Isolated Distal Deep Vein Thrombosis Associated with Adenomyosis: Case Report and Literature Review

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1. INTRODUCTION

Thromboembolism is a common and potentially life-threatening complication that can arise from various medical conditions, including malignancy and a spectrum of benign diseases. Adenomyosis, characterized by the presence of endometrial glands and stroma within the myometrium, is a benign uterine disorder that typically presents in middle-aged postpartum women with symptoms of menorrhagia, dysmenorrhea, and chronic pelvic pain. The true prevalence of adenomyosis remains unknown, although estimates derived from patients referred for pelvic imaging suggest a range of 20% to 34%.[1] The association between adenomyosis and thrombosis was first described by Kupryjanczyk in 1991.[2] Subsequently, several isolated case reports and case series have documented the occurrence of ischemic stroke[3-20] and pulmonary embolism (PE)[21, 22] in patients with adenomyosis, suggesting a possible association with hypercoagulability induced by this disorder. However, there are few reports of cases complicated by isolated deep vein thrombosis (DVT). [23, 24] In this report, we present a case of isolated distal deep venous thromboembolism (IDDVT) in a patient with a 1.5-year history of adenomyosis. We also conducted a comprehensive literature review to explore the clinical features, treatment approaches, and prognosis of thrombotic complications associated with this condition.

2. CASE PRESENTATION

A 50-year-old woman presented at our outpatient clinic with acute pain in her right lower limb after long-distance travel. She complained of a significant exacerbation of the pain over the past 10 hours, despite the administration of oral diclofenac sodium. Given the potential diagnosis of DVT, the patient was subjected to D-dimer and lower extremity venous ultrasound examinations. As expected, her D-dimer and fibrin degradation product (FDP) levels were found to be markedly elevated at 6.19 μg/mL (normal <0.5 μg/mL) and 25 μg/mL (normal <5.0 μg/mL), respectively. The ultrasound examination revealed the presence of intermuscular vein thrombosis in her calf of the right lower limb (Figure 1A). She was admitted to our hospital and initiated anticoagulation therapy using heparin in combination with Panax notoginseng saponins, a traditional Chinese medicine known for its anti-inflammatory, antioxidant, and cardiovascular protective effects.

The patient presented with stable vital signs on admission, including a blood pressure of 120/80 mmHg, heart rate of 80/min, respiratory rate of 20/min, and a temperature of 36.2°C. She reported no chest pain, chest tightness, or dyspnea. A chest computed tomography (CT) scan was unremarkable. She married at an adult age (G1, P1), and her husband and child were in good health. She was on the third day of her menstrual cycle. She had no history of hypertension, diabetes mellitus, cardiovascular disease, or venous thromboembolism. She also denied any family history of these conditions. The patient had previously been

diagnosed with adenomyosis 18 months ago and had undergone three months of gonadotropin-releasing hormone agonist (GnRHa) therapy. Laboratory tests revealed significantly elevated levels of carbohydrate antigen 125 (CA125) at 687 U/mL (normal range < 35 U/mL) and carbohydrate antigen 199 (CA199) at 64.5 U/mL (normal range < 35 U/mL), along with a slight reduction in hemoglobin concentration (111 g/L, normal range: 115-150 g/L). There were no significant findings for coagulation function, platelet count, biochemistry panel, autoantibodies, antiphospholipid antibodies, or lupus anticoagulant tests. Given the elevated tumor biomarker levels, an abdominal CT scan and gynecologic ultrasound were performed to rule out malignancy. No space-occupying lesions were detected, but a significantly enlarged uterine corpus (Figure 1B) confirmed adenomyosis via ultrasonography (Figure 1C).

After a 7-day course of heparin anticoagulation therapy, the patient experienced complete resolution of lower limb pain and a reduction in D-dimer levels to 0.87 μg/mL. However, her CA125 level remained high at 401.8 U/mL (Figure 2). Since she did not report any discomfort, she was discharged from our hospital with a prescription for oral rivaroxaban. A 6-month telephone follow-up was conducted to monitor her prognosis. In the second month post-discharge, the patient underwent a hysterectomy procedure at a specialized hospital for adenomyosis treatment. One month after the procedure, repeat tests for CA125, CA199, and D-dimer revealed values within the normal range. No thrombus recurrence was observed during the 6-month follow-up period. The patient signed an informed consent form for publication on December 20, 2023.

3. DISCUSSION

This report presents a case of IDDVT that was potentially associated with adenomyosis. The patient experienced a thrombotic episode on the first day following long-distance travel, which coincided with her menstrual period. We suggest that the combined effects of hypercoagulability related to adenomyosis and blood stasis from long-distance travel may have contributed to the development of IDDVT. The patient was effectively managed with heparin bridging rivaroxaban and subsequently underwent a hysterectomy at another hospital to reduce the risk of recurrence.

The true incidence of adenomyosis-related thrombotic complications remains uncertain. Ischemic stroke has been observed in 0.1˜0.8% of patients with adenomyosis according to early single-center studies.[25] Limited data are available regarding other types of thrombotic events. To further investigate this association, we conducted a literature search on PubMed using the terms “adenomyosis” AND (thrombo* OR infarction). This search strategy yielded a total of 22 eligible case or series reports (Table 1), including 25 cases of ischemic stroke,[3-20]6 cases of PE,[21, 22] and 3 cases (including the present case) of DVT.[23, 24] Most patients were between the ages of 30 and 50, and they tended to experience thrombotic events during their menstrual periods. These findings can be attributed to the estrogen-dependent nature of adenomyosis, which is rarely diagnosed in premenarchal or postmenopausal women.

As summarized in Table 1, both arterial and venous thromboembolism have been documented in patients with adenomyosis. Among these cases, multiple cerebral infarctions were the most frequently observed, accounting for 18 out of 34 cases. Notably, 7 of these cases exhibited systemic embolism,[3,4,9,13,15,17] resembling Trousseau’s syndrome commonly seen in cancer patients. These observations suggest that hypercoagulability may play a significant role in the pathogenesis of thrombotic complications related to adenomyosis. Consistent with this speculation, previous studies have identified a procoagulant state in patients with adenomyosis, which is further exacerbated during menstruation and accompanied by activation of fibrinolysis.[26]

The mechanisms underlying hypercoagulability in patients with adenomyosis may be multiple. First, an early study found increased tissue factor (TF) reactivity in ectopic endometrium obtained from women with adenomyosis compared to normal endometrium, and elevated TF activity was associated with the severity of disease.[27] Second, the normal endometrium experiences repeated proliferation, decidualization, and shedding across the menstrual cycle, which is a fine balance between tissue injury and repair tuned by the endocrine, immune, vascular, and coagulation systems.[28] However, adenomyosis may disrupt this balance, and the ectopic endometrium and its associated vascular malformations can lead to impaired spontaneous decidualization, resulting in persistent inflammation and hemorrhage, which subsequently triggers the throm-

boinflammation pathway. Last, it is worth noting that the thickened endometrium during menstruation is rich in mucins, which have been associated with thrombophilia in individuals with mucinous cancer. The endometrium serves as the primary source of CA125 in females, which can rise to 8 times the upper reference limit (35 U/mL) during menstruation and return to baseline at the conclusion of the menstrual cycle.[29] In the context of adenomyosis, it is plausible to hypothesize that impaired decidualization may lead to sustained elevation of CA125 levels for prolonged durations, thereby increasing the risk of thrombophilia in these patients. The current case, along with previous reports, provides supportive evidence for this hypothesis. Notably, elevated CA125 levels have been observed in certain patients during their nonmenstrual periods,[4,7,8] and in our case, the elevated CA125 level persisted for approximately two months until hysterectomy was performed (see Figure 2).

The association between CA125 levels and the severity of adenomyosis remains uncertain. Nevertheless, a pooled analysis of documented cases[3-20] revealed a significant increase in CA125 levels among individuals experiencing recurrent or systemic thromboembolism, in contrast to those with nonrecurrent and isolated embolism (see Figure 3). This preliminary investigation emphasizes the need for further research to establish the potential value of CA125 in the treatment and monitoring of thrombotic complications associated with adenomyosis.

The source of emboli has been investigated in several previous case studies. In a subset of 21 cases with cerebral infarcts, nonbacterial thrombotic endocarditis (NBTE) was detected in 7 cases through transesophageal echocardiography, suggesting the potential presence of cardiogenic emboli. Considering that some cases were reported a decade ago, the postulated incidence of NBTE may be higher under current circumstances due to the use of more sensitive ultrasound technology. To date, there have been 6 documented cases of PE, with 5 originating from a single center in Singapore. Of these, two were complicated by lower extremity DVT, indicating the likelihood of peripheral distal emboli. However, the remaining four cases were diagnosed as isolated pulmonary thrombus, suggesting the possible occurrence of in situ pulmonary thrombus formation due to hypercoagulability. This notion could also explain the manifestation of isolated cerebral vein thrombosis in four women with adenomyosis.[5,16,18]

Isolated DVT associated with adenomyosis is rare, with only three documented cases (including the present case). It is worth noting that two out of these three cases also presented with other thrombotic risk factors in addition to adenomyosis. Specifically, one case had hyperhomocysteinemia, while our case developed DVT after long-distance travel. These findings underscore the necessity for systematic assessment of thrombotic risk and the implementation of thromboprophylaxis education for patients diagnosed with adenomyosis.

The management of thrombotic complications associated with adenomyosis comprises two essential aspects: anticoagulation therapy during the acute phase of thrombophilia and treatment specifically targeting adenomyosis. Consensus regarding the optimal anticoagulation regimens for thrombotic complications related to adenomyosis is lacking. While conventional anticoagulation therapy has demonstrated efficacy in most patients, a significant proportion of individuals experience recurrence within one month. Further evaluation is needed to assess the potential benefits of escalating anticoagulant dosage or prolonging therapy duration. Additionally, the effectiveness of integrating anticoagulation with adenomyosis treatment to control thrombotic exacerbations or recurrences remains uncertain. Although long-term gonadotropin-releasing hormone agonist (GnRHa) therapy has successfully alleviated symptoms in certain cases, there are patients who still encounter thrombosis or experience recurrence despite undergoing GnRHa therapy.[9,14,19] In such cases, hysterectomy is often considered a final option.

4. CONCLUSION

We present a rare case of IDDVT that was potentially attributed to adenomyosis-associated hypercoagulability and travel-related blood stasis. This case study highlights the importance of etiologic investigation and treatment in the management of thrombotic complications. A comprehensive analysis of previously documented cases suggests that CA125 may serve as a promising biomarker for predicting recurrent or systemic thromboembolism in patients with adenomyosis. Additionally, it is noteworthy that the risk window

for thrombotic events in patients with adenomyosis may extend beyond the menstrual period, suggesting a longer duration of susceptibility. Therefore, adenomyosis should be regarded as a potential risk factor for thromboembolism. Aggressive thrombotic risk assessment should be considered as part of clinical practice for patients with this condition, particularly those presenting with severe symptoms and significantly elevated CA125 levels.

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Figures and Figure Legends

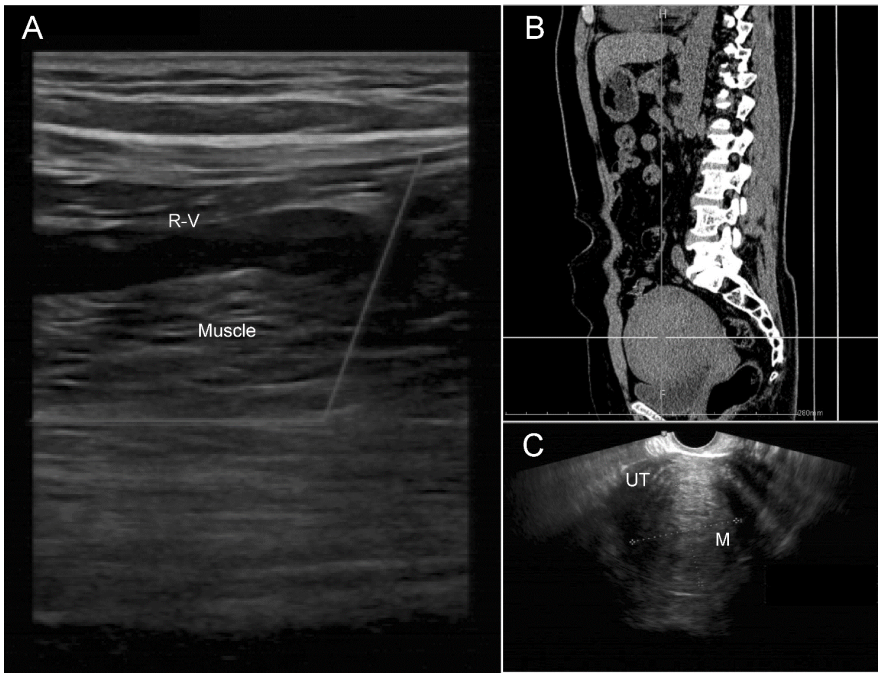

FIGURE 1 Imaging findings of the presented case. (**A**) Thrombosis of the distal intermuscular vein of the right lower extremity. The lumen of the vein exhibits a moderately hypoechoic plaque, while color Doppler flow imaging (CDFI) detects a filling defect within the lumen, along with an absence of a blood flow signal. (**B**) An abdominal CT scan illustrates a substantially enlarged uterus. (**C**) Gynecologic ultrasound confirms the presence of adenomyosis.

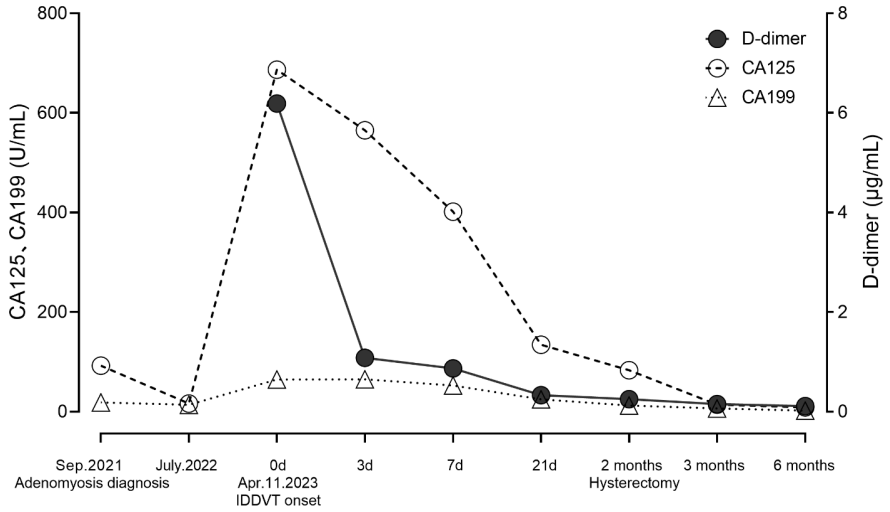

FIGURE 2 Dynamic changes in CA125, CA199 and D-dimer levels for the reported patient.

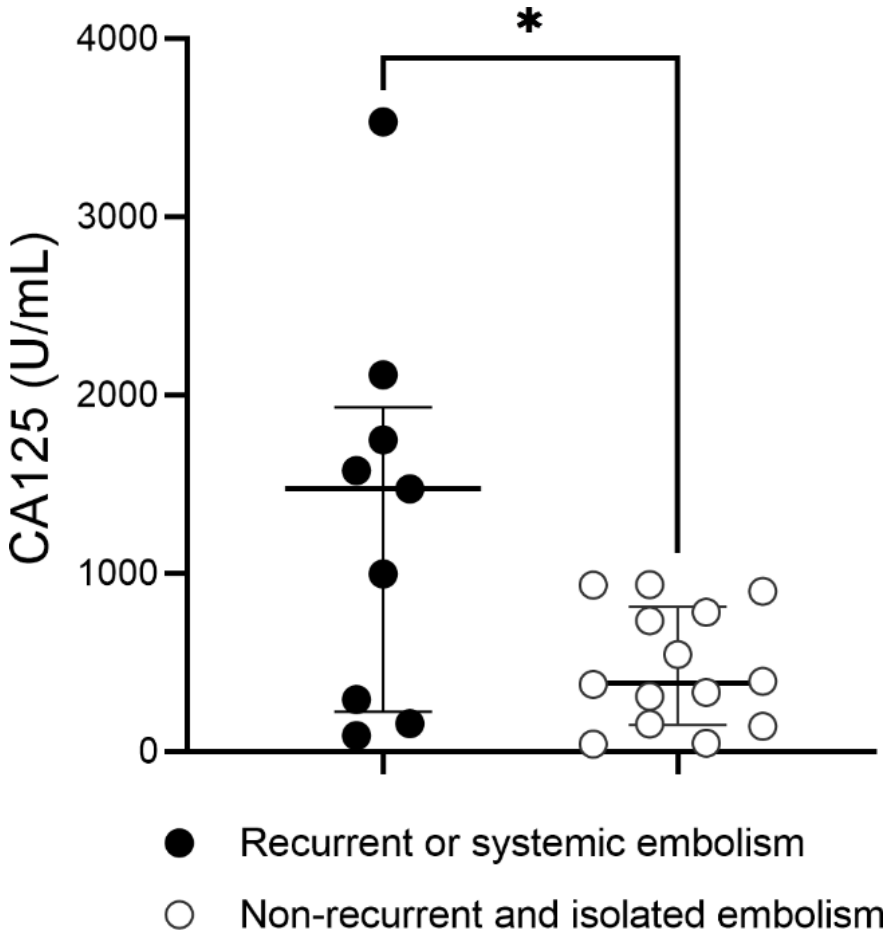

FIGURE 3 Comparison of CA125 levels between patients experiencing recurrent or systemic embolism and those with nonrecurrent and isolated embolism. *: $P = 0.015$, according to the Kolmogorov-Smirnov test.

TABLE 1 Reported cases of adenomyosis-associated stroke, PE and DVT

Ref	Case No.	Age	Onset during menstruation	Diagnosis	Thrombus location	NBTE	Systemic embolism	Recurrence (period)	CA125 (U/mL)	D-dimer (μγ/μΛ)	HGB (g/dL)	T f V (t S c
Ischemic stroke	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**Ischemic stroke**	**I s**
3	1	50	Yes	multiple CIs	Brain, Spleen, Kidney	Yes	Yes	Yes (1y)	1579	95.7	9.8	P - r r s s
4	2	45	No	multiple CIs	Brain, Left fingers	No	Yes	No	159	1.1	8.4	P - A
	3	44	Yes	multiple CIs	Brain, Kidney	No	Yes	No	-	-	7.0	P - V
	4	50	Yes	multiple CIs	Brain	No	No	No	42.6	0.57	6.9	A
	5	42	Yes	multiple CIs	Brain	No	No	Yes (1y)	1750	6.0	8.6	P - V
5	6	47	-	CVT	Brain	No	No	No	784.6	6.3	7.6	P - V f - A
6	7	59	-	multiple CIs	Brain	Yes	No	No	334.8	7.0	-	P
7	8	49	No	multiple CIs	Brain	Yes	No	No	379	3.99	9.9	P - V
8	9	48	No	multiple CIs	Brain	Yes	No	No	901	1.9	8.5	P - V
9	10	44	Yes	multiple CIs	Brain, Spleen	No	Yes	Yes (6m)	2115	17.0	7.3	P - P V
10	11	42	-	single CI	Brain	No	No	No	395	1.4	-	V
	12	50	-	single CI	Brain	No	No	No	143	3.7	-	P

Ref	Case No.	Age	Onset during menstruation	Diagnosis	Thrombus location	NBTE	Systemic embolism	Recurrence (period)	CA125 (U/mL)	D-dimer (μγ/μΛ)	HGB (g/dL)	T f V (t S c
11	13	34	Yes	multiple CIs	Brain	No	No	No	937.1	1.05	13.4	-
	14	37	Yes	single CI	Brain	No	No	No	735.7	23.4	10.8	-
	15	46	Yes	multiple CIs	Brain	No	No	No	546.5	12.04	12.1	-
12	16	34	Yes	multiple CIs	Brain	No	No	No	937.7	27.4	11.2	P - A
13	17	48	Yes	multiple CIs	Brain, Kidney	No	Yes	No	3536	79.3	8.2	T - P a - P
14	18	50	Yes	multiple CIs	Brain	No	No	Yes (7d)	999	6.4	9.2	P a
15	19	46	Yes	multiple CIs + PE + VTE	Brain, Lung, right lower limb	No	Yes	No	1477	7.4	9.6	P - t - r
16	20	42	Yes	CVT	Brain	No	No	No	155	2.5	8.4	P d

TABLE 1 continued

Ref	Case No.	Age	Onset during menstruation	Diagnosis	Thrombus location	NBTE	Systemic embolism	Recurrence (period)	CA125 (U/mL)	D-dimer (μγ/μΛ)	HGB (g/dL)	T f V (t S c
17	21	47	Yes	multiple CIs	Brain, Kidney	No	Yes	Yes (1m)	90.3	3.8	11.3	h - P
18	22	38	Yes	CVT	Brain	-	No	Yes (10d)	-	3.9	7.5	P - V

Ref	Case No.	Age	Onset during menstruation	Diagnosis	Thrombus location	NBTE	Systemic embolism	Recurrence (period)	CA125 (U/mL)	D-dimer (μγ/μΛ)	HGB (g/dL)	T f V (t S c
	23	34	-	CVT	Brain	-	No	No	312.4	18.63	7.6	P - V
19	24	42	Yes	multiple CIs	Brain	-	No	Yes (1m)	293	-	-	P - P
20	25	47	Yes	multiple CIs	Brain	Yes	No	No	48	30	3.4	V
Pulmonary embolism	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**Pulmonary embolism**	**P e b**
21	1	38	-	PE	Lung	-	No	No	-	-	-	T a a c u t
22	2	44	-	PE + DVT	Lung, left lower limb	-	Yes	No	-	-	7.3	V
	3	38	-	PE	Lung	-	No	No	-	-	4.5	P
	4	50	-	PE	Lung	-	No	No	-	-	6.2	V
	5	45	-	PE	Lung	-	No	No	-	-	11.4	V
	6	51	-	PE + DVT	Lung, left lower limb	-	Yes	No	-	-	7.9	T t a v f I f
Deep vein thrombosis	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**Deep vein thrombosis**	**I v t b s**
23	1	37	-	DVT	left femoral vein	-	No	Yes (10y)	-	-	-	C V

Ref	Case No.	Age	Onset during menstruation	Diagnosis	Thrombus location	NBTE	Systemic embolism	Recurrence (period)	CA125 (U/mL)	D-dimer (μγ/μΛ)	HGB (g/dL)	T f V (t S c
24	2	34	-	DVT	left lower limb	-	No	No	-	-	-	-
Our case	3	50	Yes	DVT	right lower limb	-	No	No	687	6.19	11.1	F - F

Note: -: data not reported. CIs: cerebral infarcts; CVT: cerebral venous thrombosis; DVT: deep vein thrombosis; GnRHa: gonadotropin-releasing hormone agonist; IVC: inferior vena cava; NBTE: nonbacterial thrombotic endocarditis; VKA: vitamin K antagonist.