Table 2. Patient characteristics. II.

Pts.	Catheter infection	Primary treatment	Response	Secondary treatment	Outcome
1	yes	imipenem+amikacin	refractory	CR	cure
2	yes	imipenem+amikacin	refractory	CR	cure
3	yes	imipenem+amikacin	cure	none	cure
4	yes	ceftazidime+amikacin	refractory	CR	cure
5	yes	ceftazidime+amikacin	relapse	CR	cure
6	yes	ceftazidime+amikacin	cure	none	cure
7	yes	CR	cure	none	cure
8	no	clinafloxacin+CR	cure	none	cure
9	yes	imipenem+amikacin	refractory	CR	cure

CR: catheter removal.

gram-negative infections were documented, including the 9 cases of *A.xylosoxidans* reported herein (1.6% of all patients). This bacteria can be found in aqueous environments, such as disinfectants and fluids. Thus, we might suspect in a common source, but there was no epidemiological relationship between cases. Moreover, three episodes were diagnosed as outpatients.

In our series, the majority of patients had severe neutropenia, and it is possible that mucositis and breakdown of the intestinal barrier allowed invasion of bloodstream by *A.xylosoxidans*. Clinical presentations were highly persistant fever and chills, but neither death nor sepsis syndrome occurred despite poor response to antibiotic therapy in most cases. This is in contrast with other non-fermenting gram-negative infections, which are often associated with high morbidity and mortality.[9]

The antibiotic susceptibility profile of isolates was similar to previous reports,[4,5] with special emphasis on the almost universal *in vitro* resistance to aminoglycosides and aztreonam. As with other non-fermenting gram-negative bacilli, most isolates were susceptible to broad-spectrum β-lactams, co-trimoxazole and fluoroquinolones. The most important conclusion, from our experience, is that these infections are usually catheter-related, and despite their apparently low morbidity, removal of the catheter is generally required for definitive eradication of the microorganism. Appropriate *in vitro* antibiotic therapy may be ineffective or lead only temporary control of the infection.

Key words

Bacteremia, hematologic malignancies

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Unexpected late graft failure 9 months after HLA-identical bone marrow transplant (BMT) for chronic myeloid leukemia (CML): treatment with a second BMT

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We describe a patient with CML in 1st chronic phase (CP) who experienced a graft failure 9 months after an HLA genotypically identical sibling BMT. Drug toxicity, viral infections, chronic graft-versus-host-disease (GVHD) or leukemic relapse were excluded. Chimerism study showed 85% of donor marrow cells. She underwent a second BMT, reengrafted but died of grade IV acute GVHD.

Late graft failure (LGF) after allogeneic BMT is defined as pancytopenia with marrow hypoplasia after complete engraftment.[1] It is observed after haploidentical or T-cell-depleted BMT and in heavily transfused aplastic anaemia patients receiving identical grafts.[2,3] The reported incidence of this event is about 0.4%.[3] Cyclosporine withdrawal, interferon-α treat-

ment, human herpes virus-6 (HHV-6) infection, chronic GVHD, increasing recipient's age or lower dose of BM cells infused,[1,4-7] have been implicated.

A 44-year-old female was diagnosed of CML in CP on July 1993. She was treated with hydroxyurea and subsequently interferon-α2b for 24 months without cytogenetic response. On January, 1996, she underwent a BMT from her genotypically identical brother conditioned with busulphan (16 mg/kg) and cyclophosphamide (120 mg/kg) and 4.81×10^8 unmanipulated bone marrow (BM) mononuclear cells (MNC) per kg of recipient were infused. GVHD prophylaxis consisted of cyclosporine and short methotrexate. She reached complete engraftment with complete cytogenetic chimerism on day +90. Chronic GVHD screening was negative and cyclosporine was withdrawn on day +180. On day +258, isolated thrombocytopenia of 61×10^9/L was detected, that evolved to pancytopenia (leukocytes 0.6×10^9/L, hemoglobin 9.6 g/dL and platelets 2×10^9/L) in 12 days. Physical exam revealed fever and herpetic lesions on nose and lips. Biochemical parameters, including serum B_{12} and folic acid levels, liver function tests and LDH, were normal. Autoimmunity screening, Coombs' and Ham's tests were negative. Chest-x-ray was normal. Red blood cell group and antigens were of donor type (O^+). A marrow aspirate was very hypocellullar with lymphocytes, hystiocytes, plasma cells and mastocytes. Genomic amplification studies by PCR in marrow specimen were negative for herpes viruses (HSV-I and II, CMV, EBV, HZV, HHV-6) and Parvovirus B19. BM cariotype was 46 XY and RT-PCR of bcr/abl transcripts was negative. Chimerism study by PCR of VNTR loci on MNC from BM revealed 85% of MNC of donor and 15% of receptor. Hemotherapy, broad spectrum antibiotics, amphotericin-B and acyclovir were administered.

Idiopathic LGF was diagnosed. She underwent a second BMT from the same brother conditioned with cyclophosphamide (50 mg/kg/day for 4 days) and ATG (30 mg/kg/day for 3 days), with infusion of 4.6×10^8 unmanipulated marrow MNC per kg of recipient 17 days after LGF observation. Cyclosporine with short methotrexate were again given. She recovered >1.0×10^9/L neutrophiles on +15 and >50×10^9/L platelets on +40. Acute GVHD of skin and liver developed and she was treated with corticosteroids (2 mg/kg/day). On +33, she was discharged in good condition with 6.8×10^9/L leukocytes and 43×10^9/L platelets. BM karyotype was 46 XY and 100% of BM cells were of donor origin. She was readmitted on day +51, with nausea, vomiting and slight diarrea. Intravenous nutrition and increased immunosuppression were given under suspicion of GVHD progression. CMV antigenemia was detected and Gancyclovir started. Her condition worsened, with liver function deterioration (bilirubin 40 mg/dL), gastrointestinal bleeding and hepatic encephalopathy. She died on day +75. Autopsy was denied.

LGF has been rarely described after unmanipulated grafts for CML.[5,6] In previously reported cases of LGF occurring more than 6 months after BMT, a cause could be found (T-cell depletion, HHV-6 infection, chronic GVHD or leukemic relapse).[5,6,8] The mechanism of LGF is unknown.[11] Residual host lymphocytes with *in vitro* inhibitory effect against donor hemopoietic cells have been sometimes detected.[6] Second transplants have a high transplant-related mortality in this condition[2,10] and immunosuppression treatment alone or combinated with stem cells reinfusion or hematopoietic growth factors.[1,4,5,8,9] frequently induce autologous hematopoietic recovery.[1,8]

In our patient, a second BMT was decided because of the long interval between first BMT and graft failure and good patient's performance status. Engraftment was successful but lethal GVHD developed. The optimal approach to manage this complication is unclear and reports are scarce and heterogeneous.

Key words

HLA-identical BMT, chronic myeloid leukemia, graft failure

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Primary orbital lymphoma: contralateral relapse after six years in complete remission

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We report a patient diagnosed of an intermediate-grade primary orbital lymphoma with relapse in the other orbit after six years in complete remission (CR).

Primary orbital lymphoma (POL) comprises about 5-10% of all orbital neoplasms.[1] Most common symptoms are exophtalmus and diplopia.[2] POL is usually diagnosed at early stage, and shows low to intermediate-grade histology. Radiotherapy (36-40 Gy) is a successful treatment in most patients, so this entity has a favorable prognosis, with long free disease survival.[3-5] However, we report a patient diagnosed of an intermediate-grade POL with relapse in the other orbit after six years in complete remission (CR).

A 35-year-old man with persistent right exophtalmus and visual impairement, was diagnosed of intermediate-grade POL after undergoing biopsy of a retrocular mass. The extension of disease was evaluated by computerized tomography (CT) scan and magnetic resonance (MR). No other lymphomatous locations were found. CR was achieved after systemic chemotherapy and local radiotherapy (40 Gy). After 6 years, left exophtalmus was noticed. A left orbital mass was detected by MR. The histological examination revealed the same intermediate-grade pattern.The imaging diagnosis showed no spread disease. Chemotherapy and radiotherapy were administered. Nowadays the patient remains in CR.

We have not found any other reference in the literature about contralateral relapse of POL. However, although POL usually shows indolent course and good prognosis, we suggest a long term folllow up, in order to diagnose late relapse .

Key words

Orbital neoplasms, relapse, extranodal lymphoma

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Recent advances in myelodysplastic syndromes (MDS)

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This year Haematologica reports a series of review articles on *Recent Advances in Myelodysplastic Syndromes*: the first one appeared in the January issue,[1] the second one is found in this issue.[2] Future articles will analyze prognostic factors, secondary MDS and therapy of these disorders. The basis for this series has been the *Fourth International Symposium on Myelodysplastic Syndromes* held in Barcelona, Spain, on April 24-27, 1997. The Meeting organizers – Guillermo F. Sanz, Miguel A. Sanz and Teresa Vallespi – have done a remarkable job as Guest Editors. In 1997 Haematologica published several articles on MDS[3-12] and is now proud of publishing this series, which will hopefully appear also as a separate print and electronic volume.

Key words

Myelodysplastic syndromes

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