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Case Report

Trousseau's syndrome with cerebral infarction as the first manifestation

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Abstract: Objectives: This study aims to highlight the clinical significance of Trousseau's syndrome in cancer patients, particularly focusing on arterial thromboembolism (ATE), which remains understudied compared to venous thromboembolism (VTE). This report emphasizes the need for clinical vigilance in diagnosing Trousseau's syndrome when cancer patients present with atypical thromboembolic events. Methods: The analysis proposes diagnostic criteria for suspected Trousseau's syndrome in cancer patients presenting with acute multiple cerebral infarctions. Key indicators include: a) Absence of severe cerebrovascular disease risk factors; b) Imaging evidence of lesions spanning multiple arterial territories or exhibiting the "Three Territory Sign" (TTS); c) Elevated D-dimer levels; d) Abnormal tumor markers or confirmed tumor lesions. Results: Clinical observations reveal that cancer patients with acute multifocal cerebral infarction, particularly those demonstrating multi-arterial territory involvement or TTS in neuroimaging, coupled with significantly elevated D-dimer (>5 μg/mL) and tumor markers (e.g., CEA, CA125), show high correlation with Trousseau's syndrome. These findings differentiate it from conventional atherosclerotic thromboembolism. Conclusions: Trousseau's syndrome should be prioritized in differential diagnoses for cancer patients presenting with unexplained arterial thromboembolic events. Early anticoagulation therapy (e.g., low molecular weight heparin) rather than antiplatelet therapy is recommended as the primary intervention. This approach addresses the unique hypercoagulable state driven by tumor-associated fibrinolysis abnormalities, potentially reducing recurrent thromboembolic complications.

Keywords: Trousseau's syndrome, cerebral infarction, three territory sign, tumor markers, D-dimer

Introduction

The first clinical observation of a connection between cancer and thrombosis was reported in 1823 by Jean Baptiste Bouillaud [1]. In 1865, Armand Trousseau was the first to describe the relationship between venous thrombosis and malignancy, suggesting that cancer is closely linked to an increased risk of thrombotic embolism, which is characterized by spontaneous intravascular coagulation [2]. All instances of thrombotic embolism in cancer patients resulting from abnormalities in fibrinolysis and coagulation mechanisms during the course of the disease are now collectively referred to as Trousseau's syndrome [3]. Current research on Trousseau's syndrome primarily focuses on venous system embolism, including pulmonary embolism and deep vein thrombosis. In contrast, there have been few reports and studies on arterial system embolism, especially in patients without a history of cancer, which often leads to underdiagnosis and misdiagnosis, with no standardized treatment approach. This

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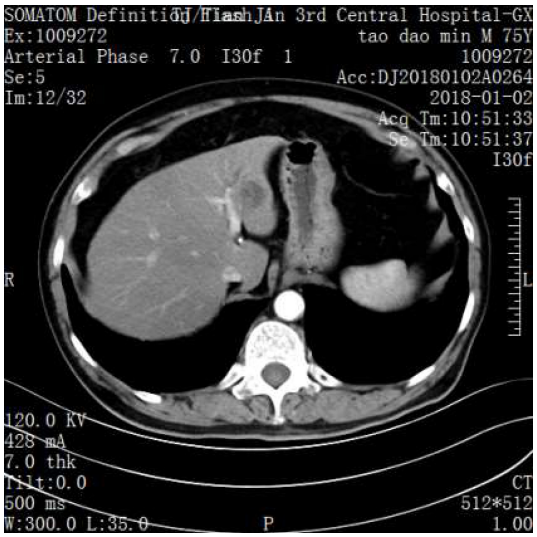

Figure 1. Abdominal CT. Abnormally enhanced nodule in the left outer lobe of the liver.

study aims to discuss the pathogenesis, clinical and imaging features, and treatment strategies of Trousseau syndrome, with cerebral infarction as the main presentation, in order to enhance clinicians' understanding of diagnosing and treating this condition.

Case reports

A 75-year-old male presented to the hospital for surgery on 02.01.2018 due to a hepatic occupying lesion that had been detected during a physical examination more than 6 months prior. An abdominal Computed Tomography (CT) (**Figure 1**) revealed an abnormally enhancing nodule in the left outer lobe of the liver, which was suspected to be a malignant lesion. The patient underwent left hemicolectomy + cholecystectomy and the pathology results showed cholangiocarcinoma with moderate to low differentiation. He was discharged after surgery on 23.10.2019. Later, on 23.10.2019, the patient returned to the hospital presenting symptoms of "numbness and weakness of the left limb, dysarthria for 4 hours". A Cranial Magnetic Resonance Imaging (MRI) (**Figure 2A-C**) revealed multiple infarcts in the brainstem (**Figure 2A**), right posterior horn of the lateral ventricle (**Figure 2B**), and Bilateral hemioval central area (**Figure 2C**). Following treatment and good control of blood glucose, blood pressure, and blood lipids, the patient was discharged from the hospital after 14 days. However, the patient was readmitted to the hospital on 17.11.2019, just 24 days after the first infarction, with "aggravated weakness of left limb and dysarthria for 3 days". Another Cranial MRI (**Figure 2D**) revealed a recurrent brainstem infarction. It was determined that the patient had Trousseau's syndrome combined with cerebral infarction and he was treated with anticoagulants. After showing improvement, he was discharged. Unfortunately, the patient was readmitted on 26.12.2019 with symptoms of "choking on food with left lower limb weakness for 1 day". A cranial MRI (**Figure 2E**, **2F**) indicated that the infarction was likely caused by atherosclerosis and a malignant tumor. The patient was treated with anti-platelet and anticoagulant medications before being discharged without recurrence. The patient's treatment, tumor markers, D-dimer levels, and MRI results are summarized in **Table 1**.

In a separate case, a 72-year-old male was admitted to the hospital with the main complaint of "dizziness for 10 days". He had a history of hypertension with well-controlled blood pressure and had experienced deep vein thrombosis in his right lower extremity over 20 days ago. Upon examination, cranial MRI (**Figure 3**) revealed multiple cerebral infarcts in the bilateral cerebellar hemispheres (**Figure 3A**, **3B**), left occipital lobe and right frontal lobe (**Figure 3C**), bilateral lateral paraventricular (**Figure 3D**), left frontal lobe and bilateral parietal lobes (**Figure 3E**, **3F**). An intensive chest CT (**Figure 4A**) showed a partial filling defect in the pulmonary artery in the lower lobe of the left lung, indicating a small embolism. Additionally, an intensive abdominal CT (**Figure 4B**, **4C**) revealed small hypointense shadows without enhancement in the spleen and both kidneys, which were suggestive of embolisms. Further tests showed elevated blood chemistry tumor markers (**Table 2**) and abnormal coagulation, with significantly elevated D-dimer levels (**Table 3**), indicating a hypercoagulable state. As a result, the patient started oral anticoagulation treatment with rivaroxaban. While the D-dimer levels did not decrease significantly, the patient's dizziness improved, and he was discharged with the prescribed medication. However, two days later, the patient was readmitted to the hospital with weakness in his right limb for one day. A repeat cranial MRI showed an increase in the number of infarct

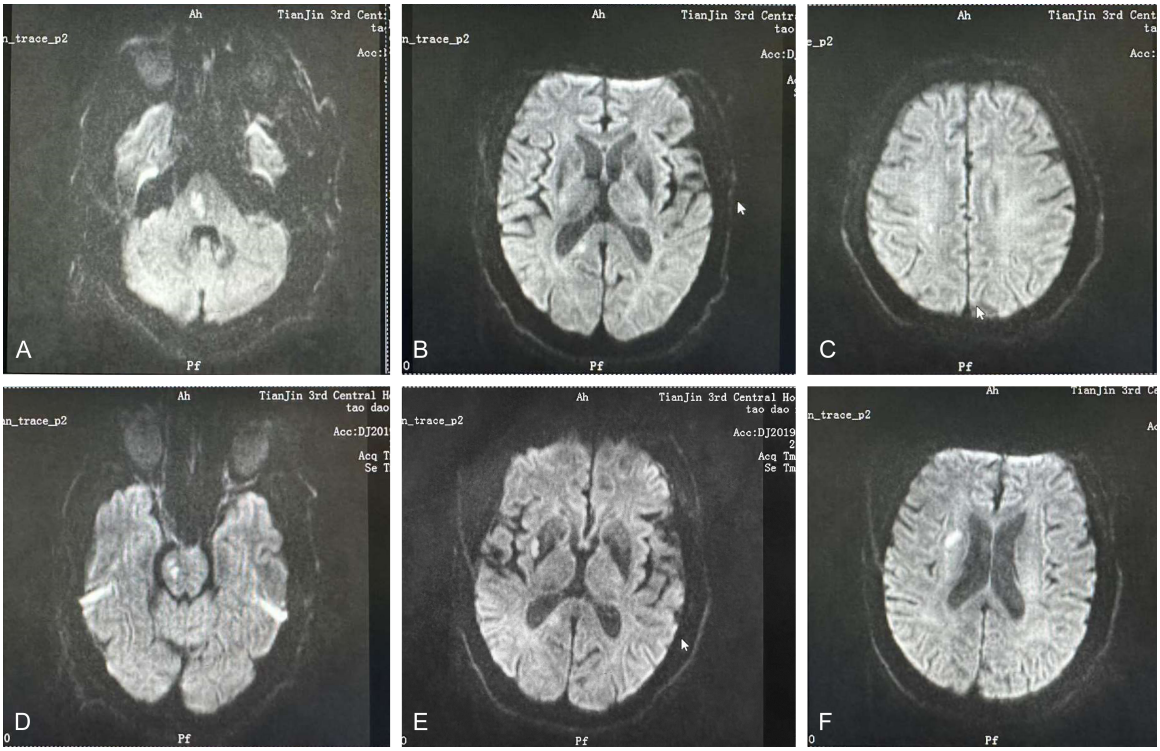

Figure 2. Cranial MRI. A. Multiple infarcts in the brainstem area. B. Right lateral ventricular posterior horn infarct. C. Bilateral hemi-oval central area. D. Brainstem infarct infarct. E. Infarct in the right basal ganglia. F. Infarct in the right basal ganglia.

Table 1. Tumor markers, D-dimer, head MRI performance and treatment during the patient's hospitalization

	Tumor markers		D-dimer (<0.3 mg/L)	MRI manifestations of the head	Treatment
	Squamous epithelial cells (<1.5 ng/ml)	Glycoantigen 72-4 (<8.2 u/m)			
1.2.2018 (pre-op)	4.6	214.7	0.21		
1.17.2018 (post-op)			1.97		
1.23.2018 (post-op)	1.8	53.77			
10.24.2019	1.2	52.58	0.28	Multiple infarcts in the brainstem area, right lateral ventricular posterior horn parietal and right hemi-oval central area	Clopidogrel hydrogen sulfate
11.18.2019			0.26	Brain stem infarction	Rivaroxaban
12.26.2019			<0.1	Multiple infarcts in the right basal ganglia area	Clopidogrel hydrogen sulfate + Rivaroxaban

foci in various parts of the brain. The D-dimer levels remained elevated, so the patient was switched to natriuretic heparin calcium subcutaneous injections to enhance anticoagulation therapy before being discharged in a stable condition. The patient's recurrent cerebral infarctions within one month, combined with pulmonary embolism, lower limb venous thrombosis, splenic embolism, renal embolism, elevated tumor markers, and hypercoagulable state, led to the diagnosis of Trousseau's syndrome. A Positron Emission Tomography (PET) examina-

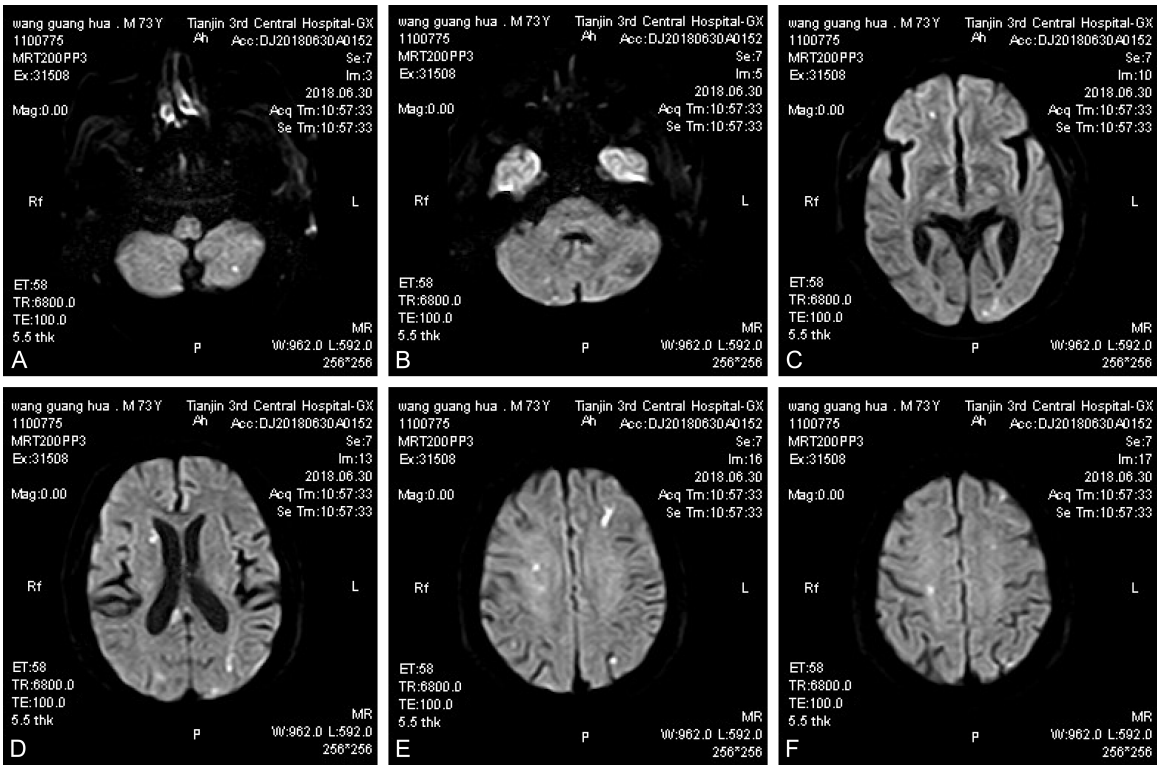

Figure 3. Cranial MRI. A, B. Bilateral cerebellar hemispheres. C. Left occipital lobe and right frontal lobe. D. Bilateral lateral paraventricular. E, F. Left frontal lobe and Bilateral parietal lobes cerebral infarcts.

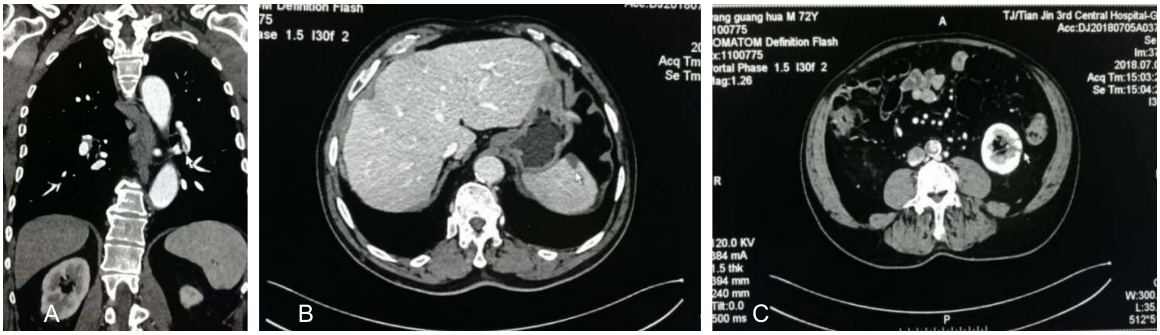

Figure 4. Intensive chest and abdominal CT. A. Intensive chest CT. Intra-pulmonary filled defect in part of the lower lobe of the left lung, with consideration of a small embolism. B. Intensive abdominal CT. Small hypointense shadow without enhancement is seen in the spleen with clear borders, consider embolism. C. Intensive Abdomen CT. Multiple non-enhancing hypodense shadows with well-defined borders are seen in both kidneys, consider considering embolism.

tion revealed multiple enlarged lymph nodes in the neck and chest with high metabolism, raising suspicion of malignancy. Subsequently, the patient was referred to a specialized hospital for further treatment.

Discussion

Trousseau's syndrome was initially identified as a connection between thrombosis in the venous system and malignancy, with patients who have malignancies accounting for 17-29% of all patients with VTE [4]. In a prospective study on thrombosis, 7.7% of patients diagnosed or detected with cancer developed VTE within a year [5]. The focus has traditionally been on the link between thrombosis and malignancy, with previous research exploring their relationship and mechanisms. Recent cohort studies have shown that cancer also sig-

Table 2. Significantly elevated tumor markers

Tumor markers	Result	The normal range of values
Squamous epithelial cell carcinoma antigen	0.80 ng/ml	<1.5
Glycoantigen 19-9	24.46 u/ml	<39.0
Glycoantigen 72-4	>300 u/ml ↑	<8.20
carcinoembryonic antigen	44.82 ng/ml ↑	<5.00
alpha fetoprotein	3.73 ng/ml	<15.00
Neuronal dilute alcoholase	20.28 ng/ml ↑	<15.2

Table 3. Coagulation abnormalities

Inspection items	07-02	07-07	07-16	07-18
PT	46	90	29	81
INR	1.74	1.07	2.62	1.14
PT-sec (S)	20.2↑	14.0	27.7↑	14.8
APTT	42.1	37.9	40.2	33.7
Fig (4 g/L)	5.57↑	4.40↑	2.92	2.39
D-Dimer (<0.3 mg/L)	0.92↑	1.18↑	1.18↑	2.10↑

nificantly increases the risk of arterial thrombosis [6], with cancer patients experiencing a higher risk of ischemic stroke [7]. Zoller observed a 1.6% incidence of cerebral infarction within six months of detecting malignancy [8], indicating a potential direct or indirect link. This article discusses two patients with a combination of cerebral infarction and systemic arterial thrombosis due to malignancy.

In the first case discussed in this paper, the patient had a history of hypertension and recurrent cerebral infarction following pancreatic cancer. In the second case, the patient was also hypertensive, but his blood pressure was well managed. He was hospitalized due to recurrent cerebral infarction, and his Cranial MRI revealed multiple vascular supply areas in the anterior and posterior circulation. During his hospital stay, he experienced a pulmonary embolism, splenic embolism, renal embolism, prehospital lower limb venous thrombosis, significantly elevated tumor markers, elevated D-dimer levels, difficulty controlling blood hypercoagulability with early rivaroxaban, eventually stabilized with subcutaneous anticoagulation using calcium natriuretic heparin. Additionally, PET-CT scans showed lymph node enlargement, raising suspicion of a malignant tumor.

Combining these two patients, we consider that the occurrence of cerebral infarction in patients with malignancy may be related to the following aspects: Risk factors associated with the patient's own arterial embolism include age, as tumor patients are older at the onset of the disease, which is also the age of high incidence of cardiovascular and cerebrovascular disease. It is possible that some of these patients already have a history of embolism when they develop cancer. In addition, this group of patients may have one or more risk factors for atherosclerosis. Risk factors for malignancy, such as the hypercoagulable state associated with the tumor, the type of cancer, and the extent and degree of the tumor can all influence the risk of ATE. The risk of ATE associated with malignancy has been reported to vary over the disease period, being highest in the first 6 to 12 months after diagnosis and then decreasing [8-10]. The two patients in this paper are consistent with these reports. Conversely, the risk of VTE peaks within the first six months after diagnosis. Additionally, risks associated with malignancy treatment, such as platinum drugs, vascular endothelial growth factor inhibitors, tamoxifen, erythropoietin, and radiation therapy, can cause a hypercoagulable state leading to embolism. The pathogenesis of tumor-combined stroke differs from the conventional pathogenesis of stroke in that chronic inflammation and an activated coagulation system may be involved in the causative mechanisms [11]. Tumor cells express procoagulants and release inflammatory cytokines and vascular endothelial growth factor, which enhance procoagulant activity and angiogenesis [12]. Trousseau's syndrome appears to occur more frequently in older male patients and is predominantly associated with adenocarcinoma of the lung, esophagus, liver, stomach, colon, and breast [13]. A large retrospective cohort study found a higher incidence of cerebral infarction in patients with adenocarcinoma compared to other types of malignan-

cies [14]. All 31 patients reported in a related study had confirmed adenocarcinoma upon biopsy. Similarly, a recent case series study found that 7 out of 8 patients with ischemic stroke caused by Trousseau's syndrome were diagnosed with adenocarcinoma [15]. These findings suggest that adenocarcinoma is a significant risk factor for brain infarction in Trousseau's syndrome. The reason for the prevalence of adenocarcinoma in this syndrome is not yet clear, but it is hypothesized that adenocarcinoma releases mucins that interact with leukocytes and platelet selectin to form platelet-rich microthrombi, leading to thromboembolism. In this paper, both patients affected were elderly males. One patient had cholangiocarcinoma of the adenocarcinoma type, while the other had suspected lung cancer based on elevated levels of carcinoembryonic antigen and neuronal enolase, consistent with existing literature.

Trousseau's syndrome blood chemistries, along with possible elevation of corresponding tumor markers, are usually accompanied by elevated D-dimer levels. Some studies have confirmed an association between elevated D-dimer levels and cancer-related cerebral infarction. This suggests that elevated D-dimer levels (>5.5 mg/L) have a sensitivity and specificity exceeding 93% [16]. In an analysis case series by Bao, 29 out of 31 patients with Trousseau's syndrome (93.54%) had significantly elevated D-dimer levels. Patients with Trousseau's syndrome also had the highest plasma D-dimer levels compared to other stroke subtypes, with a mean of 6.545 mg/L [17]. In conclusion, a significantly elevated D-dimer level can help differentiate cerebral infarction with Trousseau's syndrome from other causes and Trousseau's syndrome may be a cause of cryptogenic stroke.

In the diagnosis and research of Trousseau's syndrome, thromboelastography, as a common clinical method for detecting the entire coagulation cycle, should theoretically play a significant role in this study [18]. This technique can provide information on multiple aspects of coagulation, including initiation, fibrin formation, platelet aggregation, and fibrinolysis, aiding in a comprehensive assessment of the patient's coagulation status [19]. However, during the actual treatment and diagnosis of this case, this technology was not used, which may introduce certain limitations to the study of the disease. On one hand, the lack of thromboelastography may lead to insufficient understanding of the severity and specific aspects of coagulation abnormalities in patients. For patients with Trousseau syndrome, accurately assessing changes in coagulation function is crucial for diagnosing the severity of the condition, predicting the risk of thrombosis recurrence, and formulating appropriate anticoagulant therapy plans. On the other hand, this omission also highlights the importance of using comprehensive methods to evaluate coagulation function in similar case studies. Future research should strive to improve diagnostic tools to enhance our understanding of diseases like Trousseau syndrome, which are closely related to coagulation abnormalities. Although thromboelastography was not used in this case, Trousseau syndrome can still be diagnosed based on stroke symptoms, combined with the cancer history of both patients and other relevant test results. This highlights the need for the integration of multiple pieces of clinical information for diagnosis and decision-making, rather than relying solely on a single specific testing method.

Trousseau's syndrome also shows a unique "Three Territory Sign" on imaging, with acute infarction (DWI) in both the anterior and posterior circulation [20]. Two possible mechanisms of formation are considered: First, the hypercoagulable state of blood caused by malignancy can result in intracranial arterial microthrombosis and disseminated intravascular coagulation (DIC), with no regional differences in intracranial microthrombi in situ. Second, chronic inflammation caused by malignancy may lead to non-bacterial thrombotic endocarditis, where the small organisms involved can easily dislodge and circulate as microemboli, causing embolization of small branches of intracranial arteries. These factors contribute to a greater number of acute infarct foci and a wider area of vascular involvement in patients with Trousseau's syndrome [21, 22].

The literature states that the treatment for Trousseau's syndrome involves anticoagulation and addressing the underlying tumor. While the cancer is not under control, low molecular weight heparin or warfarin are the only options for treating Trousseau's syndrome. If the ma-

lignancy is managed, D-dimer levels decrease, reducing the risk of cerebral infarction [23, 24]. In the first patient, we utilized a new oral anticoagulant, rivaroxaban, to manage the patient's condition due to the limitations of heparin and warfarin, based on literature and the pathogenesis of cerebral infarction in Trousseau's syndrome. The second patient received oral rivaroxaban treatment after heparin therapy, and both patients achieved satisfactory results with no recurrence of infarction. In cases where patients display acute multiple foci of cerebral infarction, without severe cerebrovascular disease risk factors, and lesions affecting multiple arterial supply areas or the "Three Territory Sign", along with significantly high D-dimer levels and elevated tumor markers or confirmed tumor lesions, there should be a suspicion of Trousseau's syndrome. Anticoagulation therapy is crucial for treatment.

Disclosure of conflict of interest

None.

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