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CASE REPORTS

Antiphospholipid syndrome in an adolescent with refractory immune thrombocytopenia and massive central venous thrombosis

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ABSTRACT

Introduction and aim. Immune thrombocytopenia (ITP) and antiphospholipid syndrome (APS) may coexist, creating a clinical paradox of simultaneous bleeding risk and thrombophilia. In children and adolescents, APS can remain unrecognized for years when thrombocytopenia dominates the early course. The thrombotic risk associated with thrombopoietin receptor agonists (TPO-RAs) in this setting remains uncertain.

Description of the case. A 17-year-old boy with refractory ITP during romiplostim therapy developed massive upper-extremity and central venous thrombosis with near-complete superior vena cava obstruction. Persistent lupus anticoagulant, anticardiolipin, and anti-β2-glycoprotein I antibody positivity established APS. He was treated with heparin, alteplase, and long-term warfarin plus low-dose aspirin, while romiplostim was continued to support safe anticoagulation. No recurrent thrombosis or major bleeding was observed during 20 months of follow-up.

Conclusion. Thrombocytopenia does not protect against thrombosis in APS. In adolescents with chronic or refractory ITP, especially when vascular symptoms, unexplained prolonged aPTT, or other autoimmune red flags are present, targeted antiphospholipid antibody testing should be considered. Management of combined ITP and APS requires individualized balancing of anticoagulation and platelet support, with vitamin K antagonists remaining the preferred long-term anticoagulant strategy in high-risk APS.

Keywords. adolescent, antiphospholipid syndrome, immune thrombocytopenia, romiplostim, venous thrombosis

Introduction

Primary immune thrombocytopenia (ITP) is an acquired autoimmune disorder characterized by isolated thrombocytopenia (platelet count<100×10^9/L) in the absence of other causes of thrombocytopenia or disorders associated with thrombocytopenia.[1,2] It is the most common cause of thrombocytopenia in children and adolescents.[1] In a population-based study, the annual incidence of pediatric ITP was 4.2 per 100,000 person-years.[3]

Antiphospholipid syndrome (APS) is an acquired autoimmune thrombotic disorder defined by the combination of at least one clinical criterion (vascular thrombosis or pregnancy morbidity) and one laboratory criterion, namely persistent antiphospholipid antibodies (aPL) – lupus anticoagulant (LA), anticar-

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diolipin antibodies (aCL), and/or anti-β2-glycoprotein I antibodies (anti-β2GPI) – present on at least two occasions at least 12 weeks apart.[4] In children, APS is rare, with an incidence of 0.2 per 100,000 reported in some populations.[5]

The coexistence of chronic refractory ITP and primary APS represents a major paradox in pediatric hematology: the same patient may be predisposed to bleeding because of thrombocytopenia while remaining at risk of thrombosis driven by aPL-mediated hypercoagulability. Standard ITP therapies can raise platelet counts but do not address the underlying APS-related prothrombotic tendency; accordingly, observational data suggest that thrombotic event rates during TPO-RA therapy are markedly higher in patients with definite APS.[6,7]

Aim

This report describes an adolescent with refractory ITP who developed massive central venous thrombosis during romiplostim therapy and was subsequently diagnosed with APS. The case is notable for the pediatric age, delayed APS recognition, unusually extensive central venous thrombosis, and the need for continued TPO-RA support despite a major thrombotic event. Importantly, its clinical significance arises from the coexistence of refractory ITP, previously unrecognized primary APS, and exposure to TPO-RA, which together created a high-risk and diagnostically misleading phenotype. This combination illustrates the potential for APS to remain masked by thrombocytopenia while clinically manifesting only after severe thrombotic complications during therapy. We highlight the diagnostic challenges of this rare overlap and discuss the implications for anticoagulation, platelet-supportive therapy, and earlier recognition of APS in adolescents with refractory thrombocytopenia.

Description of the case

A 17-year-old boy was admitted to the Department of Pediatric Oncology and Hematology in August 2023. On admission, he was 182 cm tall and weighed 108 kg, corresponding to a body mass index of 32.6 kg/m^2 (class I obesity). He had initially been diagnosed with immune thrombocytopenia in November 2019 after presenting with petechiae, ecchymoses, and severe thrombocytopenia (platelet count $<5\times10^9$/L). Bone marrow examination excluded malignancy and myelodysplasia. Over the following years, he received multiple therapies, including intravenous immunoglobulin, repeated courses of glucocorticoids, azathioprine, mycophenolate mofetil, and eltrombopag. Eltrombopag produced an initially encouraging response, with the platelet count reaching 140×10^9/L in May 2021; however, the response was not sustained, and a subsequent downward trend was observed, with the platelet count decreasing to 20×10^9/L at the last visit before treatment modification. Eltrombopag was also discontinued because of severe headaches. Because of refractory thrombocytopenia, romiplostim was started in December 2022 and titrated individually at 1–3 μg/kg weekly, with platelet counts usually maintained between 47 and 110×10^9/L. Previous coagulation assessments performed during the earlier course of the disease did not indicate coagulation factor deficiencies. Although intermittent aPTT prolongation had been observed, there were no thrombotic manifestations or other clinical features suggestive of APS at that time. Broader autoimmune evaluation did not establish systemic autoimmune disease: ANA showed only low-titer positivity in 2020 and was negative on repeat testing in August 2023. Therefore, the overall clinical picture – severe isolated thrombocytopenia, bleeding symptoms, exclusion of bone marrow pathology, and lack of clinical features of systemic autoimmune disease supported the diagnosis of primary refractory ITP. Relevant comorbidities included selective IgA deficiency (IgA 0.53 g/L; reference range 1.00–4.00), hypertension treated with hydrochlorothiazide, and obesity. In July 2023, he was treated with doxycycline for erythema migrans associated with Borrelia burgdorferi IgG seropositivity.

Thrombotic event presentation (August 2023)

He was admitted with a 7–10-day history of progressive edema, pain, and erythema of the left upper limb extending to the left side of the neck and anterior chest wall, accompanied by low-grade fever (up to 38.0°C). On examination, the left upper limb was markedly swollen, with predominantly non-pitting edema extending to the shoulder girdle and neck. Prominent superficial collateral veins were visible over the thorax and abdomen. According to the family, the first thoracic collateral veins had become visible in mid-July 2023, suggesting that the thrombotic process had been evolving for more than 4 weeks before admission.

Laboratory findings

Laboratory evaluation demonstrated markedly prolonged aPTT (>100 s), persistent lupus anticoagulant positivity, and repeated positivity of anticardiolipin and anti-β2-glycoprotein I antibodies on follow-up testing performed more than 12 weeks apart. Because local assays reported combined rather than isotype-specific results, formal "triple positivity" could not be assigned despite persistent antibody positivity for all three aPL categories. Platelet count on admission was 95×10^9/L, and D-dimer was markedly elevated (61,485 ng/mL).

Imaging

CT angiography showed extensive thrombosis of the left internal jugular, subclavian, axillary, brachial, and brachiocephalic veins, with near-complete superior vena cava obstruction and extensive collateral venous circulation. Doppler ultrasonography confirmed occlusion of the left upper-extremity and cervical veins, with markedly reduced flow on the right side; no lower-extremity thrombosis was detected. Echocardiography showed no right ventricular strain, while bilateral pleural effusions were consistent with chylothorax secondary to central venous obstruction. Brain MRI was performed in November 2023, three months after the index event, to exclude cerebral venous sinus thrombosis in view of the extensive cervico-thoracic venous involvement and persistent headache. The examination excluded cerebral venous sinus thrombosis.

Diagnosis

The patient fulfilled the revised Sydney classification criteria for definite antiphospholipid syndrome, based on objectively confirmed extensive venous thrombosis and persistent lupus anticoagulant positivity on repeat testing performed more than 12 weeks apart. Repeated positivity of anticardiolipin and anti-β2-glycoprotein I antibodies further supported the diagnosis, although formal triple aPL positivity was not assigned because the local assays reported combined isotypes. Thus, these findings indicate a high-risk aPL profile rather than strictly confirmed triple positivity. No clinical features of systemic lupus erythematosus or another systemic autoimmune disease were identified.[4]

Management and outcomes

In the acute phase, anticoagulation was initiated with continuous intravenous unfractionated heparin (UFH), monitored by anti-Xa activity because of lupus anticoagulant-related interference with aPTT. Given the extent of thrombosis, near-complete superior vena cava obstruction, and a platelet count above 50×10^9/L, systemic thrombolysis with alteplase was administered, resulting in partial reperfusion. Dabigatran was used only as a short transitional regimen while the APS work-up was being completed. After APS was confirmed, long-term therapy was changed to warfarin, consistent with recommendations favoring vitamin K antagonists over direct oral anticoagulants in thrombotic APS, particularly in high-risk aPL profiles.[6,8,9] Romiplostim was continued as an individualized supportive measure to maintain platelet counts compatible with safe anticoagulation, rather than as APS-directed therapy.[7,10]

Complications included puncture-site bleeding and pleural effusions. Catheter-directed thrombolysis and thrombectomy were not pursued because the extensive collateral circulation suggested partial chronicity of the process and the risk of procedure-related pulmonary embolism was considered high. Central venous access was obtained via the right femoral vein, and right-sided thoracocentesis was performed. A short transitional oral regimen with dabigatran 150 mg twice daily plus acetylsalicylic acid (ASA) 75 mg daily was used pending completion of the antiphospholipid antibody work-up; after APS had been confirmed, long-term treatment was changed to warfarin.

For long-term secondary prevention, warfarin was introduced with a target INR of 2.0–3.0, in line with adult EULAR recommendations for venous thrombotic APS and with pediatric APS practice, in which initial heparin therapy is followed by warfarin.[6,8] Low-dose ASA 75 mg daily and romiplostim (125 μg subcutaneously every 2 weeks) were continued as part of an individualized strategy aimed at maintaining platelet counts compatible with safe anticoagulation. At 20-month follow-up, the patient remained clinically stable, with no

Table 1. Timeline of the patient's disease course

Date	Event	Platelet count ($\times10^9$/L)	Treatment/key interventions	Important findings/comments
November 2019	Diagnosis of primary immune thrombocytopenia (ITP)	<5	IVIg, glucocorticoids	Petechiae and ecchymoses
2019–2022	Multiple lines of therapy for refractory ITP	Variable (usually<30)	Glucocorticoids (repeated), azathioprine, mycophenolate mofetil, eltrombopag	Transient or no response; eltrombopag discontinued due to severe headache
December 2022	Initiation of romiplostim	47–110	Romiplostim (1–3 μg/kg weekly)	Partial response achieved
July 2023	Erythema migrans (Borrelia burgdorferi infection)	Not reported	Doxycycline	–
Mid–July 2023	First appearance of collateral veins on thorax (retrospectively)	Not reported	–	Early sign of developing thrombosis
August 2023	Acute presentation: massive upper extremity and central venous thrombosis	95	UFH → alteplase (systemic thrombolysis), transitional dabigatran+ASA	Near-complete SVC obstruction, markedly prolonged aPTT, positive LA
August–November 2023	APS confirmation	95 → rising	Switch to warfarin (target INR 2.0–3.0)+ASA 75 mg	Persistent LA, positive aCL and anti-β2GPI
November 2023	Brain MRI	–	–	No cerebral venous thrombosis
August 2023–April 2025	Follow-up (20 months)	95 → 210	Warfarin+ASA+romiplostim (125 μg every 2 weeks)	No recurrent thrombosis or major bleeding, partial vein recanalization, persistent aPL positivity

recurrent thrombosis or major bleeding and preserved left upper-limb function. The platelet count increased from 95×10^9/L at presentation to 210×10^9/L, D-dimer normalized, and the INR was therapeutic at 2.66, while aPTT remained prolonged (121.4 s), consistent with persistent lupus anticoagulant activity. Repeat antiphospholipid testing continued to show positive lupus anticoagulant, repeated anticardiolipin antibody positivity, and persistently elevated anti-β2-glycoprotein I antibodies. Follow-up imaging demonstrated partial recanalization of the left upper-extremity veins, but superior vena cava occlusion persisted, indicating chronic central venous sequelae despite clinical improvement. Overall, the follow-up course supported ongoing long-term anticoagulation in the setting of thrombotic APS with persistent laboratory activity. The key diagnostic and therapeutic milestones of the patient's disease course are summarized in Table 1.

Discussion

The central diagnostic issue was whether thrombocytopenia initially managed as primary ITP represented an isolated disorder or an early manifestation of APS. At presentation, the patient fulfilled criteria for primary ITP, with severe isolated thrombocytopenia, bleeding symptoms, exclusion of marrow pathology, and no clinical or laboratory evidence of systemic autoimmune disease or thrombosis; therefore, the initial diagnosis was appropriate and should not be considered incorrect in retrospect. Only after APS became definite could the thrombocytopenia be reinterpreted as possibly APS-related or as an ITP phenotype within APS.

This diagnostic overlap is supported by evidence that antiphospholipid antibodies may be present in a subset of ITP patients before overt APS develops, although progression to thrombosis is variable and not predictable. Accordingly, these findings support diagnostic vigilance rather than routine aPL screening in all ITP cases.[11,12]

Thrombocytopenia does not confer protection against thrombosis in APS, where bleeding is relatively uncommon compared with thrombotic complications.[13]

In this case, extensive central venous thrombosis occurred despite a platelet count of 95×10^9/L, underscoring that platelet count alone is not a reliable indicator of vascular risk.

The temporal association between romiplostim and thrombosis should be interpreted within a multifactorial "multiple-hit" model. Previous reports have described severe thrombotic complications, including catastrophic APS (CAPS), occurring during romiplostim therapy in patients with antiphospholipid antibodies or established APS. However, the present case differs in several important aspects: APS was not initially recognized, the thrombotic presentation involved unusually extensive upper-extremity and central venous thrombosis rather than CAPS, and long-term management required continued TPO-RA support together with warfarin anticoagulation. These features illustrate a distinct and diagnostically challenging clinical phenotype at the intersection of refractory ITP, APS, and thrombopoietin receptor agonist exposure. Persistent antiphospholipid antibody activity represented the primary prothrombotic driver, while obesity, hypertension, infection, delayed diagnosis, and possibly romiplostim may have contributed. Pediatric data on TPO-receptor agonists are overall reassuring but limited in patients with APS, and available adult data suggest a higher thrombotic burden in this subgroup; therefore, causality cannot be attributed to romiplostim alone.[7,14,15]

The extent and distribution of thrombosis – multiple upper-extremity and central venous segments with near-complete superior vena cava obstruction – are consistent with pediatric venous APS but represent an unusually severe and extensive pattern compared with registry data, where lower-extremity DVT predominates.[16]

In retrospect, earlier reassessment with antiphospholipid antibody testing could have been considered due to refractory thrombocytopenia and evolving clinical features, which represent red flags for secondary causes of presumed ITP. This supports a selective rather than routine approach to aPL testing in chronic or refractory cases.[17]

To place the present case in context, the available literature at the intersection of pediatric APS, chronic ITP, antiphospholipid antibody positivity, and TPO-receptor agonist exposure is summarized in Table 2.

Collectively, the available literature addresses isolated aspects of pediatric APS, chronic ITP, antiphospholipid antibody positivity, or TPO-RA exposure, but evidence integrating all of these factors within a single pediatric clinical scenario remains extremely limited.

Once APS-associated thrombosis was diagnosed, long-term anticoagulation with warfarin was initiated in accordance with recommendations favoring vitamin K antagonists over direct oral anticoagulants in high-risk APS.[6,8,9] Continuation of romiplostim was an individualized decision to maintain platelet counts sufficient for safe anticoagulation. This approach reflected the complex therapeutic dilemma between bleeding risk and recurrent thrombosis risk and should not be interpreted as a general treatment recommendation for APS-associated thrombosis.[7]

Conclusion

This case is hypothesis-generating. It illustrates that thrombocytopenia, initially fulfilling clinical criteria for primary ITP can, in selected patients, later be reconsidered as APS-associated thrombocytopenia or as an ITP phenotype occurring in the setting of APS. The ob-

Table 2. Published evidence relevant to pediatric ITP, APS, antiphospholipid antibodies, and TPO-RA-associated thrombotic risk

Study	Year	Population type	N/population	APS/aPL status	TPO-RA exposure	Main thrombosis phenotype	Key findings relevant to this case	Remaining uncertainty/knowledge gap
Avčin et al. (Ped-APS Registry)[16]	2008	Pediatric APS	121 children with definite APS	Definite APS	No	Venous thrombosis 60%; lower-extremity DVT most common; CVST 7%	Largest pediatric APS registry; demonstrates substantial thrombotic burden, recurrence risk, and hematologic manifestations including thrombocytopenia	Does not address chronic refractory ITP, TPO-RA exposure, or management of APS-associated thrombocytopenia
Berkun et al.[18]	2006	Pediatric APS	28 children with APS without systemic autoimmune disease at presentation	Definite APS	No	Venous and arterial thrombosis; recurrent thrombosis 29%	Supports severe long-term thrombotic burden and need for prolonged anticoagulation in pediatric APS	No data on ITP-directed therapies or TPO-RA-associated risk
Tomasello et al.[13]	2021	Review / mixed	Review focused on thrombocytopenia in APS	APS-associated thrombocytopenia	Indirect discussion only	N/A	Emphasizes diagnostic overlap between APS-associated thrombocytopenia and primary ITP	No pediatric outcome data; no data on thrombosis during TPO-RA exposure
Schifferli et al. (PARC-ITP)[17]	2021	Pediatric ITP	3974 children initially diagnosed with primary ITP	Non-APS ITP cohort	No	N/A	Identifies "red flags" for later diagnostic revision of presumed primary ITP	APS-specific screening strategies and thrombotic outcomes not evaluated
Dayama et al.[11]	2017	Mixed pediatric/adult ITP	100 ITP patients	aPL-positive ITP without APS	No	No thrombotic events during follow-up	Demonstrates that aPL positivity may occur in chronic ITP even without overt APS	Short follow-up; no longitudinal APS evolution or thrombosis-risk stratification
Diz-Küçükkaya et al.[12]	2001	Mixed pediatric/adult ITP	82 newly diagnosed ITP patients	aPL-positive ITP	No	Venous and arterial APS manifestations during follow-up	Strong prospective evidence that aPL-positive ITP may evolve into overt APS; LA identified as major risk marker	No pediatric-focused analysis; no data on TPO-RA exposure
Grainger et al. (PETIT2)[14]	2015	Pediatric ITP	92 children with chronic ITP	Non-APS ITP	Yes (eltrombopag)	No thromboembolic events reported	Population-level pediatric TPO-RA data are reassuring	Children with APS or persistent aPL positivity were not specifically assessed
Neunert et al. (ICON2)[15]	2016	Pediatric ITP	79 children; 87 TPO-RA treatment courses	Mixed primary/secondary ITP; APS status not defined	Yes	2 pulmonary emboli during eltrombopag exposure	Real-world pediatric TPO-RA data suggest low absolute thrombotic event frequency	APS-specific thrombotic risk during TPO-RA therapy remains undefined
Marques et al.[7]	2025	Adult APS/aPL-associated ITP	80 adults with ITP associated with SLE/aPL/APS; definite APS subgroup n=16	Mixed SLE/aPL/APS; definite APS subgroup analyzed separately	Yes	21 thrombotic events including venous, arterial, and catastrophic APS	Most clinically relevant comparator; thrombotic events occurred in 50% of patients with definite APS during TPO-RA exposure	Findings derive exclusively from adults and cannot be directly extrapolated to pediatric patients
Present case	2026	Pediatric refractory ITP+APS	Single adolescent patient	Delayed recognition of definite APS after years of refractory ITP	Yes (romiplostim continued after thrombosis)	Massive upper-extremity and central venous thrombosis with near-complete SVC obstruction	Illustrates the convergence of refractory pediatric ITP, previously unrecognized APS, TPO-RA exposure, and need for continued platelet support during long-term anticoagulation	Evidence guiding management of this specific high-risk pediatric overlap phenotype remains extremely limited

servation does not support universal antiphospholipid antibody screening in all children with ITP. Rather, it supports targeted aPL testing when chronic or refractory ITP is accompanied by vascular, neurologic, autoimmune, or coagulation red flags.

The novelty of this report lies in the delayed diagnosis of APS after years of refractory ITP phenotype, the unusually extensive upper-extremity and central venous thrombosis with near-complete superior vena cava obstruction, and the need to balance long-term anticoagulation with continued platelet-supportive therapy. The clinical course is best explained by cumulative rather than single-drug risk. In combined ITP and APS, the therapeutic goal shifts from normalizing the platelet count to maintaining a platelet count sufficient for safe anticoagulation while reducing modifiable thrombotic risks.

Declarations

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Author contributions
Conceptualization, M.M.M. and W.B.; Methodology, G.M.W.; Investigation, M.M.M; Resources, M.M.M and W.B; Data Curation, M.M.M and W.B. and G.M.W.; Writing – Original Draft Preparation, M.M.M.; Writing – Review & Editing, M.M.M and R.C.; Supervision, R.C.;

Conflicts of interest
The authors declare no conflict of interest.

Data availability
The data presented in this study are available from the corresponding author upon reasonable request.

Ethics approval
Written informed consent was obtained from the patient.

Use of AI and AI-assisted technologies in the writing process
The authors used ChatGPT (OpenAI) exclusively for language editing and readability improvement which is allowed by the Eur J Clin Exp Med regulations concerning use of artificial intelligence. All scientific content, interpretation of data, and conclusions were developed independently by the authors, who take full responsibility for the final manuscript.

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