

When Vasculitis Is Not Vasculitis: A Case Report in Which Dynamic Immunophenotyping Revealed Hidden Angioimmunoblastic T-cell Lymphoma

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Abstract

Background/Aim: Angioimmunoblastic T-cell lymphoma (AITL) is a rare peripheral T-cell lymphoma characterized by diverse and aggressive clinical manifestations that frequently mimic autoimmune disorders. Cutaneous presentations are common and may lead to diagnostic delay.

Case Report: A 67-year-old woman first presented with non-blanchable skin rash, with normal platelet count and negative autoimmune markers. The rash improved with corticosteroids but recurred two months later, accompanied by fever, night sweats, limb edema, diarrhea, and cervical lymphadenopathy. Skin biopsy reported vasculitis and panniculitis, as direct immunofluorescence was compatible with IgA vasculitis. Subsequent laboratory tests revealed atypical lymphocytes, Coombs-positive anemia, thrombocytopenia, and detected Epstein-Barr virus DNA. Computed tomography showed new splenomegaly and periaortic lymphadenopathy. Lymph node biopsy confirmed AITL.

continued

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Although CHOP chemotherapy was planned after staging, the patient rapidly deteriorated and died of septic shock. Serial peripheral blood flow cytometry at admission, post-splenectomy, and follow-up showed dynamic immunophenotypic changes: reductions in exhaustion and senescence markers as well as activated regulatory T cells after splenectomy; and later upregulation of exhaustion markers on naïve T cells.

Conclusion: This case illustrates the misleading presentation as immunoglobulin A (IgA) vasculitis and rapid progression of AITL. While vasculitis is accompanied by cytopenia, lymphadenopathy, or aggressive clinical course, early lymph node biopsy is essential for timely diagnosis.

Keywords: Angioimmunoblastic T-cell lymphoma, immunoglobulin A, vasculitis, case report.

Introduction

Immunoglobulin A (IgA) vasculitis is the most common form of systemic vasculitis in children. Typical symptoms include non-thrombocytopenic purpura, arthralgia or arthritis. Renal involvement is more frequent and severe in adults, while gastrointestinal symptoms are less common. Laboratory findings are generally nonspecific but provide information for assessment of organ involvement and exclusion of mimics. Although there is no international consensus, the diagnosis is primarily based on clinical presentation, which require the presence of palpable purpura without thrombocytopenia, plus one of arthritis/arthralgia, abdominal pain, or renal involvement (1, 2). Notably, there are several conditions that can closely mimic the clinical and pathological features of IgA vasculitis, as infections, drug reactions, autoimmune diseases, and malignancies. Therefore, all adult patients with suspected IgA vasculitis should be evaluated for secondary causes, including underlying malignancy (2).

Angioimmunoblastic T-cell lymphoma (AITL) is a rare hematologic malignancy characterized by variable presentations and aggressive clinical course. It is mostly seen in older adults, with a median age at diagnosis older than 60 years (3, 4). Skin involvement is seen in approximately 50% of cases. Dermatological features are highly variable and may include morbilliform maculopapules, infiltrated and noninfiltrated purpura, nodules, erythroderma, urticarial plaques, and bullous eruptions. The rash often involves the trunk and extremities and may mimic vasculitis, drug

eruptions, viral exanthems, or connective tissue disorders, making early diagnosis challenging (5-7). Other clinical presentations include generalized lymphadenopathy, constitutional symptoms, hepatosplenomegaly, skin rash, pleural effusions, and ascites. Common laboratory findings are anemia, thrombocytopenia, elevated lactate dehydrogenase, polyclonal hypergammaglobulinemia, hypocomplementemia, hypoalbuminemia, hyponatremia, and positive Coombs test (8). Renal involvement was documented in recent reports (9-11).

IgA vasculitis as an initial manifestation of AITL is rare and may lead to diagnostic delay. Given the overlapping clinical and pathological features between these two conditions, recognition of atypical presentations is essential. We report a case of AITL initially presenting with IgA vasculitis-like features, highlighting the diagnostic challenges, rapid disease progression, and the importance of early histologic confirmation. This study was approved by the Institutional Review Board (IRB) of Tri-Service General Hospital, National Defense Medical University (NDMU), Taiwan (IRB No. C20240519; July 31, 2024; and IRB No. B202615070; April 14, 2026). All procedures complied with institutional guidelines and the ethical standards of the Declaration of Helsinki and its amendments.

Case Report

The 67-year-old woman presented to the internal medicine clinic with skin rashes over the trunk and four limbs for three days. Laboratory data showed normal platelet count

Table I. Laboratory data as disease progressed, and after splenectomy.

| Investigation | Results | | | | | Reference range |
|------------------------|---------------------|------------------|----------------------|----------------------|-----------|-----------------|
| | 2025/6/13 Clinic | 2025/8/27 ADM | 2025/9/5 1st week | 2025/9/10 Post-OP | 2025/10/2 | |
| Creatinine (mg/dl) | 0.72 | 0.8 | 1.3 | 3.8 | 3.8 | 0.5-0.9 |
| CRP (mg/dl) | 1.11 | 6.83 | 6.68 | 1.73 | 1.64 | 0.80 |
| WBC ($10^3/\mu$) | 7.96 | 12.25 | 10.08 | 13.32 | 4.20 | 4.50-11.00 |
| Neutrophil (%) | 79.6 | 90.7 | 86.5 | 73.7 | 37.4 | 40.0-74.0 |
| Lymphocyte (%) | 16.1 | 5.6 | 5.2 | 16.2 | 48.5 | 19.0-48.0 |
| Hb (g/dl) | 13 | 9.5 | 7.3 | 7.7 | 8.1 | 12.0-16.0 |
| PLT ($10^3/\mu$ l) | 151 | 56 | 15 | 43 | 91 | 150-400 |
| MAB/CREA Ratio (ug/mg) | 0.1 | 49.1 | | | | 30 |
| C3 (mg/dl) | 130.77 | 75.1 | | | | 87.0-200.0 |
| C4 (mg/dl) | 35.33 | <8.0 | | | | 19.0-52.0 |

ADM: Admission; OP: operation; CRP: C-reactive protein concentration; WBC: white blood cell; Hb: hemoglobin; PLT: platelet count; MAB/CREA: microalbumin/creatinine; C3: complement component 3; C4: complement component 4.

($151 \times 10^3/\mu$ l), and no specific autoimmune marker was found (Table I). She had past medical history of type 2 diabetes, and surgical history of right upper lung wedge resection for lung cancer and partial thyroidectomy for thyroid cancer. There was no family history of malignancy or autoimmune disorder. The skin rashes gradually disappeared after systemic steroids were prescribed. Two months later, the skin rash recurred with fever, night sweating, dyspnea, poor appetite, limb edema, fatigue, mild abdominal pain and diarrhea. She reported no arthralgia, unexplained weight loss, recent vaccination, or use of new medications. She was admitted for further evaluation. Physical examination was notable for multiple enlarged lymph nodes over the neck, limb edema, and multiple erythematous to purpuric non-blanchable macules and papules on the trunk and four limbs (Figure 1A).

The white blood cell count was elevated with neutrophilia, anemia, and thrombocytopenia (platelet $56 \times 10^3/\mu$ l). The serum C-reactive protein concentration (CRP) (6.83 mg/dl), erythrocyte sedimentation rate (47 mm/h), immunoglobulin E (IgE) (1,787.0 IU/ml), and ferritin (909 ng/ml) levels were elevated, as the serum complement concentration decreased. Anti-double strand DNA, and antineutrophil cytoplasmic antibody were not

detected, and cryoglobulin was not elevated. Hepatitis B virus (HBV) surface antigen and hepatitis C virus (HCV) immunoglobulin M (IgM) were negative. Rheumatoid factor IgM (9.5 IU/ml), anti-cardiolipin IgM (72.2 PL), and anti-cardiolipin immunoglobulin G (IgG) (11.17 PL) were elevated but were not specific in this situation. Urine analysis showed proteinuria and albuminuria.

Chest x-ray revealed bilateral pleural effusion. Chest-computed tomography (CT) further reported bilateral miliary nodules and enlarged lymph nodes at the bilateral axillary and perihilar regions. An echocardiogram showed no evidence of endocarditis. Abdominal ultrasound showed splenomegaly. Skin biopsy reported vasculitis and panniculitis. Direct immunofluorescence showed IgM and C3 focal deposit, weak IgA and C1q focal deposit, and IgG negative. Further examination showed galactose-deficient IgA1 (GdIgA1) [using the KM55 enzyme-linked immunosorbent assay (ELISA) method] strongly positive. Based on the initial non-thrombocytopenic purpura, following abdominal pain, proteinuria, skin biopsy result, and GdIgA1 positivity, the initial diagnosis was IgA vasculitis with secondary thrombocytopenia. Differential diagnosis included immune thrombocytopenia, hemolytic disorders, tuberculosis, infection-associated

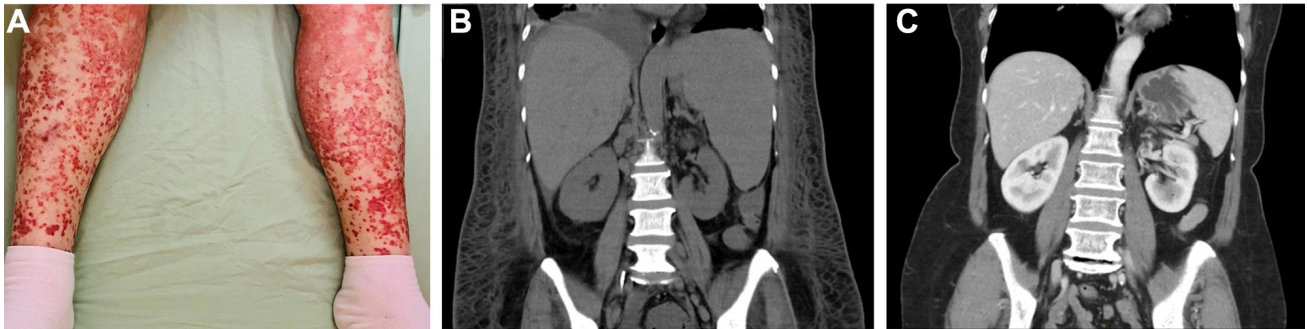


Figure 1. Cutaneous vasculitic rash and interval development of abdominal pathology on serial computed tomography. (A) Erythematous and purpuric non-blanchable maculopapular rash over the lower limbs upon admission. (B) Abdominal computed tomography on September 9, 2025, demonstrating panniculitis, ascites, hepatomegaly, splenomegaly (17.6 cm), and periaortic lymphadenopathy. (C) Abdominal computed tomography on January 23, 2025, showing no significant abnormalities.

thrombocytopenia, hypersplenism with platelet sequestration, and lymphoma.

Prednisolone 5 mg and hydroxychloroquine 200 mg daily were initially prescribed, and skin rash faded rapidly. During the second week after admission, the patient developed respiratory distress as chest X-ray revealed a notable increase in bilateral pleural effusion. Meanwhile, both hemoglobin and platelet levels declined gradually, and serum creatinine levels increased rapidly. Further exams revealed no blast or schizocytes on the blood smear, normal haptoglobin (103.9 mg/dl), and mild elevated lactate dehydrogenase (326 U/l). Direct (both IgG and C3d) and indirect Coombs' tests returned positive results. The platelet antibody was negative. Virus polymerase chain reaction (PCR) test detected herpes simplex virus (HSV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV). Both acid-fast stains and tuberculosis (TB) PCR showed negative findings. The following abdominal CT showed splenomegaly and enlarged periaortic lymph nodes (Figure 1B). Notably, chest, abdominal, and pelvic CT performed six months earlier had shown neither splenomegaly nor lymphadenopathy (Figure 1C).

In response to these findings, the patient was treated with albumin and furosemide. Meanwhile, ganciclovir, methylprednisolone, platelet transfusion, thrombopoietin (TPO) therapy, and intravenous immunoglobulin (IVIG), were prescribed. However, despite these measures, her

conditions did not improve, and intermittent dialysis was initiated. Given the rapid progression of the disease and a sudden decline in the patient's condition, an emergency splenectomy and excisional biopsy of the lymph nodes were performed simultaneously. Histopathological analysis of the perisplenic lymph node showed effacement of the nodal architecture by monotonous small-to-medium sized T cells with high mitotic activity, accompanied by proliferative vessels. Immunohistochemistry showed neoplastic cells positive for cluster of differentiation 2 (CD2), cluster of differentiation 3 (CD3), cluster of differentiation 4 (CD4), cluster of differentiation 5 (CD5), cluster of differentiation 7 (CD7), cluster of differentiation 10 (CD10), cluster of differentiation 43 (CD43), and EBV-encoded RNA. A diagnosis of angioimmunoblastic T cell lymphoma was made.

After surgery, CRP levels declined immediately, fever subsequently subsided, and platelet transfusion requirements decreased, with the interval was increased from daily to every 3-4 days. Given the concurrent administration of ganciclovir, the observed clinical improvements cannot be attributed solely to the surgical intervention. Positron emission tomography (PET)/CT and bone marrow biopsy demonstrated multiple nodal sites of involvement above and below the diaphragm, with evidence of disseminated bone marrow infiltration. The disease was classified as stage IV based on the American Joint Committee on Cancer (AJCC) 8th edition criteria.

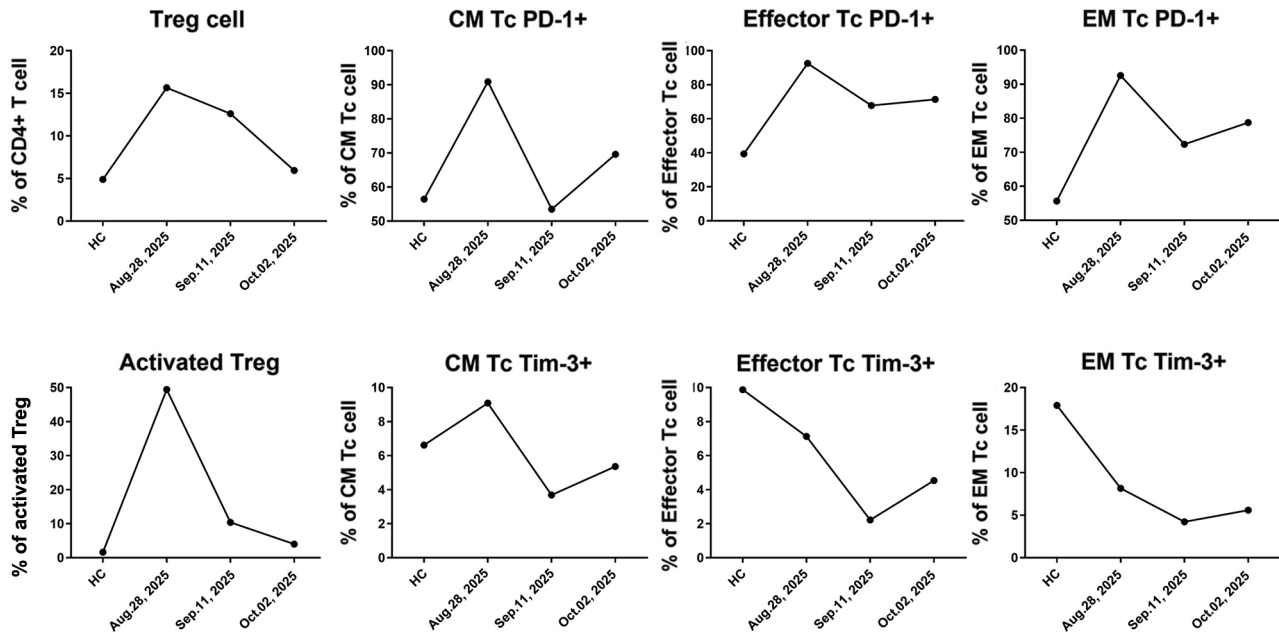


Figure 2. Reduction of exhaustion markers in cytotoxic T-cell subsets and dynamic changes in regulatory T cells following splenectomy. Longitudinal changes in regulatory T cell percentages and exhaustion markers in T cytotoxic cells. The panels show decreases in the percentages of programmed cell death protein 1 (PD-1)+ and T-cell immunoglobulin and mucin domain-containing protein 3 (Tim-3)+ T cytotoxic cells (Tc) across the central memory (CM), effector, and effector memory (EM) subsets after the splenectomy. HC: Healthy controls; data obtained from healthy volunteers.

Chemotherapy was postponed due to neutropenia. In addition, blood culture and abdominal drainage yielded *Klebsiella aerogenes*. The patient developed intra-abdominal infection and died 66 days after admission.

To evaluate phenotypic and functional changes in immune cell populations, flow cytometric analyses of peripheral blood were performed at three time points: admission (August 28, 2025), post-splenectomy (September 11, 2025), and follow-up (October 2, 2025). Following splenectomy, memory and effector T cells demonstrated reduced expression of exhaustion markers, including programmed cell death protein 1 (PD-1), T-cell immunoglobulin and mucin domain-containing protein 3 (TIM-3), as well as senescence markers, including killer cell lectin-like receptor G1 (KLRG1) and fas cell surface death receptor (Fas). In parallel, B-cell subsets showed decreased cluster of differentiation 21 (CD21) expression and increased Fas and human leukocyte antigen-DR isotype (HLA-DR) expression (Figure 2, Figure 3 and

Figure 4). At follow-up, naïve helper and cytotoxic T cells exhibited increased PD-1 and TIM-3 expression (Figure 5), coinciding with declining white blood cell counts, relative lymphocyte expansion, and the onset of severe intra-abdominal infection.

Discussion

Leukocytoclastic vasculitis with IgA deposition has been reported in patients with AITL (12, 13). AITL originates from follicular helper T cells, which play a central role in B-cell activation and immunoglobulin class switching. Malignant T-follicular helper (TFH) cells may promote aberrant B cell activation through cytokines such as interleukin-21 (IL-21) and transforming growth factor- β 1 (TGF- β 1), potentially leading to excessive or dysregulated IgA production (14). Although the underlying mechanism remains unclear, the dysregulated IgA may predispose to immune-complex deposition in small vessels and develop IgA vasculitis-like

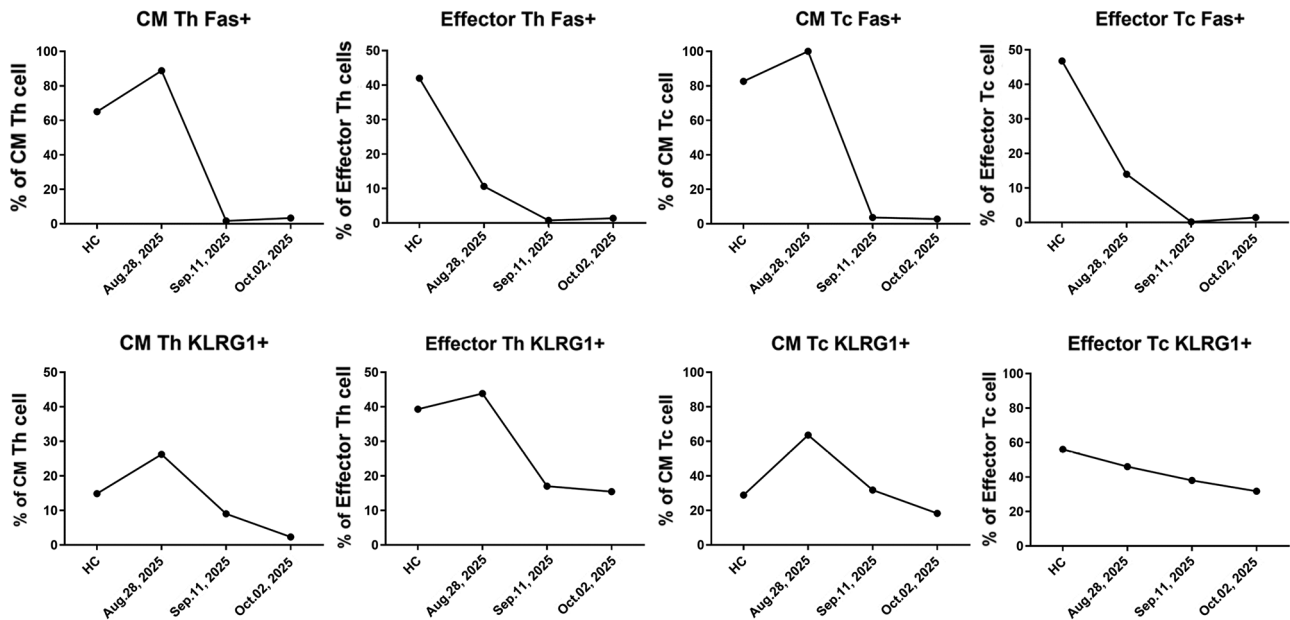


Figure 3. Decreased expression of senescence markers on central memory (CM) and effector T-cell subsets after splenectomy. Longitudinal changes in senescence markers in T helper cells and T cytotoxic cells. The panels demonstrate decreases in the percentages of fas cell surface death receptor (Fas)+ and killer cell lectin-like receptor G1 (KLRG)+ cells among CM and effector T cytotoxic (Tc) cells, as well as CM and effector T helper (Th) cells, following splenectomy. HC: Healthy controls; data obtained from healthy volunteers.

manifestations. Early diagnosis of AITL may be difficult due to its rapid progression and diverse presentation. While cutaneous manifestations occur in nearly half of AITL cases and may be the initial presentation (6), skin biopsies may demonstrate vasculitis in the absence of cytologic atypia (15). In this context, identification of clinical signs such as generalized lymphadenopathy, constitutional symptoms, polyclonal hypergammaglobulinemia, and autoimmune manifestations, along with timely lymph node biopsy, is critical for accurate diagnosis.

In our patient, the initial presentation was highly compatible with IgA vasculitis. She developed a purpuric rash, abdominal pain, proteinuria, and her skin biopsy revealed leukocytoclastic vasculitis with IgA and complement deposits. The detection of galactose-deficient IgA1 further reinforced the diagnosis. These findings are characteristic of IgA vasculitis and mislead the initial clinical impression. In the subsequent weeks, our patient experienced rapid deterioration with fever, night sweats,

generalized lymphadenopathy, splenomegaly, pleural effusion, aggressive thrombocytopenia, and progressive renal failure. Importantly, the CT scan performed six months prior to admission and the laboratory evaluation at the internal medicine clinic showed no specific abnormalities. These findings may explain why nearly 90% of AITL is diagnosed at an advanced stage (3, 4). As in this case, the clinical stage was IV at diagnosis.

Serial flow cytometry revealed phased changes in exhaustion and regulatory markers that paralleled the patient's clinical course. Splenectomy was associated with transient reductions in exhaustion and senescence markers, suggesting that removal of the spleen may have altered immune regulation and temporarily alleviated T-cell dysfunction. Concurrently, B-cell subsets demonstrated redistribution, with decreased CD21 and increased Fas/HLA-DR expression, reflecting redistribution and gradual restoration of immune homeostasis. At follow-up, elevated PD-1 and TIM-3 expression in peripheral Naïve helper T cells

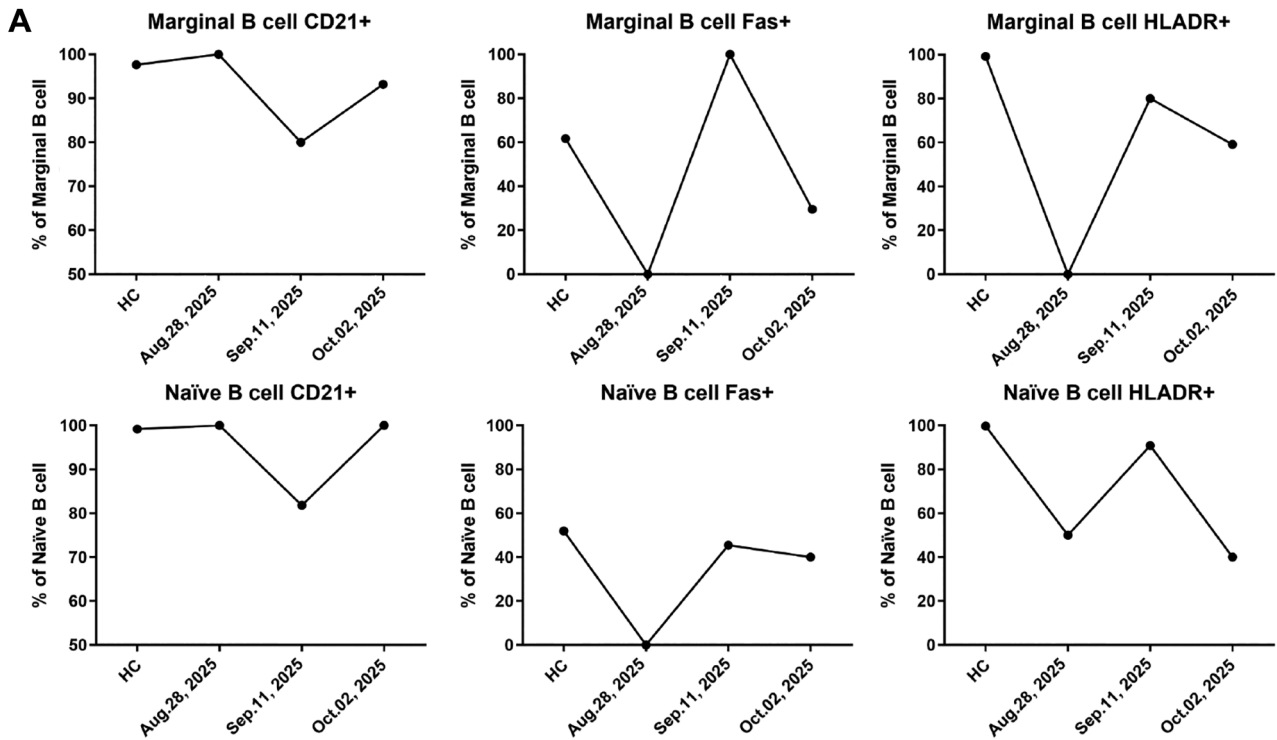


Figure 4. Continued

and cytotoxic T cells were noted (Figure 4). Concurrently, the patient exhibited a decline in white blood cell counts with relative lymphocyte expansion and subsequently developed a severe intra-abdominal infection. In chronic infection and malignancy, sustained upregulation of inhibitory receptors is commonly associated with T-cell exhaustion, and may be related to impaired infection control and tumor surveillance (16). Although a direct causal relationship cannot be established, the temporal concurrence of immunophenotypic changes and clinical deterioration may reflect an evolving exhaustion phenotype that coincided with the patient’s unfavorable outcome.

Evidence regarding the role of splenectomy in AITL remains limited. Previous studies have reported reduction of transfusion requirements in advanced hematologic malignancies following the procedure (17). Although the improvement could not be solely attributed to surgical intervention, our patient’s CRP levels decreased, fever resolved, and platelet transfusion

intervals were prolonged after surgery. Regarding the immunophenotyping findings, activated regulatory T cells were elevated at baseline, and antigen-experienced cytotoxic T cells exhibited high baseline PD-1 expression. Following splenectomy, a transient reduction in PD-1 and TIM-3 expression was observed (Figure 2). In parallel, animal studies have reported that splenectomy may attenuate immune cell exhaustion in septic mice, potentially through reducing regulatory T-cell expansion and modulating both PD-1 and TIM-3 axis, with an associated improvement in prognosis (18). Meanwhile, we detected a decline in T cell senescence markers, including KLRG1 and Fas, on T cells collected following splenectomy (Figure 3). These findings support the role of spleen in the regulation of T cell exhaustion and senescence (18, 19). Nevertheless, splenectomy carries inherent risks, including heightened susceptibility to infection and impaired tumor surveillance, as illustrated by the patient’s subsequent clinical course. This report has

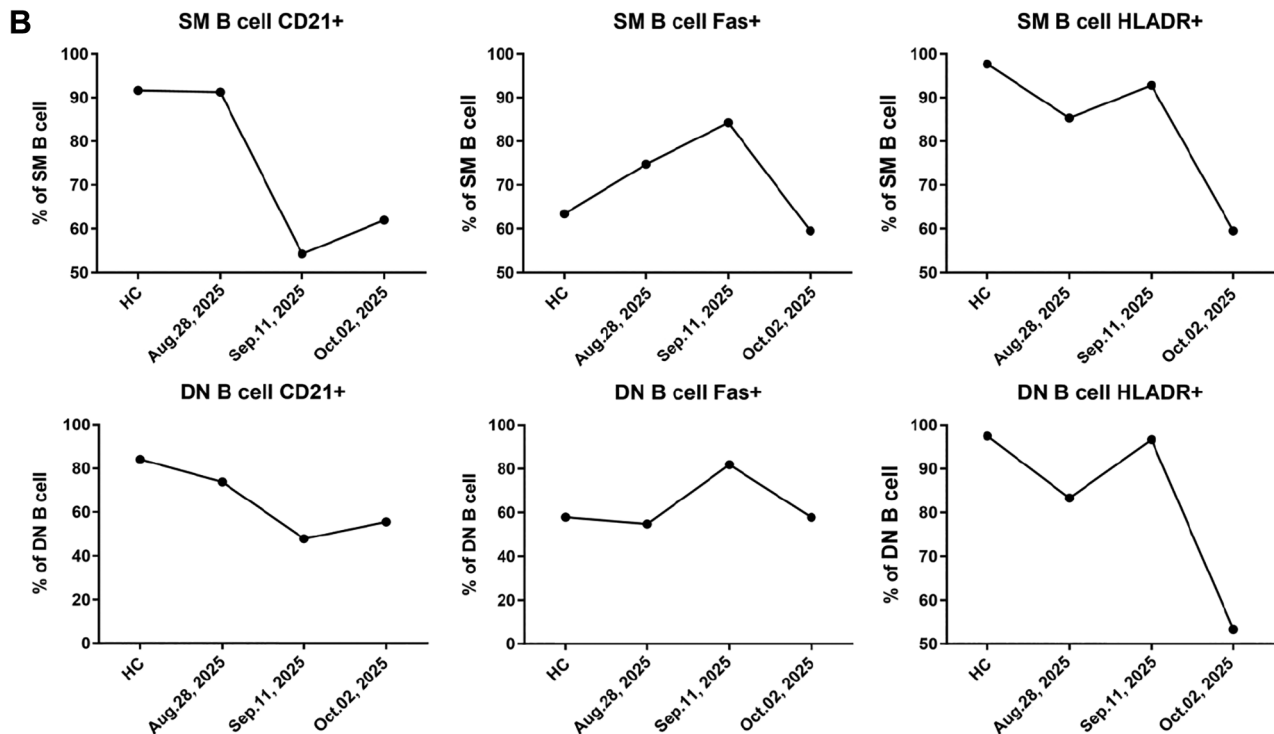


Figure 4. Dynamic alterations in B-cell phenotypes and activation markers following splenectomy. (A) Longitudinal changes in B cell phenotypes. Among marginal zone and naïve B cell subsets, cluster of differentiation 21 (CD21) expression decreases after splenectomy and subsequently increases during follow-up. In contrast, fas cell surface death receptor (Fas) and human leukocyte antigen-DR isotype (HLA-DR) expression increase post-splenectomy but decline during follow-up. (B) Similar trends are observed across all examined B cell subsets. HC: Healthy controls; data obtained from healthy volunteers.

limitations. Serial flow cytometry data were unavailable at baseline and after chemotherapy, limiting interpretation of immunophenotypic changes related to the disease and splenectomy. Additionally, as a single-case observation, these findings are exploratory and not generalizable.

Conclusion

This case highlights the diagnostic challenge of AITL presenting with IgA vasculitis-like features, which may delay recognition of the underlying malignancy. The patient's course demonstrates the complex immune dysregulation of AITL, including dynamic immunophenotypic changes associated with splenectomy and poor prognosis. Clinicians should maintain a high index of suspicion for lymphoma in patients with atypical or refractory IgA vasculitis, and early lymph node biopsy should be considered.

Conflicts of Interest

The Authors declare that they have no conflicts of interest or competing interests related to this study.

Authors' Contributions

LYL: Conceptualization, methodology, writing—original draft, writing review and editing. JWL: Conceptualization, methodology, writing—original draft, writing review and editing. YJH: Conceptualization, methodology, project administration, writing—review and editing. SWL: Conceptualization, methodology, writing—review and editing. TYH: Conceptualization, methodology, writing—review and editing. WLJ: Conceptualization, methodology, writing—review and editing. FCL: Conceptualization, investigation, supervision, writing—review and editing.

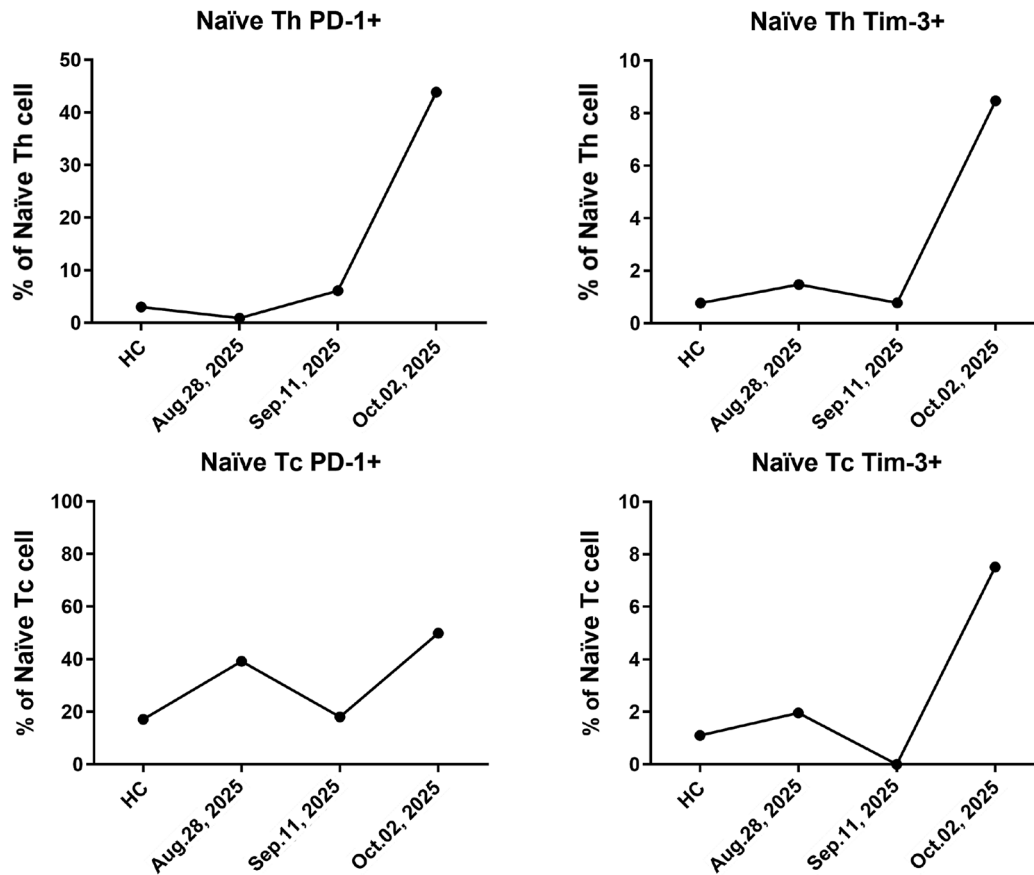


Figure 5. Progressive upregulation of T-cell immunoglobulin and mucin domain-containing protein 3 (Tim-3) and programmed cell death protein 1 (PD-1) on Naïve T helper (Th) and cytotoxic T cells during follow-up. Longitudinal changes in exhaustion markers in naïve Th cells and T cytotoxic cells. Panels demonstrate that the percentages of naïve Th cells Tim-3+, naïve Th cells PD-1+, naïve T cytotoxic cells (Tc) Tim-3+, naïve Tc cells PD-1+ increase during the follow-up period. HC: Healthy controls; data obtained from healthy volunteers.

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Artificial Intelligence (AI) Disclosure

During the preparation of this manuscript, a large language model (ChatGPT, OpenAI) was used solely for

language editing and stylistic improvements in select paragraphs. No sections involving the generation, analysis, or interpretation of research data were produced by generative AI. All scientific content was created and verified by the authors. Furthermore, no figures or visual data were generated or modified using generative AI or machine learning-based image enhancement tools.

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