Case report

Thymoma-associated aplastic anemia initially presenting as bicytopenia: a case report and therapeutic challenges

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Abstract:

Aplastic anemia (AA) is a condition characterized by bone marrow failure caused by an immune-mediated response involving cytotoxic T cells. While the etiology of AA is often idiopathic, it can also result from factors such as drugs, toxins and immunologic diseases. Thymomas, tumors located in the anterior mediastinal, are associated with various autoimmune conditions including AA. In this report, we present a case of severe aplastic anemia associated with thymoma, highlighting its clinical presentation, laboratory findings, and histopathologic features.

A 54-year-old Thai woman with a history of dyslipidemia presented with a 1-month history of chronic progressive dyspnea and significant weight loss. On examination, she had physical signs of anemia, petechiae, and a left lung mass. Laboratory tests revealed anemia with reticulocytopenia, thrombocytopenia and a normal total white blood cell count with a reversed neutrophil-to-lymphocyte ratio (absolute neutrophil count 1.4 x 10^9/L). Bone marrow studies demonstrated hypocellularity with absent megakaryocytes. A computed tomography scan identified a 12.7 cm anterior mediastinal mass, and a biopsy confirmed type AB thymoma. She received a diagnosis of thymoma-associated AA. Despite treatment with cyclosporine, eltrombopag, blood transfusions, and thymectomy, the patient succumbed to a severe infection 4 months after diagnosis.

Keywords : ● *Aplastic anemia* ● *Thymoma* ● *Thymectomy*

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รายงานผู้ป่วย

ภาวะไขกระดูกฝ่อร่วมกับเนื้องอกต่อมไทมัส มาเริ่มด้วยภาวะซีดและเกล็ดเลือดต่ำ: รายงานผู้ป่วยและความท้าทายในการรักษา

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บทคัดย่อ

โรคไขกระดูกฝ่อ (Aplastic anemia, AA) คือภาวะที่ไขกระดูกไม่สามารถสร้างเซลล์เม็ดเลือดได้ เกิดจากความผิดปกติของระบบภูมิคุ้มกันทีเซลล์ชนิดซิโตทอกซิก (cytotoxic T cells) โดยทั่วไปมักตรวจไม่พบสาเหตุ แต่อาจเกิดจากปัจจัยบางอย่างเช่น ยา สารเคมี หรือความผิดปกติในการทำงานของภูมิคุ้มกัน เนื้องอกต่อมไทมัส (thymoma) เป็นหนึ่งในก้อนเนื้องอกที่พบบ่อยในช่องอกส่วนหน้า พบว่าสัมพันธ์กับภาวะหรือโรคภูมิคุ้มกันผิดปกติ รวมไปถึงโรคไขกระดูกฝ่อ รายงานผู้ป่วยฉบับนี้นำเสนอกรณีของโรคไขกระดูกฝ่อที่เกี่ยวข้องกับเนื้องอกต่อมไทมัส โดยแสดงรายละเอียดของอาการแสดงทางคลินิก ผลการตรวจทางห้องปฏิบัติการ และลักษณะทางพยาธิวิทยา

ผู้ป่วยหญิงไทยอายุ 54 ปี มีประวัติโรคไขมันในเลือดสูง มาพบแพทย์ด้วยอาการหอบเหนื่อยเวลาออกแรงเป็นเวลา 1 เดือน ตรวจร่างกายพบภาวะซีด จุดเลือดออกใต้ผิวหนัง และอาการแสดงของก้อนที่ปอดข้างซ้าย ผลการตรวจทางห้องปฏิบัติการพบว่ามีภาวะโลหิตจาง เกล็ดเลือดต่ำ เม็ดเลือดขาวปกติแต่มีอัตราส่วนของนิวโทรฟิลต่อลิมโฟไซต์ที่กลับกัน ผลการตรวจไขกระดูกพบว่าไขกระดูกมีปริมาณเซลล์เม็ดเลือดลดลงและ ไม่พบเซลล์เมกะคารีโอไซต์ ผลการตรวจรังสีวิทยาพบก้อนเนื้องอกขนาด 12.7 เซนติเมตรที่ช่องอกส่วนหน้า ผลการตรวจชิ้นเนื้อเข้าได้กับเนื้องอกต่อมไทมัสชนิด AB ผู้ป่วยได้รับการวินิจฉัยว่าเป็นภาวะไขกระดูกฝ่อรุนแรงจากก้อนเนื้องอกต่อมไทมัส ได้รับการรักษาด้วยยา cyclosporine, eltrombopag การให้เลือดและส่วนประกอบของเลือด และการผ่าตัดเนื้องอกต่อมไทมัส อย่างไรก็ตามผู้ป่วยเสียชีวิตจากการติดเชื้อที่รุนแรงหลังจากวินิจฉัยเป็นเวลา 4 เดือน

คำสำคัญ : ● *โรคไขกระดูกฝ่อ* ● *เนื้องอกต่อมไทมัส* ● *การผ่าตัดต่อมไทมัส*

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Introduction

Aplastic anemia (AA) is characterized by bone marrow failure, with its pathophysiology primarily driven by an immune response, often involving cytotoxic T cells.[1,2] Thymomas are uncommon neoplasms derived from the epithelial cells of the thymus. However, they are among the most common mediastinal masses, accounting for up to 50% of anterior mediastinal masses.[3] Thymomas are frequently associated with various autoimmune diseases. The five most common associations include myasthenia gravis (MG), pure red cell aplasia (PRCA), lichen planus, Good syndrome and limbic encephalitis.[4]

The association between thymoma and AA is rare, AA occurring in 0 to 1.4% of thymoma cases.[5,6] Reported cases have a median age of 57 years (range: 12 to 75 years), with a slight male predominance (57.1%). Most patients present severe (SAA) or very severe AA (VSAA), which may develop as the initial manifestation of thymoma, shortly after thymoma resection, or years later. Flow cytometry analysis has revealed an inversion of the CD4/CD8 ratio in the blood and bone marrow in 80.0 and 62.5% of evaluated patients, respectively.[7] Thymectomy alone has proven insufficient to restore normal hematopoiesis.[7] However, patients with thymoma-associated AA (T-AA) exhibit treatment responses to first-line immunosuppressive therapy (IST), particularly anti-thymocyte globulin (ATG) combined with cyclosporine (CSA), with an overall response rate of 60 to 70%,[7] comparable to that observed in other forms of acquire AA.[8]

Case presentation

A 54-year-old Thai female with dyslipidemia managed with a controlled diet presented with exertional dyspnea, without associated orthopnea or paroxysmal nocturnal dyspnea. She reported increase pallor, left-sided chest tightness and significant unintentional weight loss from 56 to 47 kg within one month. At the hospital, she was afebrile with stable vital signs. Her respiratory rate and oxygen saturation were within normal limits. Physical examination revealed moderately pale conjunctivae, anicteric sclerae, petechiae on the palms and feet and small ecchymoses on the left arm. Respiratory examination revealed a midline trachea, dullness on percussion, decreased breath sounds and decreased vocal resonance over the left lung zone. The cardiovascular examination and other systems were unremarkable. Laboratory investigations revealed anemia with a hemoglobin (Hb) level of 7.3 g/dL, hematocrit (HCT) 22.0%, mean corpuscular volume (MCV) 81.2 fL, and red cell distribution width (RDW) 12.8%. The white blood cell (WBC) count was 4.9 $x10^9$/L, with a differential count of 28.9% neutrophils and 67.6% lymphocytes, yielding an absolute neutrophil count (ANC) of 1.4 $x10^9$/L. Platelets were markedly reduced to less than 2 $x10^9$/L. Reticulocytopenia was evident with a reticulocyte count of 0.1% and absolute reticulocyte count of 3 $x10^9$/L. The coagulation profile was unremarkable with a prothrombin time (PT) of 13.1 seconds (reference range: 10.1 to 12.6 seconds) and an activated partial thromboplastin time (PTT) of 23.4 seconds (reference range: 21.6 to 32.5 seconds). Blood chemistry, including renal and liver function tests, were unremarkable. Peripheral blood smear showed normochromic normocytic anemia, with decreased white blood cell and platelets, and a relatively increase in small mature lymphocytes. No leucoerythroblastic feature was observed. A bone marrow examination revealed hypocellular marrow with approximately 10% cellularity, no morphologic evidence of dysplasia, absence of megakaryocytes and a relative increased in small mature lymphocytes. Immunohistochemistry (IHC) identified these lymphocytes as T-cell lymphoproliferation, without aberrant loss of CD2, CD5 or CD7 (Figure 1). The differential diagnosis included severe aplastic anemia and T-large granular lymphocyte (T-LGL) leukemia. PCR-based analysis for clonal T-cell receptor gene rearrangements was negative, indicating the absence of clonality, which supported the diagnosis of severe aplastic anemia. Additional investigations revealed a normal female karyotype on chromosomal

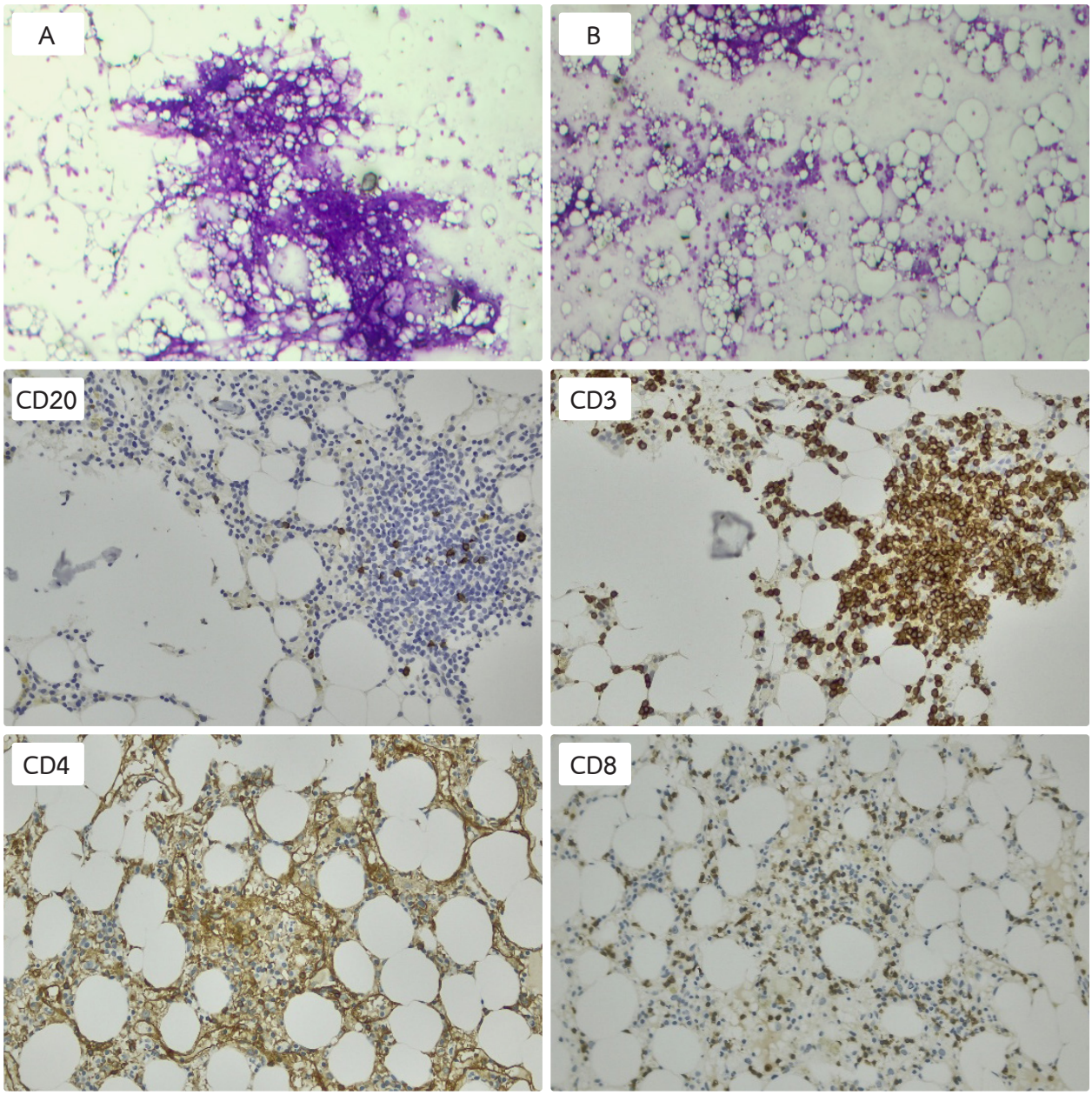

Figure 1 (A, B) Histopathologic examination of the bone marrow reveals a hypocellular marrow with an absence of megakaryocytes (A, B). Immunohistochemical analysis demonstrates T-cell lymphoproliferation, with positive expression of CD3, CD4, and CD8.

analysis, and flow cytometry for paroxysmal nocturnal hemoglobinuria (PNH) were also negative. A chest X-ray revealed a homogeneous opacity mass in the left lung. A computed tomography (CT) of the chest and abdomen demonstrated a large heterogeneous enhancing mass with intratumoral enhancing vessels at anterior aspect of left mid hemithorax, overall measuring 12.7x9.1x11.8 cm (Figure 2). No enlarged lymph nodes or hepatosplenomegaly were observed. A biopsy of the left intrathoracic mass revealed a neoplasm composed of mixed small lymphoid cells and mildly atypical round-to-oval cells. IHC demonstrated positivity for AE1/3 and PAX8, diffuse CD3 positivity and focal CD20 positivity, finding consistent with thymoma (histologic type AB). The patient received a diagnosis of thymoma-associated aplastic anemia (T-AA). Three weeks after diagnosis, disease progression was evident, with an absolute neutrophil count (ANC) of 0.34 $x10^9$/L and a platelet count below 2 $x10^9$/L. Initial treatment was commenced with cyclosporine A (200 mg/day) and eltrombopag (75 mg/

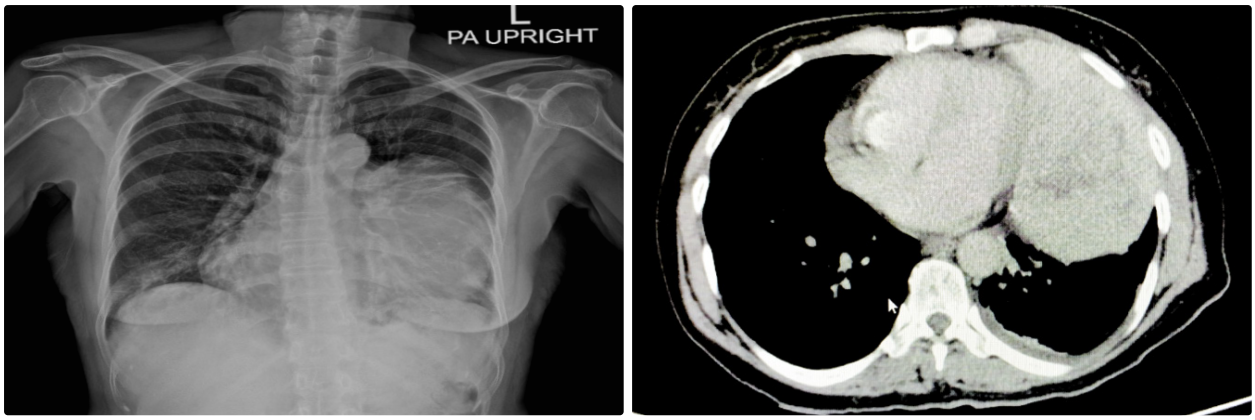

Figure 2 (A) Chest X-ray demonstrating a homogenous opacity in the left lung zone, indicative of a mass. (B) computed tomography (CT) of the chest revealing a large heterogeneous enhancing mass with intratumoral enhancing vessels (arrow) located in the anterior aspect of the left mid hemithorax, measuring 12.7 x 9.1 x 11.8 cm.

day). Antithymocyte globulin (ATG) was not administered due to the patient's frailty and clinical judgment regarding the high risk of fungal infection. She underwent total thymectomy one month after starting immunosuppressive therapy with a thrombopoietin receptor agonist (TPO-RA). At the time of surgery, her ANC was 0.21 $x10^9$/L and platelet count was 8 $x10^9$/L. Pre-operative management included temporary discontinuation of immunosuppressive therapy and TPO-RA and transfusions support to maintain a target platelet count above 80 $x10^9$/L and hemoglobin level above 7 g/dL. Estimated intraoperative blood loss was 500 mL, with no postoperative complication observed. IST and TPO-RA was resumed two weeks postoperatively. Four weeks after reinitiating treatment, mild hematologic improved, with ANC rising to 1.33 $x10^9$/L, although thrombocytopenia persisted (platelet count 7 $x10^9$/L). However, six weeks after immunosuppressive therapy and TPO-RA reinitiation, the patient developed multiple complications including upper gastrointestinal bleeding, pneumonia and respiratory failure. She ultimately succumbed to her condition four months following the initial diagnosis.

Discussion

Aplastic anemia (AA) is a rare, life-threatening disorder characterized by hematopoietic bone marrow failure and peripheral cytopenia. Its pathogenesis involves immune-mediated destruction of hematopoietic stem and progenitor cells (HSPCs), primarily driven by oligoclonally expanded cytotoxic $CD8^+$ T cells. In addition, dysregulation of $CD4^+$ T-cell subsets, with increased Th1 and Th17 responses contributes to elevated levels of IFN-γ and TNF-α, further suppressing hematopoiesis. Regulatory T cells (Tregs), although present, are functionally impaired and fail to suppress effector T-cell activity, leading to uncontrolled immune responses.[9] Flow cytometry on peripheral blood from 30 pediatric patients with aplastic anemia revealed a consistent inversion of the CD4:CD8 T-cell ratio. Patients with aplastic anemia had a mean CD4:CD8 ratio of approximately 0.65, compared with 1.2 among agematched healthy controls.[10] This imbalance in immune regulation underlies the destruction of HSPCs and results in bone marrow failure. Genetic predispositions, such as specific HLA haplotypes, may further promote autoreactive T-cell activation.[9]

Thymoma is the most common primary tumor of the mediastinum, derived from the thymic epithelium. The incidence is 1,000 cases per year, affecting males and females equally, with peak occurrence in individuals over 65 years of age.[11] The thymus is the site for T-cell maturation, playing a crucial role in adaptive immunity and contributing to various autoimmune disorders.[12] Notably MG occurs in approximately 63% of thymoma cases,[12] while PRCA is seen in about 8%.[12] The

association between thymoma and AA is rare, with an estimated occurrence in 0-1.4% of thymoma cases.[5] The immune dysfunction caused by thymic epithelium enhances the generation of mature $CD8^+$ T cells while reducing $CD4^+$ T cell production, this imbalance leads to the expansion of T-cell clone responsible for both humoral and cytotoxic immune responses, ultimately recognizing self-antigens and targeting self-reactive precursor cells in the bone marrow.[13] The pathogenesis of AA associated with thymoma seems to be linked to bone marrow suppression caused by dysregulated T-cell activity.[14,15] Differentiating T-AA from idiopathic AA based solely on clinical presentation is challenging, as both conditions share similar cytopenia-related symptoms and occur equally in males and females. Additionally, AA may present as the initial manifestation of thymoma, emerge shortly after thymoma resection, or develop years later.[7,16] However, the presence of symptoms related to an anterior mediastinal mass and abnormal chest X-ray findings may suggest T-AA. Additionally, T-AA tends to be more common among older patients compared with idiopathic AA.[7]

Thymoma is associated with aplastic anemia (T-AA) and other immune-mediated cytopenias including PRCA (56%), acquired agranulocytosis (14%), immune thrombocytopenia (ITP, 11%) and autoimmune hemolytic anemia (AIHA, 6%).[17] These conditions present lineage-specific findings: PRCA with anemia and reticulocytopenia; ITP with thrombocytopenia and bleeding; AIHA with hemolytic anemia, reticulocytosis, elevated LDH and positive DAT; and agranulocytosis with severe neutropenia. Differentiation from T-AA, presenting pancytopenia and hypocellular marrow, is critical for appropriate management.

The treatment of acquired AA is guided by its severity, as defined by the Camitta criteria[18] and age at presentation. Patients with SAA or VSAA are recommended to receive ATG-based IST combined with eltrombopag or undergo allogeneic hematopoietic stem cell transplantation (HSCT) from matched sibling donor (MSD). In contrast, patients with nonsevere aplastic anemia (NSAA) typically do not require treatment unless they develop transfusion dependency or progression to SAA, at which point a combination of ATG and CSA is recommended.[18]

The response to IST is typically delayed, beginning after an average of three to four months. In SAA, the six-month response rate is 50 to 70%, whereas In VSAA it is significantly lower, at 23%.[18] Long term survival is age-dependent with a 15-year overall survival (OS) rate of 89% for patients younger than 20 years, 81% for those aged 20 to 39 years, 55% for those age 40 to 60 years, and 32% for patients older than 60.[19] The addition of eltrombopag, a thrombopoietin receptor agonist (TPO-RA), has been associated with a significantly improved complete remission (CR) rate. As a result, the combination of eltrombopag to ATG and CSA in SAA/VSAA has been approved by the USFDA and several regulatory authorities worldwide.[18] Because response rates to a second ATG are lower, especially in refractory AA, alternate strategies, including HSCT should be considered among patients with good performance status.[18]

For thymoma, the standard treatment is complete surgical resection, serving as a crucial predictor of long term survival.[20] Data of a large French cohort of 35 patients with T-AA revealed that surgical resection was performed in 31 cases (88.6%), achieving thymoma remission among 30 patients (96.8%). However, thymoma progression occured among the four nonoperable patients. None of the patients experienced a hematologic response following thymectomy, While IST played a crucial role in achieving hematologic response. The overall response rate (ORR) of AA was 72.7% with CSA plus ATG, 76.9% with CSA alone and 100% with HSCT.[7]

Our reported case of T-AA presented anemic symptoms, petechiae, ecchymosis, a mediastinal mass and significant weight loss. Laboratory findings revealed bicytopenia with reticulocytopenia. Bone marrow aspiration and biopsy confirmed severe aplastic anemia, characterized by marked hypocellularity and the

absence of megakaryocyte. Histopathologic examination of the mediastinal mass identified a thymoma, histologic type AB. Despite receiving immunosuppressive therapy with adjusted doses of cyclosporin A and elthrombopag, along with thymectomy, the patient remained transfusion-dependent, requiring transfusions twice weekly. Hematologic remission was not achieved, ultimately resulting in death due to severe thrombocytopenia, upper gastrointestinal bleeding, pneumonia and septic shock. Apart from our case, one additional case of thymoma-associated aplastic anemia (T-AA) has been reported in Thailand. A 44-year-old female presented pancytopenia at the time of thymoma diagnosis. She was treated with IST using low dose CSA (2.5 mg/kg/day), achieving transfusion independence within two months of therapy initiation. Thymectomy was subsequently performed five months after starting IST.[21] Despite a similar severity of aplastic anemia, our case demonstrated a poorer clinical outcome. Despite the use of dual-agent immunosuppressive therapy, TPO-RA, and early thymectomy, hematologic remission was not achieved and transfusion dependence persisted. The patient also developed early life-threatening complications. These findings suggest that treatment should be individualized, and that early clinical stability and risk of complications may play a more critical role in determining outcomes than treatment intensity alone.

Conclusion

In summary, our case demonstrated the characteristic clinical, radiologic and histopathologic features of T-AA. Despite its rarity, recognizing key diagnostic features such as pancytopenia with reticulocytopenia, markedly hypocellular bone marrow and the presence of a thymoma is essential for accurate diagnosis. Although the association between thymoma and AA is uncommon, screening for thymoma at the time of AA diagnosis is warranted. Accurate diagnosis and timely management are essential for optimizing patient outcomes. The efficacy of thymectomy and IST in achieving hematologic remission varies. However, surgical resection of thymoma and HSCT from a matched sibling donor remain the preferred treatment strategies, particularly for patients under 50 years of age.

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