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TRANSIENT HAEMOLYTIC ANAEMIA AND THROMBOCYTOPENIA IN A HEALTHY YOUNG ADULT FOLLOWING INFLUENZA A INFECTION

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ABSTRACT

Background: Influenza A can trigger rare haematologic complications such as immune thrombocytopenia (ITP) and haemolytic anaemia, most commonly in paediatric or immunocompromised populations. We describe a case of post-influenza immune cytopaenias in a previously healthy adult.

Case description: A 22-year-old male presented with fever, haematuria and malaise after influenza A infection. Laboratory workup revealed severe thrombocytopenia, anaemia (haemoglobin 11.7 g/dl, haematocrit 34.1%), elevated reticulocyte count (3.7%, absolute 99 $\times 10^9$/l), acute kidney injury (serum creatinine 2.44 mg/dl), markedly elevated LDH (1,991 U/l), ferritin (6,016 ng/ml), low haptoglobin (<30 mg/dl) and iron saturation of 60%. A peripheral smear showed anisopoikilocytosis with fragmented red blood cells, including schistocytes. He was treated with oseltamivir and a four-day course of dexamethasone. Platelets normalised and renal function improved.

Discussion: The patient demonstrated an Evans-like presentation likely triggered by post-viral immune dysregulation. Although schistocytes raised concern for thrombotic microangiopathy (TMA), the absence of neurologic findings, progressive renal failure, and the need for plasma exchange or complement blockade made TMA unlikely. Unlike most similar reports requiring IVIG or plasmapheresis, this case resolved fully with steroids.

Conclusion: This case highlights the need to consider immune cytopaenias in otherwise healthy individuals following influenza A and supports early steroid intervention.

KEYWORDS

Influenza A, thrombocytopenia, haemolytic anaemia, Evans syndrome, immune cytopenia

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LEARNING POINTS

- Influenza A can precipitate Evans-like immune cytopaenias even in previously healthy young adults, a population not typically considered at risk for such haematologic complications.
- The presence of schistocytes can raise concern for thrombotic microangiopathy, but the absence of progressive renal or neurologic involvement should prompt consideration of immune-mediated cytopaenias.
- Early recognition and corticosteroid initiation can result in complete recovery without intravenous immunoglobulin (IVIG), rituximab or plasmapheresis, underscoring the importance of maintaining diagnostic vigilance in post-viral cytopaenias.

INTRODUCTION

Influenza A is well known for its respiratory manifestations, but in rare cases it can trigger serious extrapulmonary complications, including haematologic abnormalities[1,2]. While transient cytopaenias may occur during acute infection, severe immune-mediated syndromes such as immune thrombocytopenia (ITP), autoimmune haemolytic anaemia (AIHA) and thrombotic microangiopathies are typically seen in children, the elderly or immunocompromised individuals[3,4]. These cases often require high-dose corticosteroids, intravenous immunoglobulin (IVIG) or even plasmapheresis, and are commonly associated with additional systemic complications such as renal failure, neurologic changes or disseminated intravascular coagulation[5].

In contrast, immune-mediated cytopaenias in otherwise healthy young adults following influenza A are rarely reported. Cases that feature both thrombocytopenia and haemolysis mimicking Evans syndrome, but resolve with a short course of corticosteroids alone, are virtually absent from the literature. We present a unique case of a 22-year-old male with post-influenza A thrombocytopenia and haemolytic features who recovered fully after brief steroid therapy. This case highlights a potentially under-recognised immune complication of influenza A and fills an important gap in current understanding of post-viral haematologic responses in low-risk populations.

CASE DESCRIPTION

A 22-year-old previously healthy male presented to the emergency department with a three-day history of malaise, fever (peaking at 39.4°C) and a non-productive cough. He also reported non-bloody diarrhoea and, notably, the new onset of gross haematuria. He denied any chest pain, dyspnoea, bleeding from other sites, haematochezia, melena or mucocutaneous petechiae. However, on examination petechiae were noted on his hard palate and dried blood was present on cracked lips, which he attributed to lip-picking.

Initial laboratory evaluation revealed a platelet count of 7 × 10^9/l, a serum creatinine of 2.44 mg/dl and a creatine phosphokinase (CPK) of 120 U/l. A nasopharyngeal swab was positive for influenza A by polymerase chain reaction test. Haemoglobin was 11.7 g/dl with haematocrit of 34.1%; the reticulocyte count was elevated at 3.7% (absolute 99 ×10^9/l). Inflammatory markers were notable for lactate dehydrogenase (LDH) >1,900 U/l, ferritin ~6,000 ng/ml, CRP 1.4 mg/dl, serum iron 181 µg/dl, transferrin saturation 60% and D-dimer 1.71 µg/ml fibrinogen equivalent units. Haptoglobin was <30 mg/dl, and direct antiglobulin (Coombs) test was negative. Peripheral smear showed anisopoikilocytosis with fragmented red cells, including schistocytes (helmet and triangular cells), consistent with haemolysis *(Fig. 1)*. There was no clinical or laboratory evidence of disseminated intravascular coagulation or overt thrombotic microangiopathy.

The differential diagnosis included influenza-triggered immune thrombocytopenia (ITP), Evans syndrome and thrombotic microangiopathies such as atypical haemolytic uremic syndrome (aHUS) or viral-associated TMA. The patient was started on oseltamivir and IV dexamethasone (40 mg/day), along with supportive care including IV fluids. Two units of platelets were transfused on admission due to severe thrombocytopenia.

Over the next four days, the patient showed steady improvement. Haematuria resolved, creatinine gradually trended downwards, and platelets increased to 45 × 10^9/l by day two and stabilised in the 30–45 range by discharge. Inflammatory markers began to normalise, and renal function improved without the need for dialysis or additional

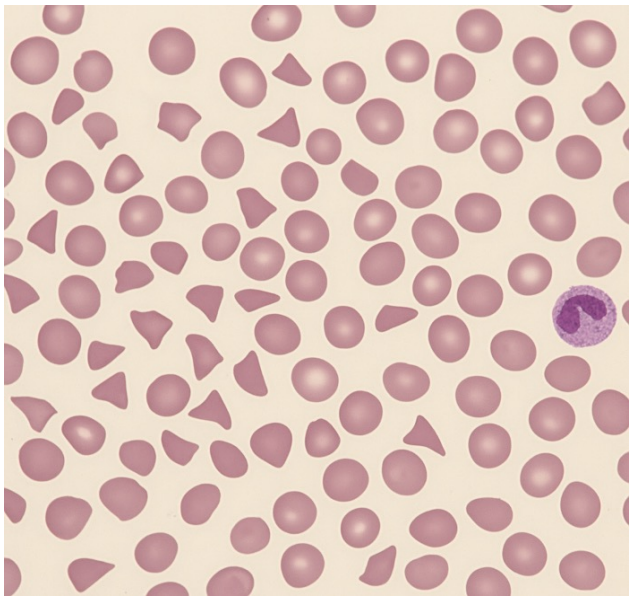


Figure 1. Peripheral blood smear on hospital day one demonstrating marked anisopoikilocytosis and schistocytes (helmet and triangular red cells). One neutrophil with normal morphology is present.

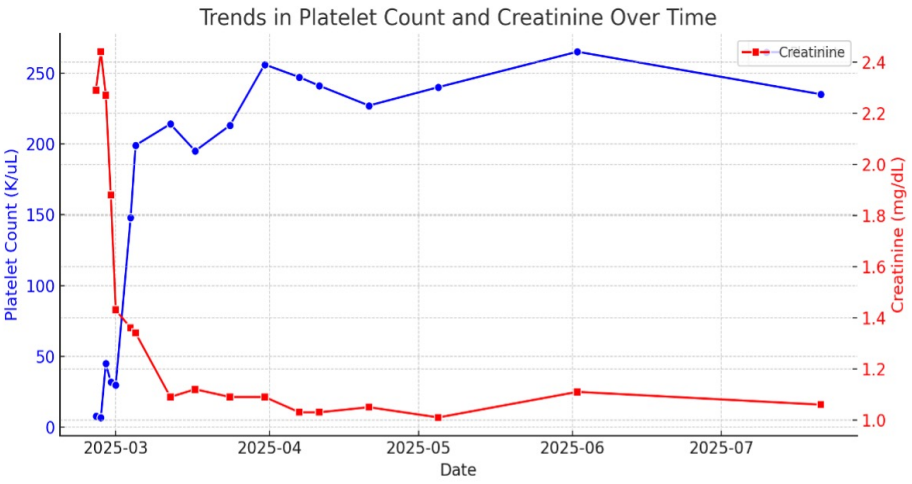


Figure 2. Trend in platelet count and serum creatinine over time following influenza A infection.

interventions. The patient was discharged on hospital day five in a stable condition, having completed four days of corticosteroids, with outpatient haematology follow-up.

At follow-up one week later, the patient reported complete clinical recovery. He was afebrile, without bleeding, and had resumed work. Platelet counts had normalised, and renal function had returned to baseline *(Fig. 2)*. Haematology assessment confirmed viral ITP as the likely aetiology of his thrombocytopenia, with supportive findings of transient normocytic anaemia, elevated ferritin and a haemolytic pattern on a smear. Given his response to dexamethasone and absence of ongoing cytopaenias, no further immunosuppressive therapy or invasive workup was pursued.

DISCUSSION

Haematologic complications associated with influenza A infection, though rare, have been increasingly recognised in the literature. The most commonly reported manifestations include transient leukopenia, thrombocytopenia and, in severe cases, microangiopathic haemolytic anaemias such as thrombotic thrombocytopenic purpura (TTP) and haemolytic uremic syndrome (HUS)[6,7]. However, most of these cases occur in the context of significant comorbidities, paediatric populations or immunocompromised states[8]. Our case of a previously healthy 22-year-old male who developed simultaneous thrombocytopenia, haematuria, elevated inflammatory markers and evidence of haemolysis after influenza A infection adds a novel dimension to this spectrum. In our case, the presence of schistocytes on the peripheral smear naturally raised concern for thrombotic microangiopathy, including atypical HUS. However, several features argued against this, such as the patient had no neurologic abnormalities, no progressive renal failure and no evidence of coagulopathy. Platelet counts and creatinine improved rapidly with corticosteroids alone, and neither plasma exchange nor complement blockade, which are typically required in aHUS, was required. The overall clinical trajectory, with rapid improvement on corticosteroids alone, was inconsistent with thrombotic microangiopathy.

While immune thrombocytopenia (ITP) is a known post-viral autoimmune phenomenon, its association with influenza A is largely underreported in adults[9]. Moreover, when ITP is accompanied by features of haemolysis, such as in Evans syndrome, more aggressive interventions such as intravenous immunoglobulin (IVIG), rituximab or even plasma exchange are often required[5,10]. Our patient, however, demonstrated an Evans-like presentation with thrombocytopenia and haemolytic features but a negative Coombs test. He responded well to a short, four-day course of corticosteroids and supportive care alone, with no recurrence during follow-up. This favourable trajectory highlights a potentially self-limited, influenza-triggered immune cytopenia with Evans-like features, not previously well-characterised in healthy young adults.

In terms of pathophysiology, post-viral cytopaenias are thought to be mediated through several mechanisms, such as molecular mimicry leading to autoantibody production, cytokine-induced marrow suppression and direct viral activation of immune effector cells[11]. Influenza A, in particular, has been shown to enhance interferon and TNF-alpha responses that may contribute to haematopoietic dysregulation. Elevated ferritin and LDH levels in our patient suggest an inflammatory milieu, although there was no evidence of macrophage activation syndrome or haemophagocytic lymphohistiocytosis. Interestingly, despite a negative Coombs test, the peripheral smear and LDH pattern pointed to subclinical haemolysis, possibly immune-mediated or inflammation-driven.

To our knowledge, this is one of the few reports detailing a transient Evans-like syndrome in a healthy adult male following influenza A, without organ failure or need for escalation to second-line therapies such as immunosuppressives or plasmapheresis. This case highlights the importance of recognising viral infections as potential triggers for haematologic dysregulation, even in populations traditionally considered low risk. As influenza remains endemic and seasonal epidemics continue, clinicians should maintain a high index of suspicion for immune-mediated cytopaenias in post-viral settings. Early identification and corticosteroid initiation may prevent complications and reduce unnecessary interventions.

CONCLUSION

Influenza A can trigger Evans-like immune cytopaenias even in otherwise healthy young adults, a population rarely described in existing literature. This case underscores the importance of considering autoimmune complications in the differential diagnosis of post-viral fatigue and cytopaenias and highlights the importance of early recognition and potential for full recovery with timely corticosteroid therapy.

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