APGHN Archives of Pediatric Gastroenterology, Hepatology, and Nutrition

Case Report

Silent Splenic Infarction Following Infectious Mononucleosis Associated with Antiphospholipid Antibodies in a Pediatric Patient: A Case Report

Davide Scarponi[1], Branislava Ćosić[1], Costanza Fiorilli[1], Anna Rita Di Biase[2], Lorenzo Iughetti[1]
[1]University of Modena and Reggio Emilia, Modena, Italy
[2]Azienda Ospedaliero - Universitaria of Modena, Modena, Italy

e-ISSN: 2830-5442

Corresponding author:
Davide Scarponi
davide.scarp93@gmail.com

Published:
31st May 2025

DOI:
https://doi.org/10.58427/apghn.4.2.2025.78-82

Citation:
Scarponi D, Ćosić B, Fiorilli C, Di Biase AR, Iughetti L. Silent splenic infacrtion following infectious mononucleosis associated with antiphospolipid antibodies in a pediatric patient: a case report. *Arch Pediatr Gastr Hepatol Nutr.* 2025;4(2): 78-82

Abstract:

Background: The splenic infarction (SI) is a rare complication of infectious mononucleosis (IM), especially in paediatric population. The clinical presentation of this condition can vary widely, but it is most often symptomatic (e.g. pain in the left upper quadrant).

***Case*:** We report a case of a previously healthy 12 year old female with a silent splenic infarction (SI) following IM by Epstein-Barr virus (EBV), positive for antiphospholipid anti-cardiolipin IgM antibodies (ACA IgM).

***Discussion*:** Numerous pathogenetic mechanisms have been proposed for the SI in the course of IM. The definitive diagnosis of SI is made with CT; in our case, given the lack of urgent clinical indications, the definitive diagnosis was made with MRI. Follow-up was continued using ultrasound and therapeutic management was conservative.

Conclusion: We describe a rare case of asymptomatic splenic infarction, in a girl with no underlying predisposing condition. It is important for a pediatrician to be aware of this possible complication and its correct therapeutic management.

Keywords: antiphospholipid antibodies, epstein-barr virus, mononucleosis, pediatric, splenic infarction

Introduction

Splenic infarction (SI) is a complication of various pediatric pathologies: haematological (10%), cardioembolic (22%), discoagulopathies (22%), sepsis (10%), vascular disease of the spleen or connective tissue disorders. It can also be a rare complication of infectious mononucleosis (IM) and for this reason it is often underdiagnosed and remains unrecognised in the absence of associated symptoms (e.g. pain in the left upper quadrant). The association with IM was found only in 3 out of 49 patients described in the largest case series of SI in the literature.[1] Heo et al. reported between 1961 and 2015 19 cases of splenic infarction following IM by EBV infection 2. There are even fewer cases in the pediatric population.[2]

Case

The 12-year-old girl presented to the Pediatric Emergency Room with asthenia and headache in the course of IM caused by EBV, previously diagnosed by her pediatrician (EBV IgM > 160 S/CO [positive ≥ 40]; IgG 21.1 S/CO [positive ≥ 20]; EBNA negative). Additionally, an abdominal ultrasound (US), performed several days earlier, showed splenomegaly with a bipolar diameter of 15 cm without focal lesions and two accessory spleens measuring 1 cm each. Her medical history was unremarkable. Upon arrival, she presented with fever (40°C), exudative tonsillitis, splenomegaly, and signs of moderate dehydration. Laboratory tests revealed a mild rise in inflammatory biomarkers (CRP 3,1 mg/dl). She was admitted for observation and supportive care. Her clinical course was regular, and she was discharged after four days. Abdominal US follow-up, performed two weeks post-discharge, showed a 2 cm hypoechoic area suggestive of SI. The patient was otherwise well and no abdominal trauma was reported. As a result, she was re-hospitalized for further evaluation. A contrast MRI confirmed the presence of subacute splenic haemorrhagic infarction (**Figure 1.**) with no evidence of splenic rupture, malformations or vascular anomalies. Further diagnostic work-up that included thrombophilia screening, revealed presence of ACA IgM (25 MPL-U/ml [negative < 10 MPL-U/ml]); ACA IgG were negative; PT INR, aPTT ratio, liver and kidney function, ESR, haemoglobin electrophoresis, peripheral blood smear, homocysteinemia, ANA, anti-beta2-glycoprotein antibodies, ANCA, APC resistance, Protein C, Protein S, Factor VIII, Factor V and II mutations, Apolipoprotein A1 and B, C3, C4 were all within range. Additionally, elevated cholesterol levels were noted, due to previously undiagnosed familial hypercholesterolemia (cholesterol 415 mg/dl, HDL 76 mg/dl, LDL 281 mg/dl). Echocardiography and supra-aortic trunk US were normal. The clinical course remained uneventful. The SI was managed conservatively; the patient reported no pain or additional symptoms. She was discharged in good condition after four days. Subsequent follow-up showed complete resolution of the lesion on the US and negativization of ACA after two months.

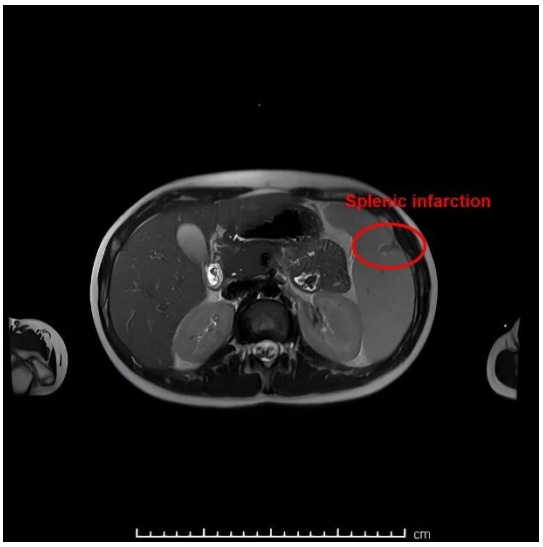

Figure 1. MRI image of splenomegaly and splenic infarction: enlarged spleen with a bipolar diameter of 13.8 cm; in the inferior anterior segment there is a triangular subcapsular area with a base of 2x1 cm and cranio-caudal extension of 1.5 cm, compatible with the early phase subacute haemorrhagic infarct.

Discussion

Splenic rupture is a rare complication of IM affecting 0.5-1% of patients, whilst the incidence of SI is unknown due to the low number of cases reported to date. Numerous pathogenetic mechanisms have been proposed for the SI in the course of IM. Some authors suggest that EBV infection may induce hypoxemia, high level of circulating immune complexes (which promote leucocyte adhesion and aggregation) or transient hypercoagulability state. Others focus on the potential role of transient antiphospholipid antibody (aPLs) production during EBV infection.[3-5] aPLs are a heterogeneous group of autoantibodies targeting phospholipid-binding proteins, such as β2-glycoprotein I (β2GPI) and cardiolipin. Persistent, high titer aPLs are a hallmark of antiphospholipid syndrome (APS) and are linked to thrombotic or obstetric complications.[6] Viral infections, including EBV, induce proliferation of B lymphocytes with a consequent production of various antibodies, including aPLs. These antibodies are typically transient (disappearing within 1-4 years), of low titer, often of the IgM isotype and usually do not corelate with the risk of thrombosis or other manifestations of APS.[7, 8]

Nonetheless, some studies have reported possible association between transient aPL positivity and ischemic events, suggesting that under certain conditions, these antibodies may have pathogenic potential.[9-11] Additionally, temporary reduction in anticoagulation factors such as Protein C or Protein S have been described in the context of acute infections.[12] Splenomegaly may further exacerbate the risk of ischemia

by creating a mismatch between oxygen supply and metabolic demand in splenic tissue.

In our case, we inferred that EBV infection triggered a transient increase of aPLs that could have played a role in pathogenesis of SI, as hypothesized in several articles.[4, 5, 7, 13, 14] Splenomegaly, that may have contributed to SI, was described in almost all cases, including ours. Furthermore, our patient was also found to have previously undiagnosed familial hypercholesterolemia (FH), a known prothrombotic condition, which may have further contributed to the thrombotic risk. Beyond the various hypotheses proposed, the mechanisms underlying SI pathogenesis remain unclear and need further research.

In reported case series it is uncommon for SI following EBV infection to be asymptomatic.[2] The definitive diagnosis of SI is made using CT.[1] However, in our case, considering the lack of urgent indications, the definitive diagnosis was made using MRI, to avoid radiation exposure. Although ultrasound has a low sensitivity in the detecting SI, it is useful for initial assessment and longitudinal monitoring of the lesion. The US follow-up of our patient documented a complete resolution of SI after two months. There is no specific treatment of SI apart from splenectomy, which should be considered only in case of surgical complications or in an unstable patient.[4]

Conclusion

In conclusion, we reported the atypical case of silent SI associated with the presence of ACA, a rare complication of IM, especially in pediatric population and in the absence of significant clinical history. It is important for a pediatrician to be aware of this possible complication and its correct therapeutic management. In case of US suspicion of SI in an asymptomatic, stable pediatric patient, it is possible to use MRI for a definitive diagnosis, when available. Subsequent follow-up can be continued with US. Surgical therapy should be reserved for selected cases with clinical instability.

Acknowledgement

This case report has been published with the consent of the patient parents. The authors would like to thank the patient and their parents.

Conflict of Interest

No potential conflict of interest was reported by the authors.

Funding Statement

This study was not supported by any external funding.

References

1. Antopolsky M, Hiller N, Salameh S, Goldshtein B, Stalnikowicz R. Splenic infarction: 10 years of experience. Am J Emerg Med. 2009;27(3):262-5. https://doi.org/10.1016/j.ajem.2008.02.014
2. Heo DH, Baek DY, Oh SM, Hwang JH, Lee CS, Hwang JH. Splenic infarction associated with acute infectious mononucleosis due to epstein-barr virus infection. J Med Virol. 2017;89(2):332-6. https://doi.org/10.1002/jmv.24618
3. Choi B, Kwon B, Kim GB, Jeon H-Y, Cheon J-E, Bae E, et al. A case of adolescent kawasaki disease with epstein-barr virus-associated infectious mononucleosis complicated by splenic infarction. Korean Journal of Pediatrics. 2009;52:1029. https://doi.org/10.3345/kjp.2009.52.9.1029
4. Naviglio S, Abate MV, Chinello M, Ventura A. Splenic infarction in acute infectious mononucleosis. J Emerg Med. 2016;50(1):e11-3. https://doi.org/10.1016/j.jemermed.2015.09.019
5. van Hal S, Senanayake S, Hardiman R. Splenic infarction due to transient antiphospholipid antibodies induced by acute epstein-barr virus infection. J Clin Virol. 2005;32(3):245-7. https://doi.org/10.1016/j.jcv.2004.07.013
6. Miyakis S, Lockshin MD, Atsumi T, Branch DW, Brey RL, Cervera R, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (aps). J Thromb Haemost. 2006;4(2):295-306. https://doi.org/10.1111/j.1538-7836.2006.01753.x
7. Ben-Chetrit E, Wiener-Well Y, Fadeela A, Wolf DG. Antiphospholipid antibodies during infectious mononucleosis and their long term clinical significance. J Clin Virol. 2013;56(4):312-5. https://doi.org/10.1016/j.jcv.2012.12.011
8. von Landenberg P, Döring Y, Modrow S, Lackner KJ. Are antiphospholipid antibodies an essential requirement for an effective immune response to infections? Ann N Y Acad Sci. 2007;1108:578-83. https://doi.org/10.1196/annals.1422.060
9. Carrasco M, Galarza-Maldonado C, Mendoza-Pinto C, Escárcega R, Cervera R. Infections and the antiphospholipid syndrome. Clinical reviews in allergy & immunology. 2009;36:104-8. https://doi.org/10.1007/s12016-008-8103-0
10. Abdel-Wahab N, Lopez-Olivo MA, Pinto-Patarroyo GP, Suarez-Almazor ME. Systematic review of case reports of antiphospholipid syndrome following infection. Lupus. 2016;25(14):1520-31. https://doi.org/10.1177/0961203316640912
11. Serrano M, Espinosa G, Cervera R, Serrano A. Covid-19 coagulopathy and antiphospholipid syndrome. Revista Colombiana de Reumatología. 2022;29:S25-S34. https://doi.org/10.1016/j.rcreu.2021.02.013
12. Gang MH, Kim JY. Splenic infarction in a child with primary epstein-barr virus infection. Pediatr Int. 2013;55(5):e126-8. https://doi.org/10.1111/ped.12143
13. Turrian U. Splenic infarction after epstein-barr virus infection: Case report. Annals of Clinical Case Reports. 2021;6. https://doi.org/10.25107/2474-1655.2045
14. Khan S, Saud S, Khan I, Prabhu S. Epstein barr virus-induced antiphospholipid antibodies resulting in splenic infarct: a case report. Cureus. 2019;11(2):e4119. https://doi.org/10.7759/cureus.4119