Case Report

Bilateral Deep Vein Thrombosis in an Adolescent with Congenital Rubella Syndrome: A Case Report

Kellyn Trycia Zenjaya [1]*, Lindawati Wijoyo [1], Santika Danubrata [1], Hendrata Erry Andisari [2], Febryanti Hartono [3]

[1] Faculty of Medicine, Hang Tuah University, Surabaya, Indonesia.
[2] Department of Internal Medicine, Dr Ramelan Naval Centre Hospital, Surabaya, Indonesia.
[3] Department of Cardiology and Vascular Medicine, Dr Ramelan Naval Centre Hospital, Surabaya, Indonesia.

Citation: Trycia Zenjaya K, Wijoyo L, Danubrata S, Erry Andisari H, Hartono F. Bilateral Deep Vein Thrombosis in an Adolescent with Congenital Rubella Syndrome: A Case Report. Res Heart Yield Transl Med 2026; 21(2):174-179.

https://doi.org/10.18502/jthc.v21i2.21698

Highlights

- Bilateral deep vein thrombosis is rare in adolescents and represents an uncommon presentation of venous thromboembolism.
- This case describes bilateral deep vein thrombosis in a patient with congenital rubella syndrome and chronic cardiovascular complications.
- Persistent bilateral lower limb edema in such patients should prompt consideration of deep vein thrombosis.

Article info:

Received: 16 Dec. 2025
Revised: 4 Jan. 2026
Accepted: 1 Feb. 2026

*** Corresponding Author:**
Kellyn Trycia Zenjaya
Faculty of Medicine, Hang Tuah, University, Surabaya, Indonesia.
Email: kellyntryciazenjaya@gmail.com

ABSTRACT

Bilateral deep vein thrombosis (DVT) is a rare condition among adolescents and represents an uncommon pattern of venous thromboembolism. We report a case of bilateral DVT occurring in the setting of severe pulmonary hypertension and right-sided heart failure secondary to congenital heart disease associated with congenital rubella syndrome (CRS). A 19-year-old female patient, with a history of CRS characterized by the classical triad of unilateral cataract, sensorineural deafness, and congenital heart disease, was admitted to our hospital with shortness of breath, swelling in both legs, and abdominal discomfort. Doppler ultrasonography demonstrated partial thrombotic obstruction of the femoral, popliteal, and posterior tibial veins in both lower extremities. A diagnosis of DVT was established on the fourth day of hospitalization. This case highlights the importance of considering bilateral DVT in patients with CRS who present with lower extremity edema, particularly in the presence of cardiovascular complications.

Keywords: Bilateral Deep Vein Thrombosis; Congenital Rubella Syndrome; Thromboembolism

Copyright © 2026 Tehran University of Medical Sciences. Published by Tehran University of Medical Sciences.
This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International license (https://creativecommons.org/licenses/by-nc/4.0/). Noncommercial uses of the work are permitted, provided the original work is properly cited

RHYTHM
Research in Heart Yield and Translational Medicine

Introduction

Deep vein thrombosis (DVT) is rare in adolescents, with an incidence of 1 per 10,000 compared to 1 per 1,000 in the general population.[1] Bilateral DVT refers to thrombus formation in both lower extremities and is associated with a higher thrombotic burden. Heart failure (HF) increases susceptibility to venous thromboembolism (VTE) and its complications, including pulmonary embolism and right ventricular failure.[2, 3]

We describe a 19-year-old female with congenital rubella syndrome (CRS) who developed bilateral DVT in the setting of long-standing cardiovascular complications. She had a history of uncorrected patent ductus arteriosus (PDA) and was found to have pulmonary hypertension and right-sided HF on echocardiography.[4]

CRS, resulting from maternal rubella infection, is associated with congenital anomalies and may lead to persistent cardiovascular abnormalities extending into adolescence and adulthood.[5, 6] These long-term cardiovascular sequelae may create hemodynamic conditions that predispose affected individuals to VTE.[7]

Case Presentation

On January 17, 2024, a 19-year-old female was admitted to our hospital with progressive shortness of breath, abdominal discomfort (bloating, epigastric pain, and nausea), and a history of bilateral lower leg swelling. She had been hospitalized for 5 days at another facility several days earlier for similar cardiopulmonary and abdominal symptoms; however, lower limb swelling developed 1 day prior to transfer.

The patient had been diagnosed with CRS at 7 months of age, characterized by the classical triad of right-eye cataract, sensorineural deafness, and PDA. Surgical closure of the PDA had been recommended but declined by her parents. She had no prior history of VTE, prolonged hospitalization, or recent medication use. Prior to the current illness, she was physically active.

On admission, the patient appeared moderately ill but was fully alert. Vital signs showed blood pressure of 142/107 mm Hg, heart rate of 111 beats/min, respiratory rate of 26 breaths/min, and oxygen saturation of 95% on room air. Cardiac examination revealed systolic and diastolic murmurs best heard at the pulmonary area. Bilateral lung auscultation revealed rhonchi. Abdominal examination demonstrated distension with epigastric tenderness. At initial evaluation, no clinically significant lower limb edema was observed.

Laboratory tests revealed leukocytosis, neutrophilia, lymphopenia (14.12×10³/uL, 10.61×10³/uL, 18.5%), elevated SGOT/SGPT (938/1141 U/L), hypoalbuminemia (3.37 mg/dL), and a markedly elevated D-dimer (9390 ng/dL). Electrocardiography showed sinus tachycardia, right axis deviation, suspected right ventricular hypertrophy, and nonspecific ST-T changes. Chest X-ray revealed cardiomegaly with features suggestive of pulmonary hypertension (Figure 1). Abdominal computed tomography and ultrasonography revealed minimal ascites, pleural effusion, and findings consistent with congestive hepatopathy.

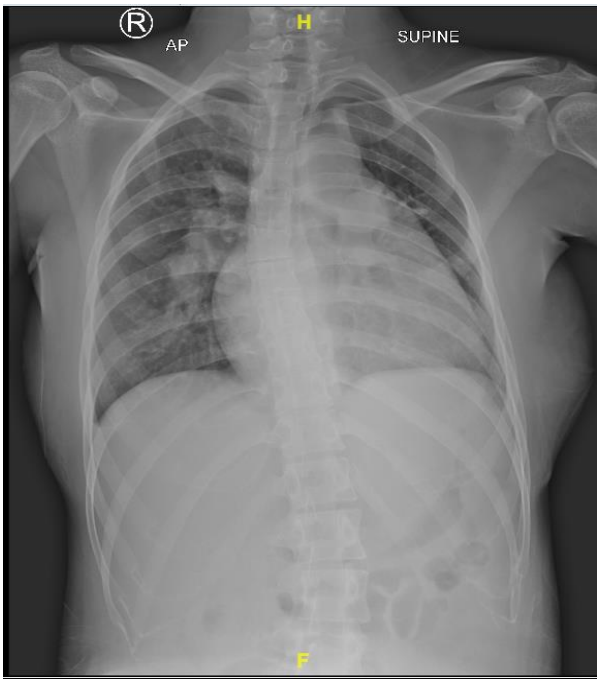


Figure 1. Anteroposterior (AP) chest X-ray of the patient

Transthoracic echocardiography revealed severe tricuspid regurgitation (maximum pressure gradient: 148.68 mm Hg), severe pulmonary regurgitation (pressure half-time: 389 ms), pulmonary artery dilatation, marked right atrial and ventricular enlargement, reduced right ventricular systolic function, mild pericardial effusion, and no

evidence of residual PDA (Figure 2).

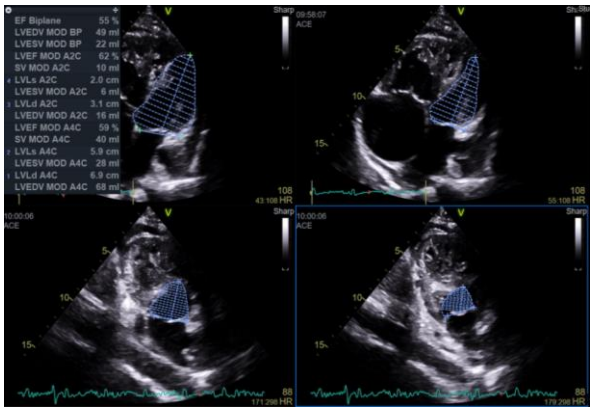


Figure 2. Transthoracic echocardiography of the patient

The patient was treated with intravenous furosemide, oral spironolactone, and sildenafil for pulmonary hypertension, along with supportive therapy for hepatic dysfunction. Serial liver function tests showed progressive improvement (day 3: SGOT/SGPT: 366/723 U/L and day 9: 22/131 U/L). Despite improvement in cardiopulmonary symptoms, bilateral lower extremity swelling persisted and became more apparent during hospitalization. On day 4, Doppler ultrasonography of the lower limbs was performed and demonstrated bilateral DVT with partial thrombotic obstruction of the femoral, popliteal, and posterior tibial veins (Figure 3 and 4). Therefore, a diagnosis of bilateral DVT was established.

The diagnosis of DVT was made on the fourth day of hospitalization. At admission, the patient's clinical presentation and laboratory abnormalities were predominantly attributed to acute right-sided HF and congestive hepatopathy, and no clinically significant lower limb edema was observed. Although the D-dimer level was markedly elevated, this finding was initially interpreted in the context of severe HF and systemic congestion. Persistent and progressive bilateral lower extremity swelling during hospitalization prompted further vascular evaluation, leading to the confirmation of bilateral DVT by Doppler ultrasonography.

Given the presence of bilateral DVT, severe pulmonary hypertension, and right-sided HF, pulmonary embolism was carefully considered. Nevertheless, pulmonary embolism was deemed unlikely based on the patient's clinical status, as there was no acute deterioration in oxygenation, no hemodynamic instability, and no sudden clinical worsening suggestive of an acute embolic event. Accordingly, further imaging, such as computed tomography pulmonary angiography, was not pursued.

Screening for inherited or acquired thrombophilia, including protein C deficiency, protein S deficiency, antithrombin deficiency, and antiphospholipid antibodies, was not performed in this patient. Given the patient's young age and the presence of bilateral DVT, this represents a limitation of the case report, as an underlying hypercoagulable state cannot be definitively excluded. Nonetheless, the strong presence of persistent hemodynamic risk factors provides a plausible explanation for thrombosis in this case.

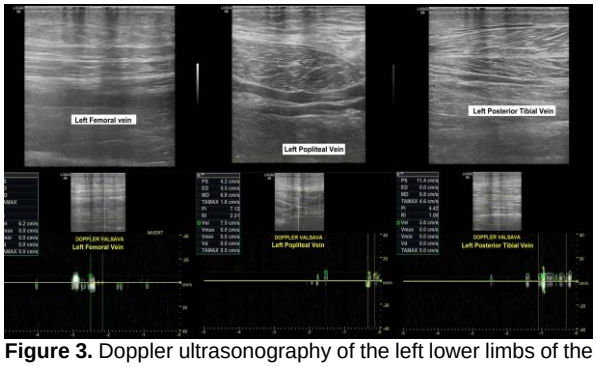


Figure 3. Doppler ultrasonography of the left lower limbs of the patient

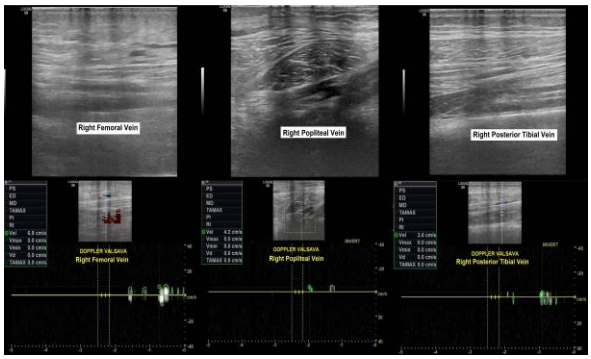


Figure 4. Doppler ultrasonography of the right lower limbs of the patient

Compression therapy with limb elevation was initiated, and anticoagulation therapy was started. Patient received warfarin (1 mg once daily) following diagnosis, with a plan for regular outpatient monitoring of the international normalized ratio (INR) and dose adjustment. The patient showed gradual clinical improvement, with a reduction in lower-limb swelling and symptom relief. She was discharged on the ninth hospital day with outpatient follow-up and regular monitoring. A chronological summary of the patient's symptoms, investigation, and diagnosis is provided in (Table 1).

RHYTHM
Research in Heart Yield and Translational Medicine

Table 1. Timeline of clinical events, investigations, and diagnosis

Time Point	Clinical Events
Infancy (7 months old)	The patient was diagnosed with congenital rubella syndrome, characterized by right-eye cataract, sensorineural deafness, and patent ductus arteriosus. Surgical closure of the patent ductus arteriosus was recommended but not performed.
Prior to current illness	The patient was physically active with no history of venous thromboembolism, prolonged immobilization, or anticoagulant use.
Day 0	The patient was admitted with progressive shortness of breath, abdominal discomfort and a history of bilateral lower leg swelling. Initial evaluation suggested right-sided heart failure and hepatic congestion.
Day 1–2	Laboratory tests revealed markedly elevated liver enzymes and D-dimer. Chest X-ray showed cardiomegaly and pulmonary hypertension. Supportive therapy and diuretics were initiated.
Day 4	Persistent and progressive bilateral lower limb edema prompted Doppler ultrasonography, which demonstrated bilateral deep vein thrombosis involving the femoral, popliteal, and posterior tibial veins. Additionally, there were progressive shortness of breath, abdominal discomfort (bloating, epigastric pain, and nausea), and a history of bilateral lower leg swelling.
Day 5–8	The patient exhibited gradual clinical improvement, including reduction of lower extremity swelling and improvement in symptoms.
Day 9	The patient was discharged in stable condition with outpatient follow-up and regular monitoring.

Discussion

Bilateral DVT is an uncommon presentation of VTE, particularly in adolescents, and is generally associated with a higher thrombotic burden and an increased risk of pulmonary embolism.[1, 2] In young patients, the occurrence of bilateral DVT more often reflects the coexistence of multiple predisposing factors rather than a single etiologic condition.[3] CRS is one of the most severe congenital illnesses caused by the rubella virus that leads to fetal malformation syndrome.[8] This case illustrates the development of bilateral DVT in an adolescent with CRS and chronic cardiovascular sequelae.

The pathogenesis of DVT is classically explained by the Virchow triad, which includes venous stasis, hypercoagulability, and endothelial dysfunction.[3, 4] In this patient, classical risk factors such as prolonged immobilization or major hospitalization were minimal, and her Wells score would likely have been low.[4] Nevertheless, imaging confirmed bilateral DVT, prompting the question of underlying predisposing mechanisms beyond typical risk factors. Importantly, CRS itself is unlikely to represent a direct prothrombotic condition. Instead, CRS should be regarded as an indirect risk factor for thrombosis through its long-term cardiovascular consequences.

The patient had a history of uncorrected PDA and, at presentation, demonstrated severe pulmonary hypertension and right-sided HF. Chronic cardiovascular sequelae from CRS, including right-sided HF and pulmonary hypertension, likely created a hemodynamic and prothrombotic environment that contributed to thrombus formation. Right-sided HF can increase venous pressures, impair venous return, and cause systemic congestion, all of which promote venous stasis. Pulmonary hypertension further elevates right heart pressures, exacerbating systemic venous congestion.[5–7] These hemodynamic disturbances are compounded by endothelial dysfunction, neurohormonal activation, and inflammation, collectively creating a milieu favoring thrombogenesis, rather than CRS acting through an intrinsic hypercoagulable state.[6, 8]

Congenital heart disease, especially when uncorrected or complicated by pulmonary hypertension, has also been associated with coagulation abnormalities and thromboembolic events. Disturbances in platelet function, plasma coagulation factors, blood viscosity, and endothelial integrity may further disrupt normal hemostasis.[9, 10] In this patient, the combination of chronic right-sided HF, pulmonary hypertension, and congenital heart disease likely contributed to the development of bilateral DVT, even in the absence of classic transient risk factors.

CRS is primarily recognized for its classical congenital manifestations, including sensorineural hearing loss, ocular abnormalities, and structural

heart disease.[11, 12] Although delayed or late-onset manifestations, including endocrine, neurologic, and vascular abnormalities, have been described, a direct causal relationship between CRS and DVT has not been established.[13–15] In this context, CRS should be considered the underlying condition that resulted in uncorrected congenital heart disease and subsequent severe pulmonary hypertension and right-sided HF. These hemodynamic abnormalities likely promoted venous stasis, which represents the primary mechanism for thrombus formation in this patient, rather than an intrinsic hypercoagulable state.

Clinically, this case emphasizes the importance of maintaining a high index of suspicion for VTE in adolescents with congenital heart disease and signs of right-sided HF, even when clinical prediction scores suggest low probability. Bilateral edema may be mistakenly attributed to venous congestion, delaying DVT diagnosis. Early recognition and timely initiation of anticoagulation are essential to prevent progression to pulmonary embolism and further clinical deterioration.[4, 7]

Conclusions

VTE can occur in patients with CRS, particularly those with congenital heart disease and resultant HF. Bilateral DVT should be considered in the differential diagnosis of lower limb edema, even when clinical prediction scores suggest low probability. Prompt recognition and initiation of anticoagulation are essential to prevent progression to pulmonary embolism and other complications. Clinicians should remain vigilant for signs of VTE in this population.

Declarations:

Ethical Approval

This study protocol received approval from the Research Ethics Committee of Dr Ramelan Naval Central Hospital and was declared eligible to conduct data collection and research at Dr Ramelan Naval Central Hospital by the intelligent staff of Main Naval Base V.

Funding

According to the authors, this article has no financial support.

Conflict of Interest

The authors declare no competing interest in this study.

Acknowledgments

The authors thank the medical staff of Dr. Ramelan Naval Centre Hospital and the patient for their cooperation and consent to publish this case.

References

1. Tagalakis V, Patenaude V, Kahn SR, Suissa S. Incidence of and mortality from venous thromboembolism in a real-world population: The Q-VTE study cohort. Am J Med. 2013;126(9):832.e13–832.e21.
2. Xu T, Huang Y, Liu Z, Bai Y, Ma Z, Cai X, et al. Heart failure is associated with increased risk of long-term venous thromboembolism. Korean Circ J. 2021;51(9):766-80.
3. Brill A. Multiple Facets of Venous Thrombosis. Int J Mol Sci. 2021 Apr 8;22(8):3853.
4. Chan NC, Weitz JI. Recent advances in understanding, diagnosing and treating venous thrombosis. F1000Res. 2020 Oct 6;9:F1000 Faculty Rev-1206.
5. Schneider DJ, Moore JW. Patent ductus arteriosus. Circulation. 2006;114(17):1873-82.
6. Vynnycky E, Knapp JK, Papadopoulos T, Cutts FT, Hachiya M, Miyano S, et al. Estimates of the global burden of Congenital Rubella Syndrome, 1996–2019. Int J Infect Dis. 2023;137:149-56.
7. Toizumi M, Do CGT, Motomura H, Do TN, Fukunaga H, Iijima M, et al. Characteristics of Patent Ductus Arteriosus in Congenital Rubella Syndrome. Sci Rep. 2019;9:1-12.
8. Kaushik A, Verma S, Kumar P. Congenital rubella syndrome: A brief review of public health perspectives. Indian J Public Health. 2018;62(1):52-4.
9. Majiyagbe OO, Akinsete AM, Adeyemo TA, Salako AO, Ekure EN, Okoromah CAN. Coagulation abnormalities in children with uncorrected congenital heart defects seen at a teaching hospital in a developing country. PLoS One. 2022;17(7):1-16.
10. Dammeyer J. Congenital rubella syndrome and delayed manifestations. Int J Pediatr Otorhinolaryngol. 2010;74(9):1067-70.

11. O'Dea AF, Mayhall CA. Delayed manifestations of congenital rubella. J Vis Impair Blind. 1988;82(9):379-81.

12. Sever JL, South MA, Shaver KA. Delayed manifestations of congenital rubella. Rev Infect Dis. 1985 Mar-Apr;7 Suppl 1:S164-9.

13. Gupta V, Verma S, Ramakrishnan S, Hote M, Seth S, Bhargava B. Congenital rubella syndrome presenting in adulthood with fatigue. Circulation. 2013;128(23):2542-5.

14. Wienecke LM, Cohen S, Bauersachs J, Mebazaa A, Chousterman BG. Immunity and inflammation: the neglected key players in congenital heart disease? Heart Fail Rev. 2022;27:1957-71.

15. Mawson AR, Croft AM. Rubella virus infection, the congenital rubella syndrome, and the link to autism. Int J Environ Res Public Health. 2019;16(19):3543.