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ABOUT COVER

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CASE REPORT

Seven-years post allogeneic hematopoietic stem cell transplantation pure red cell aplastic anemia cured with daratumumab: A case report and review of literature

Bo Deng, Rui Gao, Bing Yang, Wen-Bing Lei, Ming-Fang Xue, Ji-Shi Wang, Peng Zhao

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Abstract

BACKGROUND

Allogeneic hematopoietic stem cell transplantation (Allo-HSCT) is currently the only viable method of curing patients with acute myeloid leukaemia. In 30% to 50% of patients, donors and recipients have some level of ABO blood group incompatibility. ABO blood group incompatibility can cause antibodies against the donor's red blood cells to persist in the recipient's body, resulting in a delay of several months in the recovery of red blood cells. A number of different treatments have been reported for post-transplant pure red cell aplastic anaemia (PRCA), such as plasmapheresis, donor lymphocyte infusions, anti-thymocyte globulin, rituximab and steroids.

CASE SUMMARY

A 41-year-old female diagnosed with acute myeloid leukaemia underwent peripheral blood allogeneic haematopoietic stem cell transplantation in November 2013 from an HLA matched unrelated donor. The donor was AB-positive and the recipient was O-positive. The patient was diagnosed with PRCA three months after receiving the donor stem cell transplant. After failing multiple lines of therapy, the patient applied for daratumumab. After receiving three doses of daratumumab, the patient developed a reticulocyte response and no longer required

blood transfusions.

CONCLUSION
The use of daratumumab anti-CD38 for the remove of plasma cells is safe and effective and may be tried for refractory patients with PRCA after undergoing allo-HSCT for ABO incompatibility.

Key Words: Transplantation; PRCA; Daratumumab; Leukemia; blood-group; Case report

Core Tip: Allogeneic hematopoietic stem cell transplantation (Allo-HSCT) is currently the only possible way to cure patients with malignant hematologic diseases. In Allo-HSCT, donors have some degree of ABO blood-group incompatibility with the recipient. An outcome of ABO incompatibility caused is pure red cell aplastic anemia (PRCA). We report a 41-year-old female diagnosed with acute myeloid leukemia received a peripheral blood allo-HSCT from an HLA-matched unrelated donor in November 2013. The patient was diagnosed with PRCA three months after allo-HSCT. After failing multiple lines of treatment, daratumumab was requested. After receiving three doses of daratumumab, the patient had a marked reticulocyte response and become transfusion independent.Using of anti-CD38 therapy with daratumumab to target residual host plasma cells is safe and effective, and it can be considered in refractory recipients with PRCA after allo-HSCT secondary to ABO incompatibility.

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INTRODUCTION

The incidence rate of pure red cell aplasia (PRCA) after transplantation is 6%–30%, and it varies with conditioning. Host hematopoietic function may persist after nonmyeloablative stem cell transplantation, and for a considerable duration, host and donor hematopoietic and immune functions may coexist (mixed chimerism). The risk of PRCA is increased when the blood type of the recipient is O and the blood type of the donor is A[1,2]. These patients often have undetectable reticulocytes, become dependent on red blood cell infusion, and are at risk of transfusion-related iron overload. Current treatments include tapering immunosuppressants, erythropoietin, glucocorticoids, plasmapheresis, rituximab, and donor lymphocyte infusion. CD38 is expressed at high levels in plasma cells and Daratumumab is a human IgG1κ monoclonal antibody against CD38. Considering that pure red cell aplastic anemia after transplantation is a pathophysiological change caused by antibodies produced by residual plasma cells of the recipient, we hypothesised that selective elimination of pathogenic plasma cell populations could overcome refractory erythrocyte-only aplastic anaemia. There have also been sporadic reports of successful use of daratumumab in patients with post-transplant pure red cell aplastic anemia[3-11] (Table 1). We present a patient who had pure red cell aplasia for 7 years, successfully treated with daratumumab after HLA-matched unrelated transplantation. This was the longest post-transplantation case reported in the literature but was still effectively treated after using daratumumab.

CASE PRESENTATION

Chief complaints

A 41-year-old female patient was admitted to our center 7 years ago due to gingival bleeding and was diagnosed with acute myeloid leukemia M2. Cytogenetic tests showed no positive findings.

History of present illness

After chemotherapy, the patient achieved complete remission and received unrelated HLA 10/10 matched hematopoietic stem cell transplantation (HSCT) (AB + donor, O + recipients). On + 106 days after transplantation she was diagnosed as PRCA, and she failed multiple therapies for it.

History of past illness

Conditioning began on November 17, 2013, performed with busulfan and cyclophosphamide + antithymocyte globulin (BU/CY + ATG) regimen. She received peripheral blood hematopoietic stem cells and CD34 + cells and mononuclear cell count was 4.67106/kg and 6.11108/kg, respectively. After transplantation, on day 11, neutrophil engraftment (0.65109/L)

Table 1 Summary of daratumumab utilization in post-transplant major ABO mismatched hematopoietic stem cell transplantation and delayed red blood cell engraftment

Ref.	Age/sex	Diagnosis	Type of transplant	ABO mismatch (D/R)	Conditioning	Source	GVHD prophylaxis	CD34+ cells (× 10^6/kg)	Neutrophil	Platelet	Previous therapies	Initiation	Doses	Time to response
Grigg *et al*[15]	17/F	SAA	MSD	B/O	FLU + Cy + ATG	PBSC	Tacrolimus; MTX	3.34	D+14	D+11	Tapering of IST; Glucocorticoids; IVIG; Bortezomib	D+163	4	After 3rd doses
Chapuy *et al*[3]	72/M	MDS	MUD	A/O	RIC	PBSC	Tacrolimus; MTX	5.01	D+18	D+25	Tapering of IST; Glucocorticoids; Rituximab; Darbepoetin	D+390	6	After 1st dose
Bathini *et al*[5]	60/F	MDS	MUD	A/O	RIC	PBSC	Tacrolimus; MTX; ATG	6.0	D+11	D+18	Tapering of IST; Glucocorticoids; Rituximab; Bortezomib	D+411	4	After 1st dose
Rautenberg *et al*[4]	43/F	AML	MUD	A/O	MAC	PBSC	Tacrolimus; MMF	8.0	D+9	D+17	Tapering of IST; Rituximab	D+206	2	After 2nd dose
Salas *et al*[7]	34/M	AA	MSD	AB/O	RIC	PBSC	MTX; CSA	6.91	D+13	D+11	Tapering of IST; Rituximab; Glucocorticoids; PX; Bortezomib	D+700d	6	After 6th dose
Henig *et al*[8]	25/M	Chronic neutropenia	MSD	A/B	MAC	PBSC	MTX; CSA	7.9	D+23	D+12	Tapering of IST; Rituximab; Bortezomib	D+320	5	After 4th dose
Yates *et al*[9]	14/F	DOCK8 deficiency	Haplo	A/O	MAC	BM+PBSC	CTX; tacrolimus; MMF	9.3	D+15	D+22	Tapering of IST	D+397	3	After 3rd doses
Martino *et al*[10]	77/F	AML	NR	A/O	RIC	NR	Sirolimus tacrolimus	NR	NR	NR	NR	D+205	3	After 1st dose
Martino *et al*[10]	62/F	MDS	NR	AB/O	RIC	NR	CTX tacrolimus	NR	NR	NR	NR	D+270	2	After 2nd dose
Asawapanumas *et al*[11]	49/M	AML	MSD	B/O	MAC	NR	Pt-Cy; CSA; MMF	7.17	D+21	D+16	Tapering of IST	D+146	1	After 1st dose
Our patient	41/F	AML	MUD	AB/O	MAC	PBSC	MTX; CSA	4.67	D+11	D+12	Tapering of IST; PX; IFN; MNC	D+2701	5	After 2nd dose

M: Man; F: Female; MSD: Matched sibling donor; MUD: Matched, unrelated donor; MMF: Mycophenolat mofetil; PX: Plasma exchange; RIC: Reduced intensity conditioning; MAC: Myeloa-blative conditioning; IFN: Interferon; MNC: Mesenchymal stem cell; Haplo: Haploidentical allogeneic stem cell transplantation; DLI: Donor lymphocyte infusion; Pt-Cy: Post-transplantation cyclophosphamide; MMF: Mycophenolate mofetil; MTX: Methotrexate; CSA: Cyclosporine; CTX: Cyclophosphamide; IST: Immunosuppressive therapy.

and on day 12, platelet engraftment (31109/L) were performed. The number of neutrophils and platelets was 2.3109/L on day 14 and 112109/L on day 21. She had donor chimerism of 98.42% on day 29. Bone marrow aspiration and biopsy on day 31 post-transplant showed 45% cellularity with erythroid suppression, 36 megakaryocytes/1.5 cm × 3.0 cm, naive megakaryocytes 3%, granular megakaryocytes 45%, thrombocytogenic megakaryocytes 50%, and naked megakaryocytes 2%. Direct Coombs test was negative with reticulocytopenia (0.1%, 3000/μL), and we considered PRCA. Due to hypoplasia of the erythrocytes caused by blood incompatibility between donor and recipient, the patient relied on frequent blood transfusions, persisting over 96 d after transplantation, which resulted in secondary iron overload. The blood tests showed the concentration of serum ferritin was between 854 and 9069 ng/mL; hence, she was treated with deferasirox 1250 mg/d (20 mg/kg/d) for iron removal. She did not have any evidence of graft *vs* host disease (GVHD); therefore, cyclosporin taper was initiated on day 87 and was stopped on day 101. She continued to have reticulocytopenia and required blood transfusion every 2 weeks. The blood type identified remained O for multiple blood examinations, while the donor's type AB blood was not detected. On day 106 after transplantation, she was diagnosed with PRCA, and she failed multiple therapies for it. Glucocorticoids were started on day 124 after transplantation, and were slowly tapered over a period of several weeks. This was followed by three sessions of plasma exchange as second-line treatment on day 150, with no clinical response. From October to November 2014 (10 months after transplantation), interferon at a dose of 30 g subcutaneous injection three times a week was given without response. On day 630, 106 mesenchymal stem cells (MSCs) per 10 kg body weight was given intravenously twice weekly, and no MSC infusion-related adverse effects were noted. However, this was unsuccessful.

Personal and family history

The patient denied any family history of malignant tumours.

Physical examination

Physical examination was unremarkable.

Laboratory examinations

Bone marrow aspiration and biopsy on day 31 post-transplant showed 45% cellularity with erythroid suppression, 36 megakaryocytes/1.5 cm × 3.0 cm, naive megakaryocytes 3%, granular megakaryocytes 45%, thrombocytogenic megakaryocytes 50%, and naked megakaryocytes 2%. Direct Coombs test was negative with reticulocytopenia (0.1%, 3000/μL).

Hemoglobin 50 g/L, reticulocyte count 0.57% (17100/μL), IgM anti-A1: 128, IgG anti-A1: 512, IgM anti-B negative, IgG anti-B 1:512, 12 days after daratumumab infusion. On day 21 after daratumumab infusion: Hemoglobin 45 g/L, reticulocyte count 4.25% (127500/μL), IgM anti-A 1:64, IgG anti-A 1:128, IgM anti-B negative, and IgG anti-B 1:128, at which point the patient began to be transfusion independent. On day 28 after daratumumab infusion: Hemoglobin 60 g/L, reticulocyte count 10.37% (311100/μL), IgM anti-A1: 16, IgG anti-A1: 64, IgM anti-B negative, IgG anti-B negative, positive type O, and negative type B.

Imaging examinations

No abnormalities on imaging.

FINAL DIAGNOSIS

Combined with the patient's medical history, the final diagnosis was PRCA.

TREATMENT

On July 1, 2020, we noticed a report that patients with PRCA following allo-HSCT were successfully treated with daratumumab[4]. Prior to treatment with daratumumab (on day 2694 post-transplantation), laboratory findings indicated a paucity of erythroid precursors on bone marrow examination. Erythroid elements accounted for < 1% of total cellularity. Other tests showed that the concentration of Ferritin was 4530 ng/mL, and the blood routine test showed anemia (white blood cell count 3.34 × 10^9/L, hemoglobin 37 g/L and platelet count 213 × 10^9/L). The first dose of daratumumab (16 mg/kg) was administered at day 2701 after transplantation, followed by four doss at days 2708, 2715, 2722 and 2731. We found that after a three-dose infusion of daratumumab, the patient's haemoglobin and reticulocytes began to rise and antibody titres fell, suggesting that the treatment was effective.

OUTCOME AND FOLLOW-UP

At 124 days following infusion of daratumumab, hemoglobin was 150 g/L, anti-A and anti-B were negative, and blood type was AB. In the last test, at day 3098 after transplantation: Hemoglobin 151 g/L, reticulocyte count 5.05% (151500/μL), anti-A, anti-B still negative, and blood type AB (Figures 1 and 2). A bone marrow aspirate revealed a normal number of erythroid precursors (28%) and no evidence of recurrent acute myeloid leukemia.

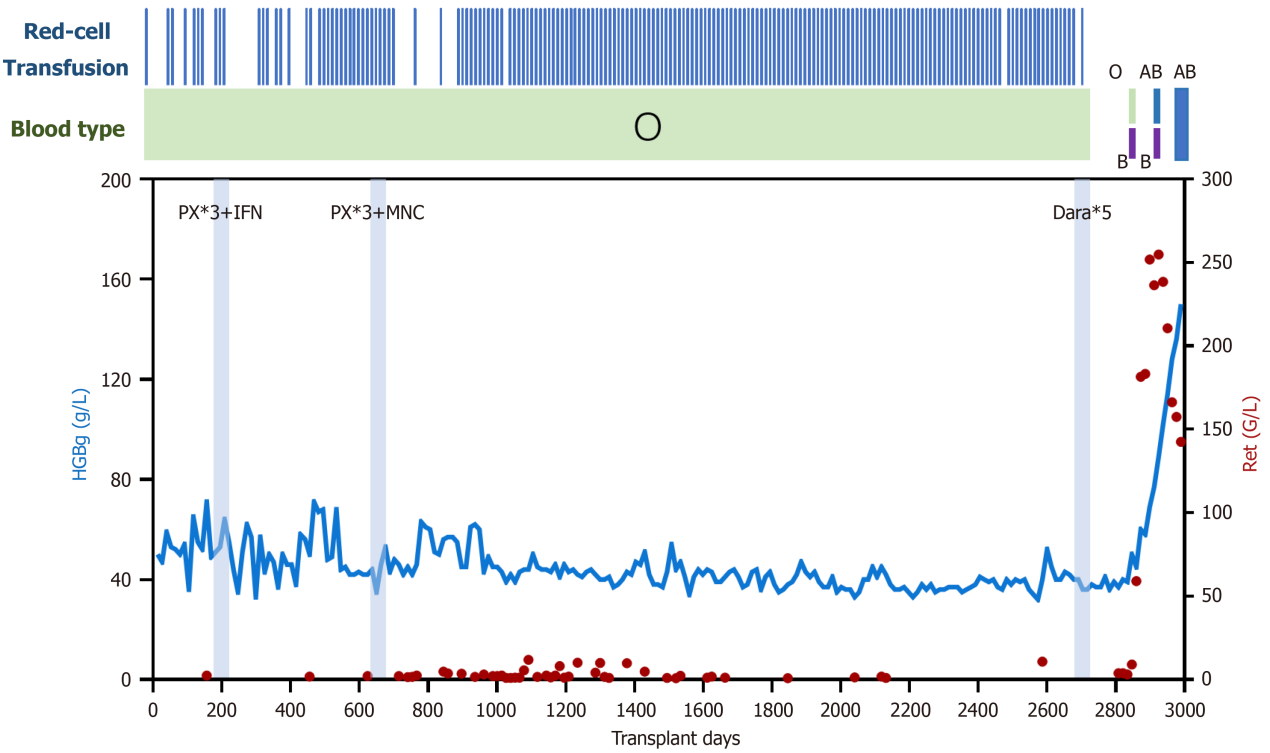


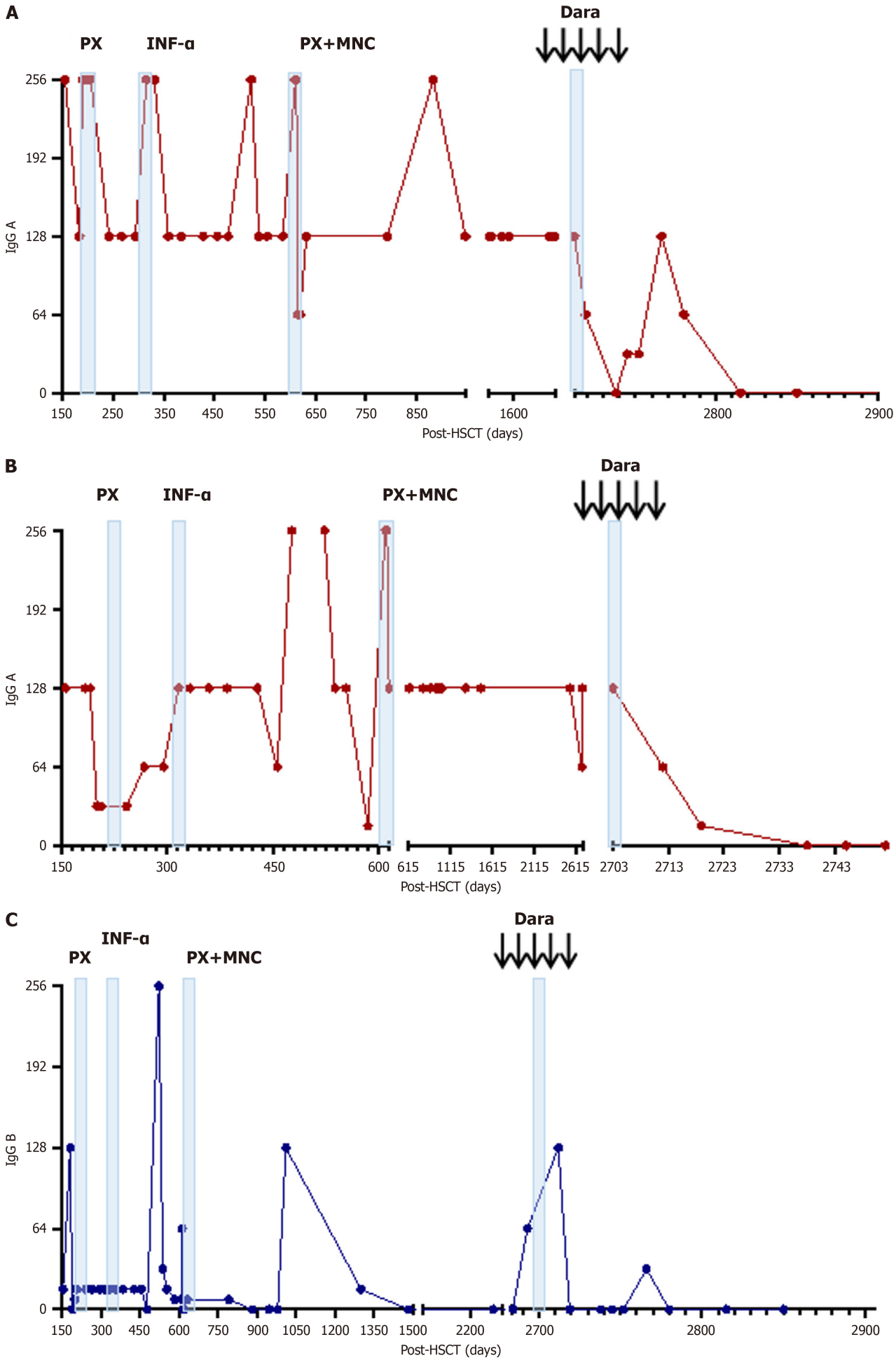
Figure 1 Treatment process of pure red cell aplastic anaemia patients after unrelated human leukocyte antigen-matched AB + /O + allo-transplantation. Hemoglobin (Y axis), transplant days (X axis), absolute value of reticulocyte, frequency of red blood cell infusion, and changes in blood type before and after transplantation are shown here, and the trend of treatment with plasmapheresis, interferon, mesenchymal stem cells, and Daratumumab (shaded) is shown. Red blood cell transfusion frequency showed no change before and after plasmapheresis, interferon, and mesenchymal stem cell treatment, but after 5 dosages of Daratumumab, hemoglobin significantly increased, red blood cell transfusion frequency decreased.

DISCUSSION

Pure red cell aplastic anemia is a complication associated with ABO mismatched allo-HSCT characterized by anemia, reticulocytopenia, and lack of erythrocytes, disregarding other causes such as infection, hemolysis, disease relapse, or drug toxicity. Since PRCA is self-limiting, it may resolve spontaneously within weeks to years. To date, there is no standard treatment for PRCA following transplantation[12]. The earliest treatments used were to taper immunosuppressants to promote graft resistance to plasma cell effects[13-15]. Yamaguchi *et al*[14] reported a case of PRCA anemia following HLA-matched HSCT, in which cyclosporine decreased by 25% every 2 weeks from day 123. Skin and liver GVHD occurred during tapering, and the patient was free from transfusion dependence at day 167. We found that nine cases reported an increase in reticulocyte count and blood transfusion independence at days 128 to 376 after transplantation using tapering of immunosuppressive therapy[13,16-18], but there were also many unsuccessful cases[19-21]. Our patient was initially treated with cyclosporine reduction, but the bone marrow erythrocytes and reticulocytes did not increase, and GVHD did not appear after tapering immunosuppressive therapy.

Corticosteroids are often used alone or in combination with other treatments, although with unsatisfactory results[22,23]. Erythropoietin (EPO) is used for PRCA to stimulate the remaining red progenitor cells in bone marrow. This effect may have resulted from the inhibition of erythropoiesis mediated by the major ABO group incompatibility, but there are fewer reports describing success of EPO therapy[24,25] compared with failure[14,26,27]. Plasma exchange is a widely used treatment, often in combination with other therapies, but is less effective in treating PRCA[28]. In patients with effective plasma exchange, initiation of therapy began > 100 d after transplantation, and > 200 d in most transfusion-dependent patients[22,29]. Donor lymphocyte infusion (DLI) treatment of PRCA after transplantation has rarely reported successful outcomes[19,28]. The donor in our case was unrelated to the patient; therefore, DLI could not be performed. The patient's bone marrow was completely chimeric with the donor during multiple examinations. DLI was not performed due to the possibility of GVHD[30]. Anti-CD20 chimeric IgG1 monoclonal antibody therapy (rituximab) is mainly used to remove the residual B lymphocytes of the host. At present, there are few successful reports on PRCA after transplantation, and the treatment course and dose are different; mostly at 375 mg/m^2, for four doses[20]. Treatment can also be 300 mg/m^2/dose[27], 235 mg/m^2/dose for four doses[31] and 150 mg/m^2/dose for three doses[27], but the practitioner should be vigilant of the risk of infection.

MSCs can significantly inhibit the proliferation, differentiation and immunoglobulin secretion of B lymphocytes, thus becoming a new treatment method for PRCA[32]. Fang *et al*[21] reported two cases of PRCA that failed with EPO and rituximab, but then received 1.5 × 106/kg donor adipose-derived MSCs at days 197 and 233 days, respectively, and achieved blood transfusion independence at days 220 and 250 days, respectively. Treatment of PRCA has also been reported sporadically with bortezomib[33-35] and Eltrombopag[36], but large-scale clinical data are lacking.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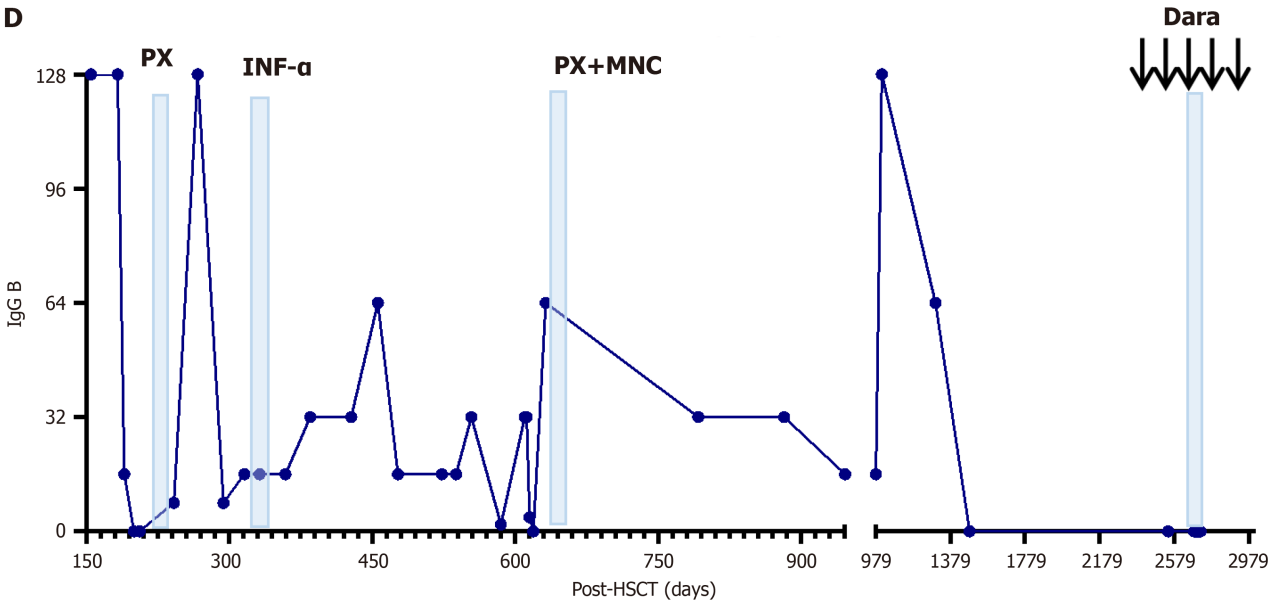

Figure 2 Changes in anti-A and anti-B titers (IgG + IgM) after transplantation. A: IgG anti-A, no significant changes in plasma exchange, interferon, and mesenchymal stem cell therapy + 1d to + 2700d after transplantation, and antibody levels gradually turned negative after Dara treatment; B: IgM anti-A, no significant changes in plasma exchange, interferon, and mesenchymal stem cell therapy until Dara treatment; C: IgG anti-B, no significant changes in plasma exchange, interferon, and mesenchymal stem cell therapy until Dara treatment; D: IgM anti-B, no significant changes in plasma exchange, interferon, and mesenchymal stem cell therapy + 1d to + 2700d after transplantation, and antibody levels gradually turned negative after Dara treatment. PX: Plasma exchange, INF: Interferon, MNC: Mesenchymal stem cell, Dara: Daratumumab.

CONCLUSION

Although there are only a few cases of PRCA treated with daratumumab, all the reported cases were cured. In addition, we noted a 74-year-old woman with a 10-year history of treatment-refractory idiopathic acquired PRCA with rapid and sustained response to daratumumab. At baseline, the patient was transfusion-dependent every 3 wk and reticulocyte percentage was < 0.28. One week after initiation of therapy, hemoglobin was > 8 g/dL and 1 and 2 mo post-therapy, hemoglobin level was 9.4 and 11 g/dL, respectively[37]. The median time of starting daratumumab varies between 146 and 700 d (median 295 d) after transplantation. Our patient received red blood cell infusion every 2 wk for > 7 years after transplantation. Due to a lack of precedence on administering daratumumab for PRCA treatments, the efficacy remained uncertain. Families of patients were fully informed of the circumstances concerning the efficacy prior to committing to the trial. The dose we used was consistent with that reported in the literature, 16 mg/kg, once a week. Antiallergic drugs were given before infusion. As the patient experienced fatigue and bone pain, the fifth dose was administered 9 d later. No serious adverse events occurred. In addition, no GVHD or opportunistic infections were observed during 397 d of follow-up after daratumumab treatment, suggestin.

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FOOTNOTES

Author contributions: Deng B and Gao R contributed to manuscript writing and editing, and data collection; Yang B, Lei WB, Xue MF contributed to data analysis; Wang JS performed the treatment; Zhao P contributed to conceptualization and supervision; all authors have read and approved the final manuscript.

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