American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e952933
DOI: 10.12659/AJCR.952933

Received: 2026.01.25
Accepted: 2026.07.28
Available online: 2026.08.07
Published: 2026.XX.XX

Pure Red Cell Aplasia Following Neoadjuvant Docetaxel/Carboplatin/Trastuzumab (TCbH) in a 45-Year-Old Woman With Breast Cancer Successfully Treated With Cyclosporine

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1,2 Yuanyuan Chen*
ACDEF 3,4 Fang Chen*
BCD 4 Rongkui Luo
ACE 1,2 Xin Ye

1 Breast Department, The International Peace Maternity and Child Health Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, PR China
2 Shanghai Key Laboratory of Embryo Original Diseases, Shanghai, PR China
3 Department of Medical Imaging, Suzhou Kowloon Hospital, School of Medicine, Shanghai Jiao Tong University, Suzhou, Jiangsu, PR China
4 Department of Pathology, Zhongshan Hospital, Fudan University, Shanghai, PR China

* Yuanyuan Chen and Fang Chen contributed equally to this work

Corresponding Author: Xin Ye, Phone: 15216613178, e-mail: yexin1148@hotmail.com
Financial support: None declared
Conflict of interest: None declared

Patient: Female, 45-year-old
Final Diagnosis: Acquired PRCA after TCbH • invasive ductal carcinoma of the right breast (cT3N2M0, HER2-positive)
Symptoms: Severe anemia with hemoglobin dropping to 34.2 g/L • right breast mass • weakness in both lower limbs
Clinical Procedure: —
Specialty: Hematology
Objective: Rare disease

Background: Human epidermal growth factor receptor 2 (HER2)-positive breast cancer is commonly treated with docetaxel/carboplatin/trastuzumab (TCbH). While anemia is a common complication, progression to pure red cell aplasia (PRCA) is extremely rare. This report presents the case of a 45-year-old woman with right breast ductal carcinoma (cT3N2M0, HER2-positive) who developed PRCA following neoadjuvant TCbH and was effectively treated with cyclosporine.

Case Report: A 45-year-old woman with right breast invasive ductal carcinoma (cT3N2M0, HER2-positive) received neoadjuvant TCbH. After the sixth cycle, she developed progressive anemia refractory to erythropoietin, with hemoglobin (Hb) decreasing to 34.2 g/L. After transfusion, she underwent modified radical mastectomy with pathological complete response. Postoperatively, anemia worsened with marked reticulocytopenia (reticulocytes 3.8×10^9/L; 0.2%). Bone marrow examination revealed markedly reduced erythroid precursors. Acquired PRCA was diagnosed after excluding iron deficiency, megaloblastic anemia, hemolytic anemia, hematologic malignancies, autoimmune disorders, aplastic anemia, thymoma, and infections. Oral cyclosporine (100 mg twice daily) induced complete hematologic remission at 7 weeks and was tapered over 2 years. During the adjuvant trastuzumab/pertuzumab (HP) treatment, Hb remained ≥110 g/L. A heterozygous germline *BRCA1* mutation (c.788delG) was identified. At the 4.5-year follow-up, there is no recurrence of either breast cancer or anemia.

Conclusions: This case highlights PRCA as a severe complication of TCbH therapy. Reticulocytopenia with refractory anemia warrants timely bone marrow examination. In this patient, cyclosporine was effective; after anemia correction, HP was tolerated. The observed gBRCA1 mutation is a hypothesis-generating finding requiring further validation.

Keywords: BRCA1 Protein • Breast Neoplasms • Case Reports • Cyclosporine • Pure Red-Cell Aplasia
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/952933

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Introduction

Breast cancer is the most prevalent malignancy in women [1]. The human epidermal growth factor receptor 2 (HER2)-positive subtype, accounting for about 20% of all cases, is commonly treated with platinum-taxane chemotherapy in combination with HER2-targeted agents. For HER2-positive breast cancer, docetaxel/carboplatin/trastuzumab with pertuzumab (TCbHP) is now the preferred neoadjuvant regimen [2], while TCbH (without pertuzumab) was a recommended alternative according to the 2021 Chinese Society of Clinical Oncology Breast Cancer guidelines [3]. Anemia is the most common hematologic adverse event of TCbH, followed by neutropenia; grade 3/4 anemia occurs in 12% of patients [4]. Although 3 cases of pure red cell aplasia (PRCA) have been reported with carboplatin/paclitaxel in patients with endometrial carcinoma [5], recurrent thymoma [6], and metastatic basal cell carcinoma [7], no cases of PRCA following TCbH have been reported in breast cancer patients, particularly those with germline *BRCA1* (gBRCA1) mutations.

Pure red cell aplasia is a rare bone marrow failure disorder characterized by isolated anemia with reticulocytopenia and a marked reduction in erythroid precursors. Etiologically, it is congenital (Blackfan-Diamond Anemia, DBA) or acquired. DBA primarily affects infants and is often caused by mutations in ribosomal protein genes (eg, RPS19). Acquired PRCA can be associated with thymomas, viral infections, lymphoproliferative disorders, autoimmune diseases, solid malignancies, or drug exposures [8]. The annual incidence of acquired PRCA in Asian populations is estimated at 1.06 per million [9].

This report presents the case of a 45-year-old woman with right breast ductal carcinoma (cT3N2M0, HER2-positive) who developed PRCA following neoadjuvant TCbH and was effectively treated with cyclosporine. This case report aims to raise clinician awareness of this rare complication, emphasize the importance of bone marrow examination in reticulocytopenic anemia, demonstrate the efficacy of cyclosporine in drug-associated PRCA, and confirm the feasibility of HER2-targeted therapy after anemia correction.

Case Report

A 45-year-old Chinese woman with no comorbid conditions, developmental abnormalities, or significant family history of anemia or malignancies discovered a mass in her right breast in June 2021. Imaging, including mammography and ultrasound (**Figure 1A-1D**), suggested malignancy, which was confirmed

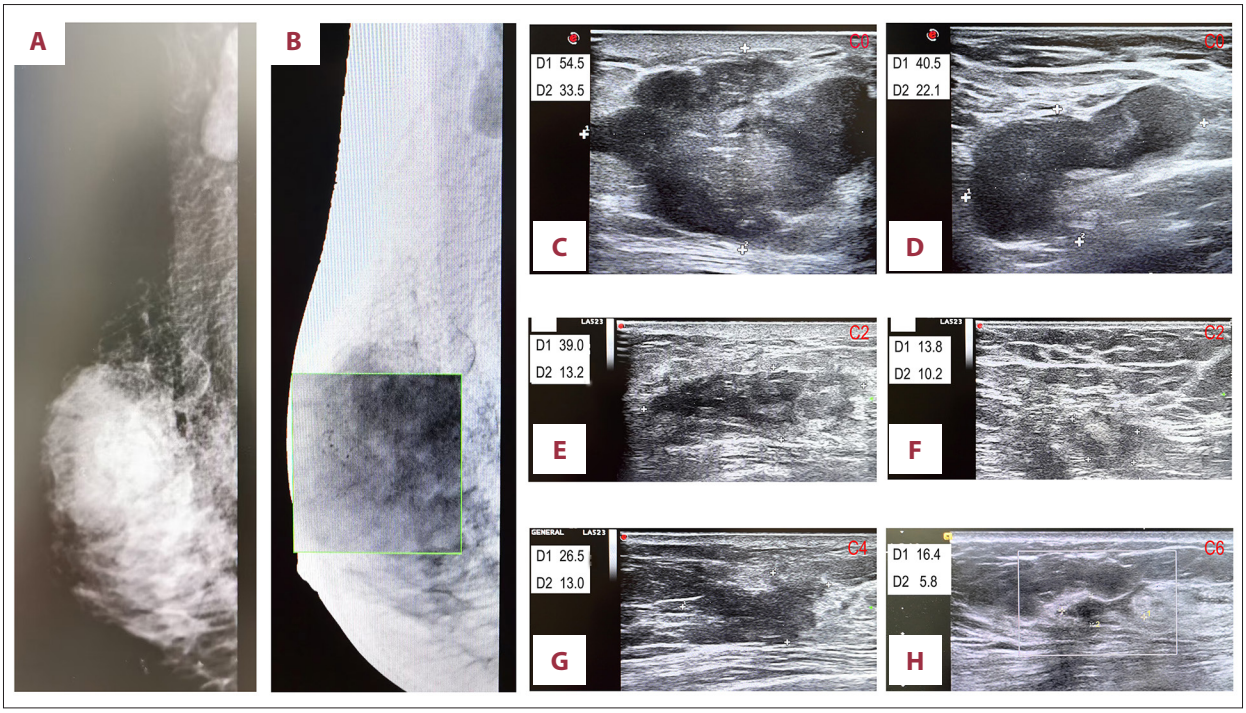

Figure 1. Tumor imaging at diagnosis and during neoadjuvant therapy. (**A, B**) Baseline mammography reveals a breast mass with internal calcifications and an enlarged axillary lymph node. The calcifications are better visualized on the inverted image (**B**). (**C**) Baseline ultrasound reveals the breast mass and (**D**) an enlarged axillary node. (**E, F**) Ultrasound after cycle 2 (C2) demonstrates the breast mass (e) and the axillary node (**F**). (**G, H**) Ultrasound after C4 (**G**) and C6 (**H**) demonstrates progressive tumor shrinkage. C, cycle; D1, longest diameter; D2, shortest diameter (mm).

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Table 1. Laboratory findings before neoadjuvant therapy and after six cycles neoadjuvant therapy.

Item	Before therapy	After six cycles	Reference ranges	Unit
White blood cell	4.3	3.1*	3.69-9.16	10^9/L
Platelet	352	157	101-320	10^9/L
Red blood cell	4.08	0.98*	3.69-5.13	10^{12}/L
Hemoglobin	98*	34.2*	113-151	g/L
Mean corpuscular volume	81.9*	98	82.6-99.1	fL
Mean corpuscular hemoglobin concentration	293*	354	320-355	g/L
Total protein	73.5	72.2	65-85	g/L
Albumin	49.3	47.9	40-55	g/L
Total bilirubin	4.37	5.8	< 15	umol/L
Direct bilirubin	2.42	1.3	< 5	umol/L
Alanine aminotransferase	7	22	7-40	U/L
Aspartate aminotransferase	18	31	13-35	U/L
Sodium	141	140	135-145	mmol/L
Potassium	3.9	4.07	3.5-5.3	mmol/L
Chlorine	102	101	95-110	mmol/L
Creatinine	70	65	45-84	umol/L
Serum iron	–	54.88**	7.8-32.2	umol/L
Ferritin	–	350.99**	15-150	ug/L
Serum transferrin	–	252.00	230-410	mg/dL
Total iron-binding capacity	–	58	50-77	umol/L
Vitamin B12	–	579	138-652	pmol/L
Folic acid	–	12.8	7-46.4	nmol/L
Soluble transferrin receptor	–	0.55	0.76-1.76	mg/L
Erythropoietin	–	> 750**	4.3-29	mIU/mL

* Below reference limit, ** above reference limit.

by biopsy on June 16, 2021, revealing a grade 3 invasive ductal carcinoma. Immunohistochemistry showed estrogen receptor-negative (ER [-]), progesterone receptor-negative (PR [-]), HER2-positive (HER2 3+) and Ki-67 50%. Distant metastasis was excluded by chest computed tomography (CT), abdominal ultrasound, and bone scan. According to the TNM classification (American Joint Committee on Cancer 8th edition) [10], clinical staging was established as cT3N2M0 (IIIA). Neoadjuvant therapy with the TCbH regimen (docetaxel 110 mg, carboplatin 550 mg, trastuzumab 300 mg [loading dose 400 mg], day 1, every 3 weeks) was initiated. Ultrasound reassessment after every 2 cycles demonstrated a partial response (**Figure 1E-1H**).

Pre-neoadjuvant hemoglobin (Hb) was measured at 98 g/L (reference ranges: 113-151g/L, grade 1 anemia) [11]. Given that both mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration levels (MCHC) were below the reference range, oral iron supplementation was administered. During cycles 1 to 4, Hb fluctuated between 82 g/L and 99 g/L, but it declined progressively after the fifth cycle. Two weeks after the sixth cycle, Hb decreased to 59 g/L. The patient exhibited weakness in both lower limbs, without signs of infections, gastrointestinal bleeding, or thymoma on chest CT. Laboratory results showed significantly elevated serum iron, ferritin, and erythropoietin levels, while transferrin, folic acid, vitamin B12, and bilirubin remained normal (**Table 1**). Transferrin saturation

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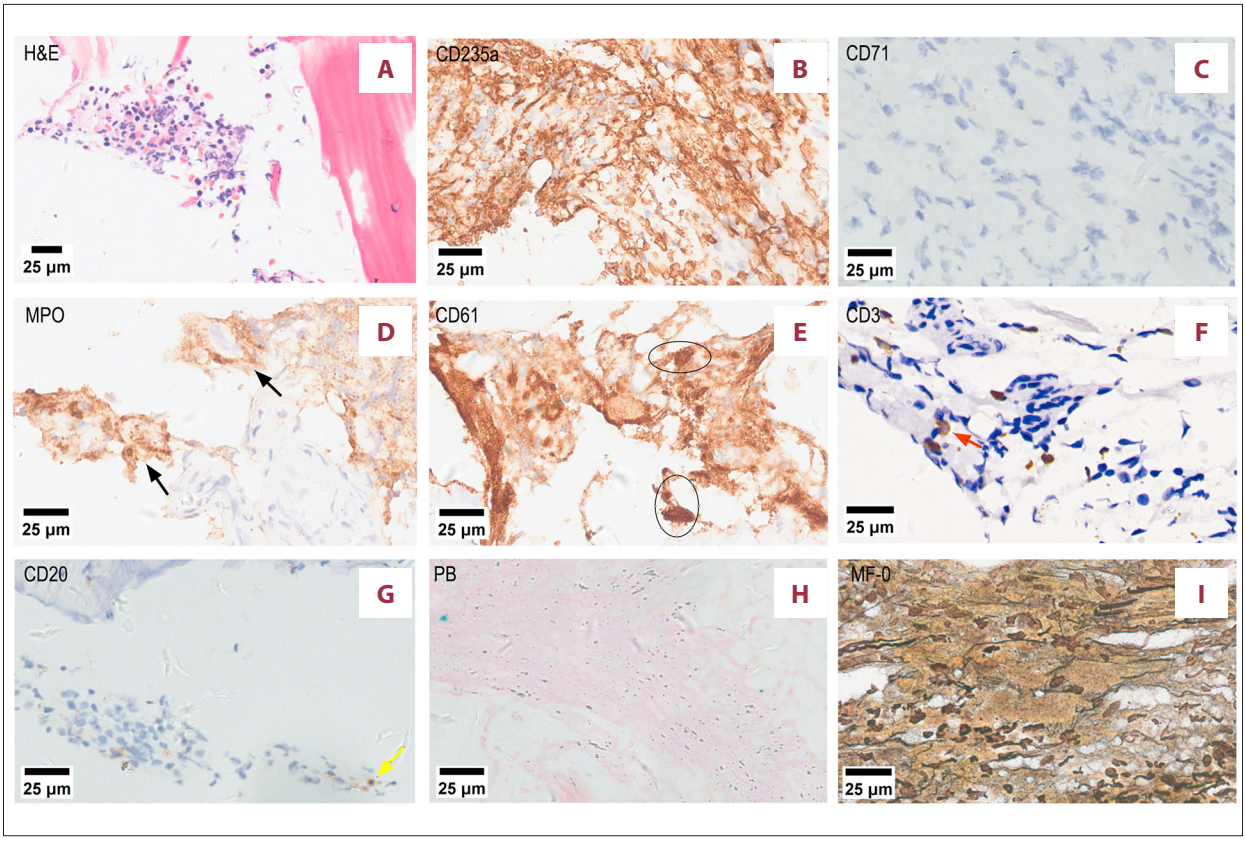

Figure 2. Histopathological and immunohistochemical findings of bone marrow biopsy. (**A**) Hematoxylin and eosin (H&E) staining shows markedly decreased erythroid precursors with disruption of erythroblastic islands (magnification × 400). (**B**) CD235a (glycophorin A) staining confirms the marked reduction of erythroid precursors; most of the CD235a-positive cells are red blood cells (magnification × 600). (**C**) Early erythroid precursors (CD71 [transferrin receptor 1]-positive) are absent (magnification × 600). CD71-negative/CD235a-positive pattern suggests arrested erythropoiesis. (**D, E**) Granulocytic cells (myeloperoxidase [MPO]-positive) and megakaryocytes (CD61 [integrin β3]-positive) are observed (magnification × 600). Black arrows indicate representative MPO-positive cells; black ellipses indicate representative CD61-positive cells. (**F, G**) Scattered lymphoid infiltrates consisting of CD3-positive T-cells and CD20-positive B-cells (magnification × 600). The yellow arrow indicates a representative CD20-positive cell; the red arrow indicates a representative CD3-positive cell. (**H**) Prussian blue (PB) staining for iron is negative (magnification × 600), indicating absence of iron overload or ring sideroblasts. (**I**) Gomori's reticulin staining shows myelofibrosis grade 0 (MF-0) reticulin fibrosis (magnification × 600), excluding bone marrow fibrosis.

was 98% (reference range: 16%-45%). Notably, reticulocytes were 3.8×10^9/L (reference range: 24-84 × 10^9/L), with a percentage of 0.2 (reference range: 0.5%-1.5%). MCV and MCHC levels were within normal limits after the sixth cycle (**Table 1**). These findings ruled out iron deficiency, megaloblastic anemia and hemolytic anemia, consistent with a normocytic normochromic anemia. Grade 3 neutropenia occurred after cycle 3 and 6 but resolved promptly with recombinant human granulocyte colony-stimulating factor.

Her anemia was initially attributed to drug-induced myelosuppression. However, it was refractory to subcutaneous recombinant human erythropoietin (rhEPO). Two weeks after rhEPO initiation, Hb fell to 34.2 g/L (grade 4 anemia), requiring red blood cell (RBC) transfusion. A total of 8 units of RBC suspension were transfused, raising Hb to 89.4 g/L. Subsequently, she underwent a right modified radical mastectomy. The pathology examination confirmed a pathological complete response (pCR). Postoperatively, Hb continued to decline, reaching 54.2 g/L 2 weeks after surgery. This prompted bone marrow examination, which was performed on December 10, 2021.

Bone marrow aspirate showed absence of erythroid precursors with mild granulocytic hyperplasia (consistent with post-chemotherapy reactive changes) and normal differential counts. Bone marrow histology demonstrated markedly reduced erythroid precursors, normal morphology and distribution of both granulocytic and megakaryocytic lineages, and a few scattered lymphocytes (**Figure 2A-2G**). Special staining was negative for iron and showed myelofibrosis grade 0 for reticulin fibrosis (**Figure 2H, 2I**),

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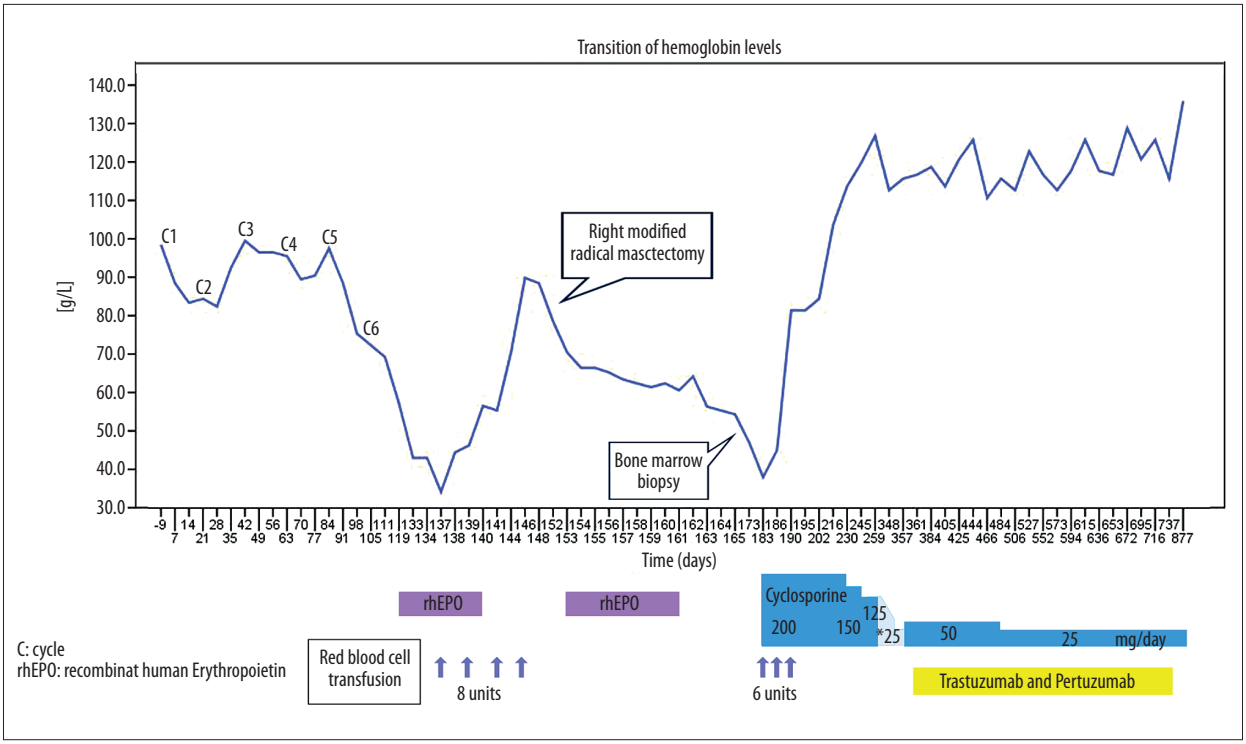

Figure 3. Hemoglobin levels and treatment timeline. * The patient self-adjusted the dosage due to COVID-19 lockdown drug shortage.

ruling out iron overload and bone marrow fibrosis. Flow cytometry revealed no abnormal immunophenotype and conventional cytogenetic analysis showed a normal female karyotype (46, XX [20]). The above findings excluded acute leukemia, myelodysplastic syndrome, lymphoma, aplastic anemia, large granular lymphocytic leukemia, and other T-cell clonal disorders. Screening for secondary causes, including autoimmune diseases, viral hepatitis, and thymoma, was negative. She did not take any other medications prior or during neoadjuvant therapy. Parvovirus B19 infection was considered unlikely based on the absence of typical clinical symptoms (fever, arthralgia, rash) and the lack of giant proerythroblasts on bone marrow morphology. Testing of B19 was not performed. Based on these findings, the patient was diagnosed with acquired PRCA.

Oral cyclosporine (100 mg twice daily; 4 mg/kg/day) was initiated. Within the first week, the patient received 3 transfusions (2 units each), with Hb levels rising from 38 g/L to 81 g/L. Hb began to rise steadily after 3 weeks, achieving complete remission by week 7. The dosage was gradually tapered, but approximately 4 months later she self-adjusted the dose to 25 mg once daily (0.5 mg/kg/day) due to drug shortages caused by COVID-19 lockdowns. After lockdown, on follow-up with a Hb level of 115 g/L, the dosage was adjusted to 25 mg twice daily (1 mg/kg/day). After 5 months, it was reduced to 25 mg once daily (0.5 mg/kg/day) and ultimately discontinued after a total treatment duration of 2 years.

After anemia was corrected with cyclosporine, the patient received adjuvant radiotherapy (25 fractions) and 1 year of dual HER2-targeted therapy (trastuzumab/pertuzumab, HP) while continuing cyclosporine. Throughout this period, the Hb level remained at ≥110 g/L. The clinical timeline of Hb levels and key treatments is shown in **Figure 3**.

Given the young age at onset and the rarity of PRCA, genetic testing was performed via whole-exome sequencing, identifying a heterozygous gBRCA1 mutation (c.788delG, p. Gly263Valfs*35), classified as likely pathogenic. At the 4.5-year follow-up, 2 years after cyclosporine cessation, the patient remains free of both oncologic and hematologic recurrence.

Discussion

This case highlights 3 clinically relevant observations: a temporal association between TCbH and PRCA, sustained hematologic remission with cyclosporine, and safe administration of HP after anemia correction.

In patients with malignancy, especially those with gBRCA1 mutations, the etiology of PRCA is multifactorial, potentially arising from the cancer itself, chemotherapy, or host-treatment interactions. In our case, PRCA was diagnosed concurrently with pCR, suggesting a treatment-related etiology rather than a

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Table 2. Clinical characteristics of the three documented cases.

Pt no	First author, year of publication [ref]	Sex/age (years)	Underlying disease	Clinical presentation	Treatment regimen	Outcome
1	Zheng C, 2025 [5]	F/55	Endometrial carcinoma (stage IA, p53 high)	Severe anemia with reticulocytopenia following paclitaxel/carboplatin; near-complete absence of erythroid precursors	Prednisone	Hemoglobin normalized within 4 weeks; PRCA completely resolved
2	Koshio J, 2007 [6]	M/71	Recurrent invasive thymoma (stage III)	Severe anemia occurred after completion of paclitaxel/carboplatin	Prednisolone	Improvement of anemia and regression of recurrent thymoma
3	Carneiro BA, 2006 [7]	M/65	Metastatic basal cell carcinoma	Severe anemia following paclitaxel/carboplatin; very low reticulocyte count (0.2 percent), unresponsive to erythropoietin; absence of erythroid precursors	Prednisone	Anemia initially improved but recurred upon rechallenge with carboplatin/paclitaxel; transfusion-dependent; patient died

paraneoplastic syndrome. However, no standardized approach exists to differentiate the exact cause in such overlapping settings.

The pathogenesis of drug-induced PRCA is not fully understood. It primarily involves direct toxicity or immune-mediated mechanisms [8]. In our case, the sustained response to cyclosporine and the infiltration of $CD3^+$ T-cell and $CD20^+$ B-cell in the bone marrow support an underlying immune-mediated process rather than direct myelotoxicity. The TCb backbone is presumed to be the key trigger, although the causative agent cannot be identified from this single case.

Until now, only 3 cases of PRCA associated with platinum-taxane chemotherapy have been reported, all involving carboplatin and paclitaxel (PCb) and treated with corticosteroids [5-7] (**Table 2**). One additional case of PRCA following docetaxel and cisplatin has been reported; however, that patient also had a concurrent thymoma, a well-recognized paraneoplastic cause of PRCA, and the original authors attributed PRCA to thymoma rather than to chemotherapy [12]. Therefore, this case is not included in **Table 2**. In contrast, our patient received docetaxel, a semi-synthetic taxane derivative, and achieved a favorable response to cyclosporine. Docetaxel and paclitaxel share a core mechanism of action—stabilizing microtubules, inhibiting mitosis and arresting cell cycle progression. Our case, together with the 3 documented cases, supports an immune-mediated mechanism. However, none of these cases has identified drug-specific antibodies or elucidated the precise underlying mechanism.

A distinctive feature of our case is the patient's gBRCA1 (c.788delG) frameshift mutation, predicted to produce a truncated protein lacking the BRCT domains and leading to dysfunction of BRCA1-mediated DNA repair [13]. *BRCA1* is integral to the Fanconi anemia -BRCA DNA repair pathway, which coordinates repair of DNA interstrand crosslinks and maintains genomic stability. *BRCA1* mutations have been associated with an increased risk of the hematologic toxicities of taxane-based [14] and platinum-based chemotherapies [15]. Biallelic pathogenic variants of BRCA have been reported to be associated with hypersensitivity to DNA crosslinking agents and severe bone marrow suppression [16]. Carboplatin induces DNA interstrand crosslinks [17], while docetaxel promotes mitotic arrest. Given our patient's underlying *BRCA1* deficiency, we hypothesize that this inherent DNA repair defect may have impaired the recovery capacity of bone marrow from chemotherapy-induced damage. Persistent damage and repeated drug exposure may trigger an aberrant immune response selectively against erythroid precursors, culminating in isolated erythroid failure. Notably, this remains a hypothesis generated from a single observation, and current evidence is insufficient to validate gBRCA1 mutation as an independent risk factor for drug-induced PRCA. Based on our literature review, this appears to be the first documented case of TCb-associated PRCA in a patient with *gBRCA1*-mutated breast cancer.

The first-line treatment of acquired PRCA typically involves immunosuppression with cyclosporine or corticosteroids. One meta-analysis has reported that corticosteroid monotherapy achieves an overall response rate of 47%, while cyclosporine monotherapy yields a rate of 83.2%. Cyclosporine/corticosteroids combination therapy offers no clear superiority in response rate or time to response [18]. In addition, combined therapy is

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associated with higher risk of corticosteroid-related infection than cyclosporine monotherapy [19]. Given the adverse effects of long-term corticosteroid therapy, cyclosporine monotherapy was chosen, resulting in sustained remission in our case.

This report has several limitations. First, drugspecific antibodies and immune biomarkers were not tested, leaving the causative drug and mechanism unknown. Second, as a single case observation, the findings are preliminary, and the link between the *gBRCA1* mutation and PRCA susceptibility is speculative, requiring further mechanistic and epidemiological validation. Finally, parvovirus B19 was not tested. Although infection is unlikely given the absence of typical symptoms and giant proerythroblasts, and the good response to cyclosporine, we cannot completely exclude it.

Conclusions

This case report shows that refractory anemia accompanied by reticulocytopenia during TCbH therapy should prompt consideration of PRCA and timely bone marrow examination. In this patient, cyclosporine proved to be an effective treatment for drug-induced PRCA, and subsequent administration of dual HER2-targeted therapy after anemia correction was well-tolerated without recurrence of PRCA. However, this observation is limited to a single case. The presence of a germline BRCA1 mutation in this patient raises a hypothesis regarding potential genetic susceptibility, but no causal relationship can be established based on this single case.

Acknowledgements

The authors sincerely thank the patient for her cooperation and understanding.

Institutions Where Work Was Done

The International Peace Maternity and Child Health Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, PR China, and Zhongshan Hospital, Fudan University, Shanghai, PR China.

Patient Permission/Consent Declarations

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

AI Declaration

During the preparation of this work, the authors used DeepSeek -V3 to polish language and refine grammar. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the published article.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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