Acquired severe aplastic anemia

Luiz Guilherme Darrigo Junior[1*], Gisele Loth[2], Virginio Climaco de Araújo Fernandes Júnior[3], Alexandre de Albuquerque Antunes[4], Gabriela Gaspar Filgueiras Landi[5], Carmem Bonfim[6,7]

1. Universidade de São Paulo – Hospital das Clínicas – Ribeirão Preto (SP), Brazil.
2. Universidade Federal do Paraná – Hospital de Clínicas – Curitiba (PR), Brazil.
3. Grupo de Pesquisa e Assistência ao Câncer Infantil – Sorocaba (SP), Brazil.
4. Hospital da Criança de Brasília José de Alencar – Brasília (DF), Brazil.
5. Hospital da Criança e Maternidade – São José do Rio Preto (SP), Brazil.
6. Hospital Pequeno Príncipe – Curitiba (PR), Brazil.
7. Duke University – Durham (NC), United States of America.

***Corresponding author:** darrigo.jr@gmail.com

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ABSTRACT

Abstract: Acquired severe aplastic anemia is a rare, life-threatening condition characterized by pancytopenia and hypocellularity of the bone marrow. It shows a bimodal age distribution, with approximately 70–80% of cases classified as idiopathic. Recent advances in hematopoietic cell transplantation, immunosuppressive therapy, biological agents, and supportive care have greatly improved survival rates, reaching a 95% cure rate. Differential diagnoses include refractory cytopenia of childhood, inherited bone marrow failure syndromes, and other blood disorders. The aim of this article was to update the Brazilian consensus previously published by the Brazilian Society of Cellular Therapy and Bone Marrow Transplantation in 2021, highlighting the latest developments in the treatment and monitoring of severe aplastic anemia patients undergoing hematopoietic cell transplantation.

Keywords: Anemia, Aplastic. Cytopenia. Bone Marrow. Hematopoietic Stem Cell Transplantation.

INTRODUCTION

Acquired severe aplastic anemia (SAA) is a rare and serious heterogeneous disorder characterized by pancytopenia and bone marrow hypocellularity, without significant signs of dysplasia, any increase in blasts, or bone marrow fibrosis[1]. It has a bimodal distribution, primarily affecting young individuals (10–25 years old) and the elderly (over 60 years old), with approximately 70–80% of cases considered idiopathic[1].

Survival has improved significantly, reaching a 95% cure rate due to advances in hematopoietic cell transplantation (HCT), immunosuppressive therapy (IST), biological agents, and supportive care[2]. The main differential diagnoses include refractory cytopenia of childhood, inherited bone marrow failure syndromes (IBMFS), and other disorders[3]. The current pathogenic model suggests an initial event (such as a viral infection or genetic mutation) that triggers the formation of autoreactive T cells, which attack the hematopoietic stem cells (HSC)[4]. Thus, the pathophysiology of the disease suggests two possible treatment approaches: HCT, which replaces deficient HSC with normal progenitors, or immunotherapy, that suppresses the immune process that causes the hematopoietic damage.

DIAGNOSTIC APPROACH

The diagnosis should not only aim to establish the presence of acquired SAA but also to investigate other potential causes of pancytopenia with hypocellular marrow and determine the differential diagnoses with neoplasms (including leukemias or myelodysplastic syndromes), IBMFS, that accounts for 25–30% of the cases, and autoimmune disorders/immune dysregulation[1]. A comprehensive diagnostic investigation considering family history, physical examination, and an extensive laboratory evaluation (Table 1) is crucial for accurately diagnosing acquired SAA. Among the IBMFS, are: Fanconi anemia, telomere biology disorders, Blackfan-Diamond anemia, and Shwachman-Diamond syndrome[2,3].

Table 1. Laboratory tests to define possible etiology and differential diagnoses of the bone marrow failure.

General tests	Complete blood counts with absolute reticulocyte count Peripheral blood smear analysis Liver transaminases, bilirubin Urea and creatinine Lactate dehydrogenase Uric acid Direct/indirect antiglobulin test (Coombs) Coagulation studies (PT, PTT, fibrinogen) Vitamin B12, folic acid, thyroid function tests Fetal hemoglobin test
Infectious diseases	Serologies for hepatitis (at least A, B and C), Epstein–Barr virus, varicella, cytomegalovirus, parvovirus B19, human herpesvirus 6, human immunodeficiency virus, herpes simplex virus 1 and 2.
Immunologic	Immunoglobulins: IgG, IgM, IgA Anti-nuclear antibody Lymphocyte subset immunophenotyping
Paroxysmal nocturnal hemoglobinuria screening	Flow cytometry on peripheral blood
Chromosomal fragility testing	Diepoxybutane or mitomycin C
Telomere length evaluation	Flow-FISH panel
Bone marrow evaluation	Bone marrow aspirate and biopsy for morphology and cellularity Flow cytometry Cytogenetics
Additional evaluation	Fecal pancreatic elastase, serum amylase and lipase, next-generation sequencing panels to identify cryptic mutations; TERC and TERT mutation analysis, TNF2, NHP2, NOP10, DKC1, cMPL mutation analysis, Shwachman-Diamond syndrome and Blackfan-Diamond syndrome mutation analysis. Specific patients with high suspicion for other genetic syndromes, like GATA2, SAMD9/SAMD9L, should be investigated with specific genetic tests.

Source: Adapted from Guarina et al.[1] and Darrigo Júnior et al.[5].

Aplastic anemia can be classified into three categories based on severity (Table 2).

Table 2. Diagnostic criteria and classification of aplastic anemia.

Moderate	Severe	Very severe
Hypocellular bone marrow with peripheral blood cytopenias not fulfilling criteria for either severe or very severe aplastic anemia	Hypoplastic bone marrow (< 25% of normal cellularity) or moderately hypoplastic marrow (25–50% of normal cellularity with < 30% of remaining cells being hematopoietic) PLUS At least two of the following: a) Neutrophils between 200 and 500/μL b) Platelets < 20,000/μL c) Reticulocytes < 20,000/μL	Like severe, but with neutrophils < 200/μL

Source: Adapted from Camitta et al.[6].

Accurate diagnosis is fundamental for determining the most appropriate treatment strategy, avoiding ineffective or harmful interventions, and ensuring the best therapeutic approach. Additionally, certain inherited disorders (*e.g.*, Fanconi anemia and telomere biology disorders) exhibit higher sensitivity to chemotherapy and radiotherapy, influencing the choice of conditioning regimen for HCT. Selecting the most suitable donor is also critical, as asymptomatic carriers within the family may risk HCT results. Furthermore, prognosis varies significantly, as specific diseases are associated with distinct complications, requiring tailored long-term monitoring and management[7].

FIRST-LINE TREATMENT OF ACQUIRED SEVERE APLASTIC ANEMIA

If a human leukocyte antigen (HLA)-matched sibling donor (MSD) is identified for pediatric patients, HCT should be the first-line therapy. This recommendation is based on studies comparing HCT-MSD *versus* first-line IST, in which MSD results show over 90% survival at 10 years[8]. Therefore, HLA typing for the patient and family should be conducted immediately for any newly diagnosed SAA[3]. However, approximately 70–80% of children and young adults with SAA will lack a MSD and will require IST or a transplant from an alternative donor, either a matched unrelated donor (MUD) or a haploidentical family donor (HAPLO). First-line MUD-HCT has significantly improved in recent years, showing overall survival (OS) rates similar to MSD and a superior outcome compared to MUD following the failure of IST[9]. Considering recent data, first-line HAPLO-HCT is a promising treatment option for patients with acquired SAA[10,11]. Thus, for patients lacking an MSD, MUD-HCT (if available within three months of diagnosis) or HAPLO-HCT (particularly for those with very SAA) should be considered; otherwise, IST should be initiated[12].

Bone marrow should be the preferred source of HSC for MSD, MUD, or HAPLO transplantation in patients with SAA. If a high number of cells is available, matched family umbilical cord blood (UCB) may also be an option. Peripheral blood (PB) may be used as an exception when other sources are unavailable; in this case, as published by the British group, specific protocols are recommended[13]. Two registry-based studies have demonstrated that bone marrow results in a superior outcome compared to PB due to a lower incidence of acute and chronic graft-*versus*-host-disease (GVHD) with bone marrow and a comparable risk of rejection (2.5% for PB and 1.5% for bone marrow)[8,14]. The conditioning regime (Table 3) must be non-myeloablative due to the absence of malignant cells, preserving fertility in young patients and reducing the long-term sequelae after HCT.

Table 3. Conditioning regimen.

	Preferred conditioning regimen
Matched sibling donor	Cy 50 mg/kg/day D-5 to D-2 rATG (cumulative dose 4.5–7 mg/kg, according to institutional protocol)
Matched unrelated donor	Flu 30 mg/m^2/day D-5 to D-2 Cy 60 mg/kg/day D-6 to D-5 rATG (cumulative dose 4.5–7 mg/kg, according to institutional protocol) TBI 200 cGy D-1*
Haploidentical family donor**	Cy 14.5 mg/kg/day D-6 to D-5 Flu 30 mg/m^2/day D-6 to D-2 TBI 400 cGy (single dose) D-1

rATG: rabbit anti-thymocyte globulin; Cy; cyclophosphamide; Flu; fludarabine; TBI; total body irradiation; *the addition of TBI at the dose of 200 cGy reduces the incidence of primary failure, especially in adult and/or polytransfused pediatric patients; **the risk of graft failure was reduced by intensifying the conditioning regimen with an intermediate dose of irradiation (400 cGy). In the HAPLO setting, anti-thymocyte globulin (ATG) should be included in the preparative regimen if the patient has not received any prior immunosuppressive therapy (IST) or if the IST does not include ATG. Source: Elaborated by the authors.

Thus, considering the previously published consensus[5], we recommend a non-myeloablative conditioning for patients with MSD or MUD and bone marrow as the preferred HSC. The GVHD prophylaxis scheme is demonstrated in Table 4.

When using alternative donors with mismatches, it is important to select donors against whom the patient does not have anti-HLA antibodies donor-specific antibodies (DSA).

Table 4. Graft-*versus*-host-disease prophylaxis: according to donor type.

Matched sibling donor and Matched unrelated donor	Cyclosporine 2 mg/kg/dose 12/12 h IV. Level: 150–300 ng/mL. Methotrexate (15 mg/m^2 D+1, 10 mg/m2 D+3, +6, +11)
Haploidentical family donor	Cy 50 mg/kg/d D+3 e D+4 Mycophenolate mofetil 15 mg/kg/dose three times/day D+5 to D+35 Tacrolimus or cyclosporine D+5 until D+365

Source: Elaborated by the authors.

For children and young adults who lack a suitable and readily available donor, IST has traditionally been used as the first-line therapy due to its low toxicity and overall response rates of 30–40%. The standard first-line IST in Brazil remains rATG and cyclosporine. However, complete response (CR) rates are limited, life-threatening infections pose a risk, and outcomes are further influenced by relapse rates of 30%, and a 10 to 15% chance of clonal evolution to myeloid malignancy. Although eltrombopag (EPAG) has recently emerged as a promising non-transplant therapy for SAA, reports on the efficacy of EPAG in children with SAA are limited. Currently, the available data do not support adding EPAG to IST for children with SAA[12,15].

SECOND-LINE TREATMENT OF ACQUIRED SEVERE APLASTIC ANEMIA

Patients with SAA who do not have an available MSD or who are refractory or have relapsed after IST should undergo MUD or HAPLO transplantation. Conditioning for MUD should be based on fludarabine, cyclophosphamide, and a low dose of total body irradiation (TBI), similar to that used in first-line treatment[3,12]. Patients without a suitable MUD or HAPLO may be candidates for other alternative HCT options, such as mismatched unrelated donors (MMURD) or unrelated UCB. Although these alternative HCTs can be curative, they pose challenges, including higher risks of graft rejection, infectious complications, and GVHD, especially when compared to MSD or MUD-HCT[5,12,16].

As reported in the 2021 consensus, unrelated UCB transplantation may serve as a treatment option for children lacking a donor who face emergency situations. While early studies on unrelated UCB-HCT in patients with SAA indicated limited success, subsequent studies demonstrated excellent OS in patients treated with a conditioning regimen consisting of fludarabine, cyclophosphamide or melphalan, and low-dose TBI, without ATG[17]. A French group noted similar results in a prospective study that used conditioning with fludarabine, cyclophosphamide, and a low dose of TBI with ATG, resulting in a two-year OS rate of 81% and an engraftment rate of 88%[17].

Currently, HAPLO-HCT is considered an acceptable treatment option for either first-line or second-line therapies. In the context of second-line treatment, the choice between a MMUD or a HAPLO must be made based on individual circumstances: urgency of the transplant, neutrophil count, age of the recipient, characteristics of the donor (age, gender, and ABO/CMV compatibility), and the presence of DSA. Based on the Brazilian experience, the recommended conditioning regimen for HAPLO-HCT consists of fludarabine, cyclophosphamide, and an intermediate dose of TBI (400 cGy), similar to that used in the first-line treatment. Increased doses of TBI have been associated with a reduction in the primary graft rejection rate, from 27 to 7% ($p = 0.02$), and a higher two-year event-free survival rate, from 60 to 88% ($p = 0.01$)[18]. The role of rATG in conditioning for HAPLO HCT remains controversial; it is typically considered for those who have not received ATG during immunosuppressive treatment as recommended in the first-line therapy. Interestingly, patients with unrelated donors, especially those with MMUD, may benefit from using the HAPLO platform conditioning with post-transplant cyclophosphamide[19].

CHIMERISM ANALYSIS AND FOLLOW-UP

The occurrence of mixed chimerism, common after HCT for SAA, is a predictor of secondary graft failure (SGF)[20]. Serial monitoring of chimerism should be performed at one, two, three, six, and 12 months following HCT. More frequent monitoring should occur if there is a progressive loss of chimerism or onset of a new cytopenia[4]. IST should be maintained for at least 12 months after HCT. Withdrawal should be performed

gradually and ideally with chimerism monitoring following dose reduction. Strategies described to prevent SGF in progressive loss of chimerism include adjusting immunosuppression while maintaining a serum cyclosporine level between 150 and 300 ng/mL or a serum tacrolimus level between 10 and 15 µg/mL, as well as using other drugs such as mycophenolate mofetil or glucocorticoids for no longer than three months[4,20]. Other causes of SGF need to be investigated, such as viral infections, before adjusting immunosuppression. In cases of SGF, a second transplant can be performed with acceptable results.

Follow-up should include pulmonary, cardiac, endocrine, ophthalmic, immune, and renal assessments at least once a year. Particular attention must be given to growth and pubertal development, since many patients receive TBI[21]. Gonadal dysfunction, growth disturbance, avascular necrosis, hypothyroidism, and cataracts are some of the consequences described after HCT. Surveillance for malignancies should also be part of the long-term follow-up[21].

CONFLICT OF INTEREST

Nothing to declare.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable.

AUTHORS' CONTRIBUTIONS

Substantive scientific and intellectual contributions to the study: Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Conception and design:** Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Analysis and interpretation of data:** Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Technical procedures:** Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Statistics analysis:** Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Manuscript writing:** Darrigo Junior LG, Loth G, Fernandes Júnior VCA, Antunes AA, Landi GGF and Bonfim C. **Final approval:** Darrigo Junior LG.

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