical variants. It also illustrates the clinical consequences of misclassification in patients with malignancy, where both overtreatment and undertreatment carry substantial risk. Based on the diagnostic challenges illustrated in this case, we outline a streamlined clinical pathway to assist in evaluating indeterminate venous sinus filling defects *(Fig. 2)*. Together, these issues highlight the need for clearer diagnostic frameworks and practice recommendations to guide clinicians confronted with indeterminate venous sinus filling defects in patients with cancer.

CONCLUSION

In patients with malignancy, a superior sagittal sinus filling defect presents a broad differential that includes bland CVST, tumour thrombus, mixed thrombus and benign anatomic variants such as arachnoid granulations. Because MRI findings alone may be insufficient to accurately distinguish among these possibilities, premature classification can lead either to unnecessary anticoagulation or to dangerous undertreatment of true CVST. This case demonstrates the essential role of multimodal imaging, particularly CTA or CTV in clarifying ambiguous venous sinus abnormalities and preventing misdirected therapy.

Our patient also exemplifies how patient-specific factors, such as altered gastrointestinal anatomy affecting DOAC absorption, may further complicate anticoagulation decisions. These considerations underscore the importance of individualised therapeutic planning when anticoagulation is required.

Ultimately, this case highlights a broader need for structured diagnostic approaches and clearer clinical pathways when evaluating indeterminate dural venous sinus filling defects in patients with cancer. A systematic evaluation that incorporates clinical context, careful radiologic interpretation and confirmatory vascular imaging can help ensure accurate diagnosis and guide safe, evidence-informed management.

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