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CASE REPORT

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Thrombocytopenia secondary to acute appendicitis: A case report

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Abstract

Thrombocytopenia is a rare but possible complication of appendicitis and, if severe, might be a contraindication to appendectomy.

KEYWORDS

appendectomy, appendicitis, post-ileal, thrombocytopenia

1 | INTRODUCTION

The appendix is a blind-ending tube arising from the posteromedial aspect of the cecum, with a variably located free end that is mostly retrocecal or pelvic. Appendicitis—inflammation of the appendix—is one of the most common surgical emergencies worldwide. It occurs most frequently between the ages of 10 and 20 years and is predominant in males.[1] The clinical and laboratory presentation of appendicitis is fairly well known. However, it is rarely encountered as a cause of thrombocytopenia in clinical practice. Herein, we report a case of acute appendicitis with secondary thrombocytopenia that resolved on its own after appendectomy.

2 | CASE PRESENTATION

A 26-year-old man presented to the emergency department with a 16-hour history of abdominal pain that was periumbilical during the onset and subsequently migrated to the right iliac fossa. He had first presented to his local hospital 7 hours after the onset of pain, where he was managed with intravenous antibiotics for suspected appendicitis before being referred to our center for surgical intervention. He was nauseous and anorexic on presentation but reported no history of vomiting. Furthermore, his bowel and bladder habits were normal.

On examination, the patient was afebrile, hemodynamically stable, and fully conscious. There was tenderness to palpation in the right iliac fossa along with guarding and rebound tenderness. Rovsing's and Dunphy's signs were positive, but obturator and psoas signs were negative. A complete blood count revealed leukocytosis (2.07×10^{10}/L; normal: $4.0–11.0 \times 10^{9}$/L) with neutrophilia (1.66×10^{10}/L; normal: $2.5–7.5 \times 10^{9}$/L) but normal RBC and platelet counts. Liver function tests (LFTs) and renal panel were unremarkable.

The appendix could not be visualized in ultrasonography (USG) of the abdomen. However, contrast-enhanced

computed tomography (CT) scan revealed an enlarged post-ileal appendix (diameter: 11.4 mm) with wall-enhancement, thickened mesoappendix, and periappendiceal fat strandings, confirming the clinical suspicion of appendicitis (Figure 1). The patient was thus continued on antibiotic therapy, and an open appendectomy was performed the following day. The intraoperative findings were an inflamed, gangrenous, post-ileal appendix with a healthy base and absence of periappendiceal collection.

The patient had a normal platelet count a day before the appendectomy, but about 11 hours prior to the procedure, it dropped to 1.2×10^{11}/L (normal: 1.5–4.5 $\times 10^{11}$/L). The low platelet count, however, was not of much concern to the surgery as it was well beyond the threshold for perioperative bleeding risk (0.5×10^{11}/L). The count continued to decline postoperatively and reached its lowest at 0.79×10^{11}/L on the 3rd postoperative day (POD3). D-dimer level on the same day was elevated at 5.07 mcg/ml (normal: <0.5 mcg/ml). Thereafter, the platelet level started recovering till it was last checked for before the patient was discharged (Figure 2).

The platelet decline, being acute in onset, transient, and self-resolving, was unlikely to be precipitated by any bone marrow pathology. A normal spleen size on USG ruled out splenic sequestration of platelets, and normal LFTs dismissed the possibility of impaired thrombopoietin synthesis. Likewise, a dilutional cause was eliminated as the patient had not undergone any transfusion or massive fluid resuscitation and had other blood components (RBC, albumin) within normal limits. The only cause of thrombocytopenia that could be identified was increased consumption of platelets, suggested by the elevated D-dimer level. Further, the preoperative onset of the platelet decline established that the overt platelet consumption was not merely related to surgical hemostasis but stemmed from the underlying pathology—appendicitis.

The patient had an uneventful postoperative period and was discharged on oral antibiotics on POD5. Upon follow-up on POD11, he was found to have recovered well and reported no abdominal pain or discomfort. Moreover, he had an overshoot in the platelet count (6.2×10^{11}/L), consistent with the nature of physiological thrombopoietin-induced platelet recovery.

3 | DISCUSSION

The pathophysiology of appendicitis likely stems from appendiceal obstruction. The continuous secretion of mucus into an obstructed lumen results in an increase in intraluminal pressure, which eventually exceeds the capillary perfusion pressure. As the vascular compromise progresses, the wall becomes ischemic and ultimately necrotic. Bacterial overgrowth occurs within the necrosed appendix and is predominated by aerobic organisms early in the course, while a mixed infection is common later on. The mechanism of luminal obstruction varies depending on age. In the young, lymphoid follicular hyperplasia due to infection is thought to be the leading cause, whereas, in the old, it is more likely to be brought about by fibrosis, fecaliths, or neoplasia.[2]

A periumbilical colicky pain that subsequently migrates to the right iliac fossa is the primary presenting complaint of patients with acute appendicitis. Anorexia, nausea, and vomiting also constitute the classic symptoms of the disease.[1,2] The most reliable findings of physical examination that indicate the diagnosis of appendicitis are right iliac fossa tenderness, guarding, and rebound tenderness. Further examination techniques include Rovsing's sign, Dunphy's sign, psoas sign, and obturator sign.[1]

A complete blood count is an essential step in the diagnosis of appendicitis, with neutrophilic leukocytosis being the most accurate finding.[3] An elevated blood C-reactive protein level is also highly indicative of the disease but may not always be present.[4] The clinical suspicion of appendicitis is usually confirmed by an abdominal contrast-enhanced CT scan, with the diagnostic findings being an enlarged appendix (diameter >6 mm) with enhanced, thickened wall (> 2 mm), periappendiceal fat strandings, and appendicolithiasis.[5] Likewise, on

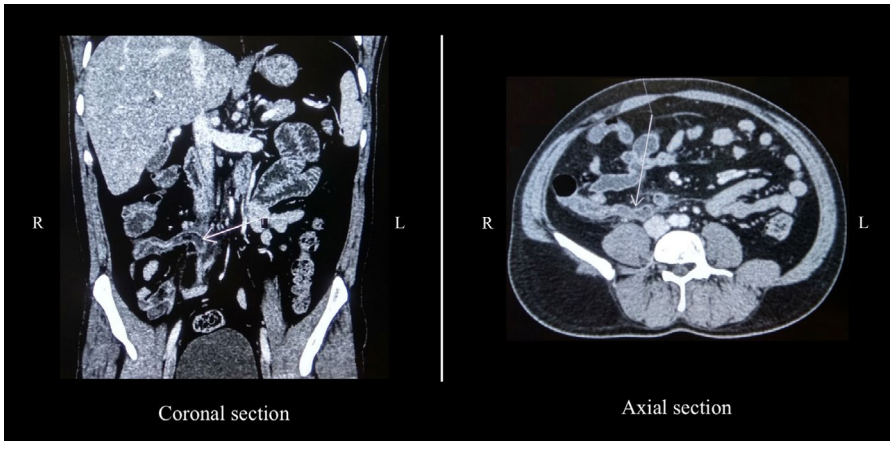

FIGURE 1 Contrast-enhanced CT scan of the abdomen with findings of acute appendicitis

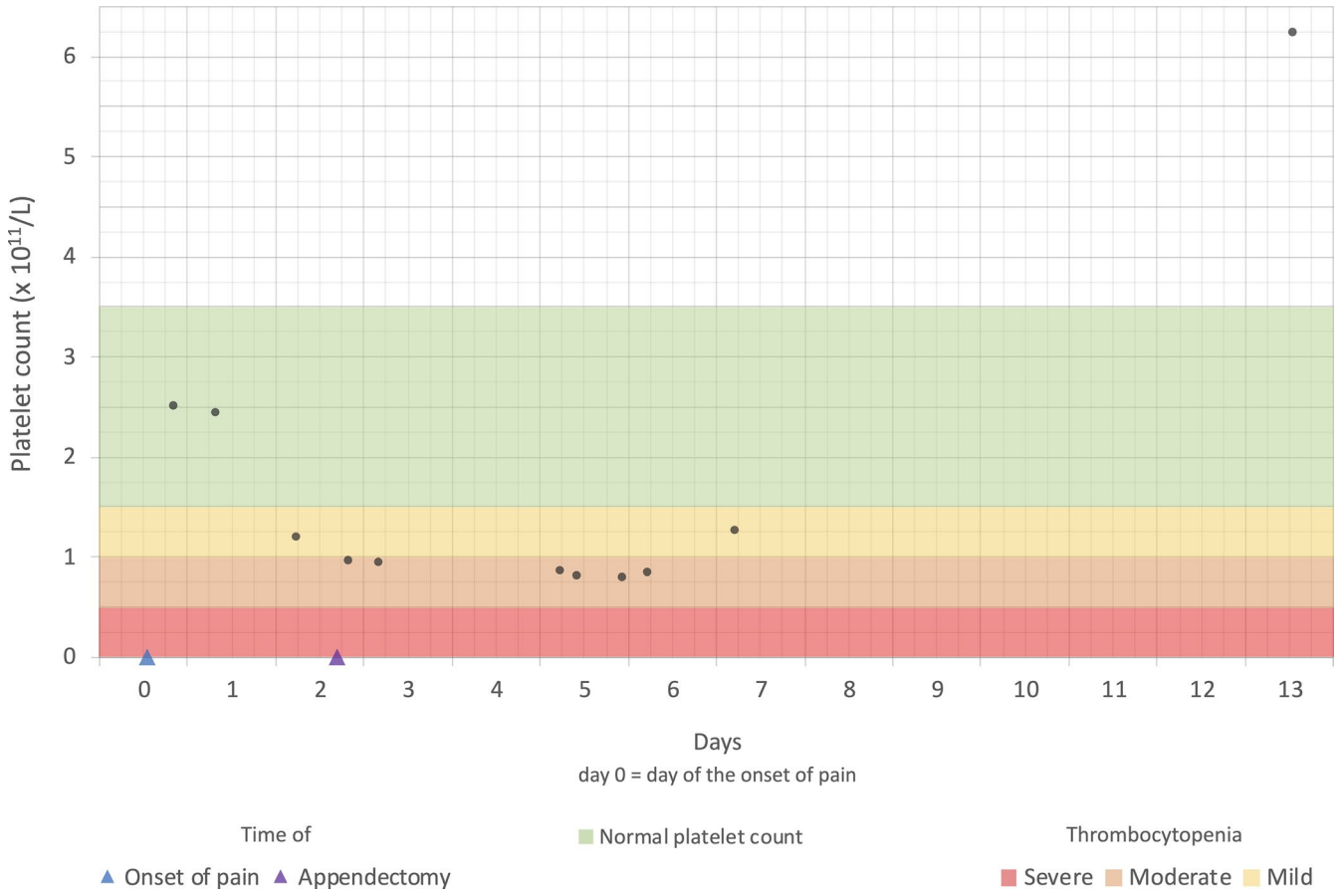

FIGURE 2 Scatterplot showing the trend in platelet count

USG, the most accurate finding for the disease is appendiceal dilatation (>6 mm); however, the appendix may not always be visualized on it.[6]

The standard treatment modality for appendicitis is appropriate fluid resuscitation, followed by expedient appendectomy—which can be open or laparoscopic, along with perioperative administration of broad-spectrum antibiotics. For uncomplicated appendicitis, there is evidence that antibiotic therapy alone can be as effective as surgery.[7] However, it is linked with higher recurrence rates, and one cannot always distinguish complicated from uncomplicated appendicitis with a preoperative CT scan. Thus, appendectomy still remains the standard of care for the treatment of appendicitis.[8]

Thrombocytopenia is defined as a platelet count below 1.5×10^{11}/L. The primary pathophysiological mechanisms that lead to it are decreased platelet production, hemodilution, increased platelet clearance due to destruction or consumption in clot formation, and splenic sequestration during splenomegaly. Decreased platelet production generally results from bone marrow disorders or liver dysfunction that causes impaired thrombopoietin synthesis. Likewise, dilutional thrombocytopenia occurs during massive transfusion and fluid resuscitation for severe bleeding, while platelet destruction is often due to autoimmune causes or increased desialylation-mediated clearance.[9]

Thrombocytopenia can result from a number of causes during infection, and the phenomenon is more apparent in sepsis, where a combination of multiple mechanisms is possible. Primarily, there occurs an increase in platelet activation, which may be pathogen- or host response-mediated, leading to overt platelet consumption. In addition, direct bone marrow suppression, increased platelet destruction, and pooling of platelets in the enlarged spleen also contribute to thrombocytopenia in patients with an infectious etiology.[10]

A low platelet count is common and expected after major surgery and primarily occurs due to transfusion- and fluid resuscitation-related hemodilution and platelet consumption in surgical hemostasis. The platelet count nadir usually occurs on POD2 to POD3 and is followed by a thrombopoietin-induced recovery that results in a physiological overshoot in the count. This overshoot is ascribable to the delay between the increase in thrombopoietin and the release of new platelets from the bone marrow. The platelet count peaks at two- to threefold the preoperative value at approximately POD14 before steadily returning to the baseline over the following two weeks.[11]

In this case, the platelet decline was established to be secondary to appendicitis; however, it might have been augmented to some degree by perioperative platelet consumption. Gradual recovery in the platelet count was observed after the appendectomy, followed by an overshoot, indicative of the compensatory thrombopoietin response to thrombocytopenia.

4 | CONCLUSION

Appendicitis is one of the most common surgical emergencies in the world; however, it is not often encountered as a cause of thrombocytopenia in practice. Appendectomy is the standard of care for the treatment of appendicitis, and the associated thrombocytopenia seems to resolve after it. However, the procedure might be contraindicated if the platelet count is severely low, requiring such cases to be dealt with cautiously.

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CONFLICT OF INTEREST
The authors declare that they have no competing interests.

AUTHOR CONTRIBUTIONS
Deechit Poudel wrote the original draft of the manuscript. All authors contributed to the review and editing of the manuscript. Naveen Chandra Bhatta, Deepak Sharma, and Diptee Poudel were involved in the diagnosis and treatment of the patient. All authors read and approved the final version of the manuscript.

ETHICAL STATEMENT
Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

DATA AVAILABILITY STATEMENT
Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

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REFERENCES

1. Humes DJ, Simpson J. Acute appendicitis. *BMJ*. 2006;333(7567):530-534. https://doi.org/10.1136/bmj.38940.664363.AE
2. Birnbaum BA, Wilson SR. Appendicitis at the millennium. *Radiology*. 2000;215(2):337-348. https://doi.org/10.1148/radiology.215.2.r00ma24337
3. Al-Gaithy ZK. Clinical value of total white blood cells and neutrophil counts in patients with suspected appendicitis: retrospective study. *World J Emerg Surg*. 2012;7(1):32. https://doi.org/10.1186/1749-7922-7-32
4. Dayawansa NH, Segan JDS, Yao HHI, Chong HI, Sitzler PJ. Incidence of normal white cell count and C-reactive protein in adults with acute appendicitis. *ANZ J Surg*. 2018;88(6):E539-E543. https://doi.org/10.1111/ans.13760
5. Choi D, Park H, Lee YR, et al. The most useful findings for diagnosing acute appendicitis on contrast-enhanced helical CT. *Acta Radiol*. 2003;44(6):574-582. https://doi.org/10.1046/j.1600-0455.2003.00146.x
6. Kessler N, Cyteval C, Gallix B, et al. Appendicitis: evaluation of sensitivity, specificity, and predictive values of US, Doppler US, and laboratory findings. *Radiology*. 2004;230(2):472-478. https://doi.org/10.1148/radiol.2302021520
7. Mason RJ, Moazzez A, Sohn H, Katkhouda N. Meta-analysis of randomized trials comparing antibiotic therapy with appendectomy for acute uncomplicated (no abscess or phlegmon) appendicitis. *Surg Infect (Larchmt)*. 2012;13(2):74-84. https://doi.org/10.1089/sur.2011.058
8. Krzyzak M, Mulrooney SM. Acute appendicitis review: background, epidemiology, diagnosis, and treatment. *Cureus*. 2020;12(6):e8562. https://doi.org/10.7759/cureus.8562
9. Smock KJ, Perkins SL. Thrombocytopenia: an update. *Int J Lab Hematol*. 2014;36(3):269-278. https://doi.org/10.1111/ijlh.12214
10. Assinger A, Schrottmaier WC, Salzmann M, Rayes J. Platelets in Sepsis: An Update on Experimental Models and Clinical Data. *Front Immunol*. 2019;10:1687. https://doi.org/10.3389/fimmu.2019.01687
11. Skeith L, Baumann Kreuziger L, Crowther MA, Warkentin TE. A practical approach to evaluating postoperative thrombocytopenia. *Blood Adv*. 2020;4(4):776-783. https://doi.org/10.1182/bloodadvances.2019001414

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