BRITISH JOURNAL OF HOSPITAL MEDICINE

CASE REPORT

Low-Dose Dabigatran for Venous Sinus Thromboembolism Associated with Hereditary Dysfibrinogenemia: A Case Report

Ying Liu[1], Jinlong Zhao[2], Jing Guo[1], Yu Liu[1], Chenlu Ma[1], Yan Zhang[3,*]

[1]The Second Department of Internal Medicine, The First People's Hospital of Urumqi, Urumqi, Xinjiang Uygur Autonomous Region, China
[2]The Department of Imaging, The First People's Hospital of Urumqi, Urumqi, Xinjiang Uygur Autonomous Region, China
[3]The Department of Inspection, The First People's Hospital of Urumqi, Urumqi, Xinjiang Uygur Autonomous Region, China
*Correspondence: 18999858692@163.com (Yan Zhang)

Abstract

Dabigatran, an anticoagulant, may increase the risk of bleeding in patients with dysfibrinogenemia because of coagulation irregularities, especially at high doses. Cranial Magnetic Resonance Imaging (MRI) and Magnetic Resonance Venography (MRV) were used in the diagnosis of venous sinus thromboembolism in a 42-year-old woman with hereditary dysfibrinogenemia, as documented in our case report. Cranial MRI suggested thrombosis in the venous sinuses, which was confirmed by MRV as thromboses in the superior sagittal, straight, left transverse, and sigmoid sinuses. Instead of the usual fixed-dose, we gave the patient dabigatran based on how the coagulation indicators changed. Forty-six days after treatment, the patient's clinical symptoms had largely resolved. Follow-up cranial MR showed that most of the venous sinus thromboses had disappeared, with some mural thrombi still present in the superior sagittal sinus and left sigmoid sinus. In this report, we optimized the dabigatran regimen adjusted to thrombin time, ensuring efficacy with low bleeding risk.

Key words: hereditary dysfibrinogenemia; venous sinus thromboembolism; dabigatran; anticoagulant drugs; thrombin time

Submitted: 12 July 2024 Revised: 5 September 2024 Accepted: 10 September 2024

Introduction

Fibrinogens play a key role in hemostasis by working with platelet aggregation as substrates for fibrin conversion and functioning as scaffolds for fibrinolysis and wound healing (Kearney et al, 2022). Fibrinogen deficiency can be divided into two types. Class I shows abnormalities in how much fibrinogen, including hypofibrinogenemia and non-fibrinogenaemia, and class II shows abnormalities in the quality of fibrinogen, including fibrinogen abnormalities and hypofibrinogenemia (Casini et al, 2022). Congenital dysfibrinogenemia (CD) is a rare hereditary blood disease caused by defects in the genes encoding fibrinogen (Fg) (*FGA*, *FGB* and *FGG*) that can clinically appear as spontaneous bleeding or thrombosis (Simurda et al, 2020; Yan et al, 2022). CD is a class II fibrinogen disease with an autosomal dominant pattern of inheritance. Just a single parent carrying a defective gene in

How to cite this article:
Liu Y, Zhao J, Guo J, Liu Y, Ma C, Zhang Y. Low-Dose Dabigatran for Venous Sinus Thromboembolism Associated with Hereditary Dysfibrinogenemia: A Case Report. Br J Hosp Med. 2025. https://doi.org/10.12968/hmed.2024.0426

the offspring can cause the disease, and the incidence of CD is higher than that of hereditary non-fibrinogenaemia (Chen et al, 2022). The clinical prevalence of CD is approximately 0.3% in Finland and 1% in the African American United States (Paraboschi et al, 2017). The clinical signs and symptoms of CD are highly variable. Only 20% developed thrombotic diseases, while 25% of patients had bleeding symptoms (Haverkate and Samama, 1995). Therefore, approximately half of the patients with CD are found incidentally (Castaman et al, 2019; Wypasek et al, 2019). Patients with CD are at high risk for major hemorrhage (including postpartum hemorrhage) and thrombotic events during a mean follow-up of 8.8 years after their diagnosis (Casini et al, 2015). Patients with Fg variants associated with thrombosis are particularly at risk and may benefit from long-term anticoagulation therapy (Casini and de Moerloose, 2021).

In this study, we reported a patient with *FGG* gene missense mutation (c.1007T >Ap.M336K). During anticoagulant therapy, the anticoagulant drug regimen was changed by the dynamic changes in thrombin time (TT) of the patient, and satisfactory efficacy was still achieved in the presence of low-dose anticoagulants, avoiding the risk of hemorrhage. This will help guide the treatment of such CD patients in the future and provide a basis for further elucidation of the relationship between genotypes and clinical manifestations.

Case Report

Chief Complaints

The patient was a 42-year-old previously healthy woman from China on 20 March 2023, who suffered from fatigue, dizziness, and headache for 3 days, accompanied by poor appetite, intermittent nausea, and vomiting.

History of Past Illness

The patient denied any history of hypertension, type 2 diabetes, cerebral hemorrhage, or traumatic brain injury. There was no reported history of infectious diseases such as hepatitis or tuberculosis, significant trauma, surgery, or blood transfusions. The patient also reported no history of allergies to food or medication.

Physical Examination

The range of motion in the cervical vertebrae was within acceptable limits. Eyeball movement, light reflex, pure tone thresholds for both ears, and muscle strength and tension in the limbs were all normal. The Dix-Hallpike test, calcaneal-knee-tibia test, Romberg test, bilateral finger-nose test, and bilateral Hoffmann sign were all negative. Physiological reflexes were present, while pathological reflexes were not observed.

Laboratory Examinations

After admission, the patient performed a laboratory test. Complete blood count and four coagulation tests (considering hemoconcentration) were all normal. Biochemical tests were negative, including blood homocysteine, anticardiolipin antibody and antinuclear antibody (Table 1).

Table 1. Patient laboratory test results.

Inspection items	WBC	RBC	HGB	PLT	PT	APTT
Result	4.74×10^9/L	3.85×10^{12}/L	128 g/L	214×10^9/L	11.74 s	29 s
Inspection items	TT	FIB	HCY	APL	ANA	
Result	17 s	2.2 g/L	11 μmol/L	(-)	(-)	

Abbreviations: WBC, white blood cell count; RBC, red blood cell count; HGB, hemoglobin; PLT, platelet count; PT, prothrombin time; APTT, activated partial thromboplastin time; TT, thrombin time; FIB, fibrinogen; HCY, homocysteine; APL, antiphospholipid antibodies; ANA, antinuclear antibodies.

Table 2. Examination results of coagulation indicators of patients and their family members.

Family members	Coagulation indicators				
	PT (s)	INR	APTT (s)	TT (s)	FIB (g/L)
Father	12	1.04	28.6	**17.5**	2.47
Mother	**14.8**	**1.29**	29.3	**19.8**	**0.84**
Proband	12.8	1.11	26.8	**19.3**	**0.94**
Brother	11.9	1.03	32.2	**19.2**	2.13
Son	12.2	1.06	30.8	**19**	3.15

Abbreviations: INR, international normalized ratio. Note: the outliers were bolded.

Family Medical History

The patient complained that she had no family medical history of genetic diseases. During the visit, the patient's mother complained that she had a history of hypofibrinogenemia at her previous visits. With their consent for whole-exome sequencing, the patient's parents, brother and son underwent four coagulation tests. Her mother was diagnosed with hypofibrinogenemia, while others showed no abnormalities (see Table 2).

Imaging Examinations

No obvious abnormalities were found on brain computed tomography (CT). The symptoms persisted and were not relieved 6 days after admission with blurred vision. Cranial Magnetic Resonance (MR) was performed on 27 March 2023, and suggested thromboses in the superior sagittal sinus, straight sinus, left transverse sinus, and sigmoid sinus, indicating venous sinus thrombosis (Fig. 1).

Gene Examination

Four coagulation tests suggested abnormal fibrinogen levels. After that, the fibrinogen level was still lower than normal by several times of Clauss method, whereas the results of prothrombin time (PT) algorithm showed a normal fibrinogen level. Based on Sanger sequencing, the genetic test results of the patient suggested a hereditary dysfibrinogenemia *FGG* gene c.1007T>Ap.M336K missense mutation (Table 3), and the related diseases were congenital hypofibrinogenemia

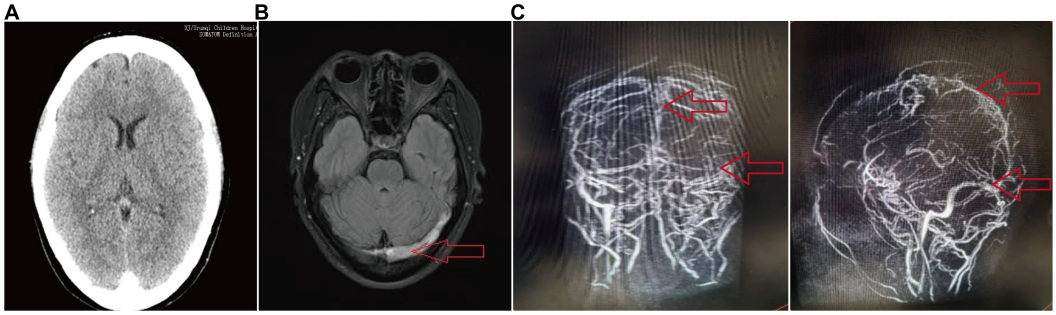

Fig. 1. Computed Tomography (CT) and Magnetic Resonance (MR) examination results of patient on 27 March 2023. (A) CT examination. (B) Magnetic Resonance Imaging (MRI) examination. Flux line-segment model for advection and interface reconstruction (FLAIR) sequence showed the abnormally high signal intensity of the sigmoid sinus. The area indicated by the red arrow is the sigmoid sinus area. (C) Magnetic Resonance Venography (MRV) examination. The superior sagittal sinus, transverse sinus and left sigmoid sinus of MRV did not show abnormally high signal intensity. The areas indicated by the red arrow were transverse sinus and left sigmoid sinus.

and congenital fibrinogenemia. To determine whether the patient's fibrinogenemia was a hereditary disease, we screened the patient's family members, and created a genealogy chart (Fig. 2). Finally, the patient was diagnosed with CD.

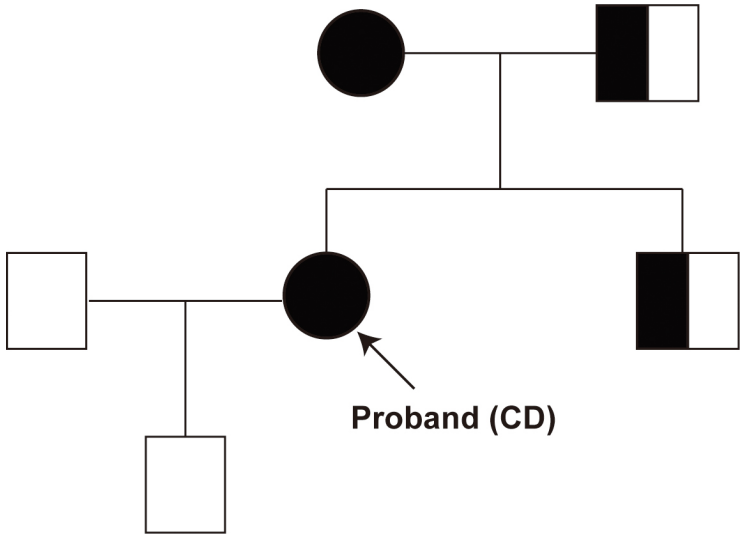

Fig. 2. Familial genetic pedigree of the patient with congenital dysfibrinogenemia (CD). Black represents carrying disease-causing genes, white represents normal gene, squares represent male, and circles represent female.

Treatment Method

Once daily, subcutaneously administered low-molecular-weight heparin sodium (0.4 mL), 20% mannitol (125 mL) was used for dehydration treatment every 12 h to lower intracranial pressure, and coagulation function was monitored. On 23 April 2023, the reexamination showed that the patient's thrombin time (TT) was 91.6 s, and dabigatran was adjusted to 110 mg orally one time each day. When TT was

Table 3. Genetic test results of the proband.

Mutated genes	Chromosome position	Transcript ID	Type of mutation	Nucleotide alteration	Amino acid changes	Mode of inheritance
FGG	Chr4:155527979	NM 021870.3	Missense mutation	c.1007T>A	p.M336K	Autosomal recessive inheritance

Table 4. Coagulation indexes and oral dosage of anticoagulants in different periods.

Inspection date	PT (s)	APTT (s)	TT (s)	FIB (g/L)		Dosage and administration of anticoagulants
				Clauss	PT-algorithm	
2023.3.31	11.2	22.2	32.9	1.01	3.27	Low molecular weight heparin sodium 0.4 mL once a day
2023.4.9	11.5	27.6	19.4	0.8	2.08	110 mg of dabigatran capsules twice a day
2023.4.23	12	33.6	91.6	1.14	2.12	110 mg of dabigatran capsules once a day
2023.5.11	12.8	36.6	114.5	1.03	1.93	110 mg of dabigatran capsules every other day
2023.5.26	11.8	29.9	76.4	1.47	2.32	110 mg of dabigatran capsules every other day

delayed more than 3 times, the dose was reduced to 110 mg orally every other day on 11 May 2023. The patient returned to our department for a second examination 46 days later, claiming that the clinical symptoms had vanished (Table 4).

Outcome and Follow-up

Following 10 days of treatment, the patient's clinical side effects, such as headache and blurred vision, significantly improved, and she was discharged. After discharge, considering that the patient was still at risk of bleeding, the anticoagulant drug dabigatran capsules were given orally at an initial dose of 110 mg two times every day. Coagulation function of the patient was monitored during medication. On 12 May 2023, brain MR was reconsidered, suggesting that most venous sinus thrombosis had disappeared, and some mural thrombi were visible in the superior sagittal sinus and left sigmoid sinus (Fig. 3).

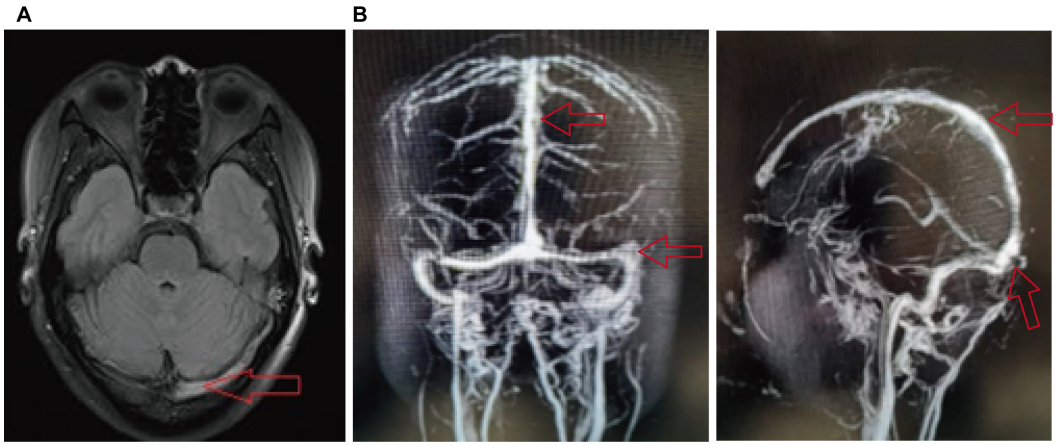

Fig. 3. MR examination results of the patient on 12 May 2023. (A) MRI examination. FLAIR sequence shows abnormally high signal intensity of the sigmoid sinus, and its volume is smaller than that on March 27. The area indicated by the red arrow in the figure is the sigmoid sinus region. (B) MRV examination. Most of the superior sagittal, transverse, and sigmoid sinuses showed signal intensity. The areas indicated by the red arrow were transverse sinus and left sigmoid sinus.

Discussion

Due to the extremely secretive etiology of CD, it is often misdiagnosed in clinical practice. Chen et al (2022) reported a case of a 23-year-old patient with CD who was misdiagnosed with hypofibrinogenemia. The patient was ultimately diagnosed with CD following genetic sequencing and inquiry into the family's past. Therefore, improving the condition of hypofibrinogenemic patients in clinical practice necessitates a precise diagnosis. In this study, the patient presented with dizziness and headache, and routine coagulation test after admission did not show any abnormalities. Later, the Clauss method was used to re-examine and found that the patient's fibrinogen was beneath the typical level. Combined with an imaging examination, the patient was initially diagnosed with hypofibrinogenemia. After completing the patient's genetic testing and investigating family members, the final diagnosis was

CD. Significant clinical efficacy was achieved using low-dose dabigatran capsules based on the patient's coagulation indicators.

Given the critical role of fibrinogen in both procoagulant and fibrinolytic pathways, patients with CD may develop both hemorrhagic and thrombotic disorders (Bor, 2024). The disease in our patient was venous sinus thromboembolism. A study has shown that the annual incidence of arterial or venous thromboembolism in patients with CD is 13.9/1000 (Casini et al, 2015). The exact mechanisms by which abnormal fibrinogen increases the risk of thrombosis are unknown (Weisel and Litvinov, 2013), mainly including increased levels of thrombin due to deficiencies in binding fibrinogen (Hulshof et al, 2021); and altered strength, structure, and stability of fibrin clots (Martinez-Vargas et al, 2023); and impaired fibrinolysis of abnormal fibrinogen (Kanji et al, 2021). The pathophysiology of venous thromboembolism (VTE) is often described as the intersection of three major abnormalities (venous stasis, vascular dysfunction/injury, and hypercoagulable state) known as the Virchow triad. In this conceptual model, the formation of a hypoxic environment due to reduced (stagnant) or turbulent (nonlaminar) blood flow around the venous valve capsule activates endothelial cells and results in abnormal expression of adhesion molecules that bind to and remain on the endothelial surface (Gonzalez-Gonzalez et al, 2021). Activated leukocytes and possibly dysfunctional endothelial cells express tissue factors that trigger the coagulation cascade. White blood cells, red blood cells, and platelets that accumulate in the valve pocket promote thrombin production and eventually form a thrombus rich in red blood cells and fibrin (Wolberg and Sang, 2022).

The recommended practice for the prevention of recurrence in patients with intracranial venous thrombosis is to use vitamin K antagonists for various periods of anticoagulation, depending on each patient's inherent risk of thrombosis (Pfeilschifter et al, 2022; Yaghi et al, 2022a). Effective treatment of venous thromboembolism depends on a balance between the prevention of recurrence and the incidence of bleeding complications (de Winter et al, 2023). In general, clinical guidelines for the treatment of VTE recommend subcutaneous injections of low-molecular-weight heparin and fondaparinux (Maughan et al, 2022; Schrag et al, 2023), followed by the addition of a vitamin K antagonist (Bhat et al, 2024). Both low-molecular-weight heparins and vitamin K antagonists (e.g., warfarin, ethyl methaminol, or phenprocoumarin) have a (potentially fatal) risk of bleeding (Brennan et al, 2019; Yang et al, 2020). Compared with vitamin K antagonists, direct thrombin inhibitors have a rapid onset of action, a fixed dose, no known food effect, few drug interactions, no need for routine monitoring of fixed doses, and a short offset period (Sun et al, 2020). Dabigatran, a direct thrombin inhibitor, is currently approved for the treatment and prevention of venous thromboembolism in the European Union and the United States. A multicenter study demonstrated that direct oral anticoagulation with dabigatran for cerebral venous thrombosis had similar clinical and radiographic outcomes and a favorable safety profile compared to warfarin (Yaghi et al, 2022b). In this case report, the oral dosage of dabigatran was adjusted according to the patient's coagulation indicators. At present, she is given 110 mg orally every other day as anticoagulant therapy. After re-examination, her

coagulation indicators were within the normal range, and she had no obvious symptoms of discomfort, with satisfactory clinical results.

Conclusion

To determine whether patients have fibrinogen abnormalities, an appropriate approach should be selected to examine the patient's fibrinogen activator and fibrinogen antigen-related indicators along with genetic screening to further determine whether it is hereditary dysfibrinogenemia associated with venous sinus thromboembolism while excluding the causes of non-genetic diseases such as liver and tumour. Furthermore, it is imperative to comprehend the patient's family medical history in order to gain insight into the disease status of other family members. For patients diagnosed with CD, the reasonable use of low-dose dabigatran for anticoagulant therapy according to the patient's coagulation-related indicators can also achieve good clinical efficacy to reduce the risk of bleeding in patients, which is worthy of promotion in clinical practice.

Learning Points

- This case study focuses on the management challenges associated with dabigatran therapy in a patient with hereditary dysfibrinogenemia and venous sinus thrombosis.
- This case study highlights initial concerns about bleeding risks due to coagulation abnormalities associated with dysfibrinogenemia.
- The patient showed significant improvement with a treatment regimen adjusted based on the dynamic changes in thrombin time.
- Genetic analysis identified a specific mutation in *FGG* linked to hereditary dysfibrinogenemia, emphasizing the genetic basis of the condition in this patient.
- This case study underscores the importance of personalized anticoagulant therapy in complex cases involving inherited coagulation disorders to optimize treatment outcomes while minimizing bleeding risk.

Availability of Data and Materials

The datasets used and analysed during the current study are available from the corresponding author on reasonable request.

Author Contributions

Programme administration: YingL. Conceptualisation: YZ. Data collection and analysis: YingL and JZ. Investigation: JG, YuL and CM. Writing—original: YingL. Writing—review editing: all authors. All authors contributed to important editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was approved by the ethics committee of The First People's Hospital of Urumqi (approval No. KY-016) and performed in accordance with the Declaration of Helsinki. The participant signed an informed consent form.

Acknowledgement

The authors express their appreciation to the staff at The First People's Hospital of Urumqi, for their technical assistance.

Funding

This research received no external funding.

Conflict of Interest

The authors declare no conflict of interest.

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