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Guillain-Barre Syndrome Associated with Rapid Immune Reconstitution Following a Tandem Autologous Hematopoietic Stem Cell Transplantation. Study of a Case and Review of the Literature

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Abstract

Introduction: Guillain-Barré syndrome (GBS) is a frequent cause of neuromuscular paralysis and usually occurs after an immunoallergic reaction, most often after respiratory or gastrointestinal infection. However, rare cases of Guillain-Barré syndrome are described, associated with hematopoietic stem cell transplantation.

Observation: Patient n° 20452690. The authors report the case of 21 years old man, treated for Hodgkin lymphoma with a recurrence at 18 months, who enjoyed second autologous hematopoietic stem cell transplantation after conditioning chemotherapy, then developed a Guillain-Barré syndrome. Plasma exchanges were made before a neurological worsening. Thereafter, five cycles of intravenous immunoglobulin were performed monthly. Unfortunately this development was hampered by mechanical respiratory complication that led to the death of the patient, the 237th day of evolution. The final diagnosis of Guillain-Barré syndrome, occurred in a context of post-transplant immune reconstitution in a patient treated for Hodgkin lymphoma was retained.

Discussion: GBS is a rare complication of hematopoietic stem cell transplantation. After literature review, we collected 33 cases related to this disease, 10 occurred after autograft. Etiopathogenic mechanisms remain obscure. Despite the use of immunomodulatory treatment, the prognosis is often severe and dark.

Conclusion: GBS is possible after autologous hematopoietic stem cell transplantation indicated for the treatment of Hodgkin lymphoma. The initial clinical severity, despite safeguards and use of immunomodulatory therapy, will cause often deleterious and poor outcome. To our knowledge, this well documented observation is the first case indicated for Hodgkin lymphoma.

Keywords: Guillain-Barré syndrome; Autologous hematopoietic stem cell transplant; Hodgkin disease; Plasmatic exchanges.

Introduction

Guillain-Barré syndrome (GBS) is a rare complication in the setting of hematopoietic stem cell transplantation (HSCT), either autologous [1] or heterologous [2]. Its pathogenicity remains unclear; an immune reconstitution inflammatory syndrome can be suspected [3]. We report the case of a patient presenting with acute polyradiculoneuropathy after autologous bone marrow transplantation. To our knowledge, it is the first case of acute polyradiculoneuropathy in a context of Hodgkin lymphoma described in literature.

Case report

A 21-year-old Caucasian man was diagnosed in May 2010 with a stage IIA mediastinal and cervical Hodgkin lymphoma. He was initially treated with four cycles of ABVD chemotherapy, associating doxorubicin, bleomycin, vincristine and dacarbazine. This chemotherapy was followed by radiotherapy on neoplastic areas and lymph nodes, especially in left costodiaphragmatic zone. A 20 Gy dose with focal radiotherapy was performed, with a 28 Gy total cumulative dose. In November 2011, the disease relapsed (stage IV A), located on previously irradiated areas. The patient received a DHAP second-line chemotherapy, associating cisplatin, cytarabine, and dexamethasone. Four cycles were performed, followed by cytapheresis, with a view to prepare first autologous bone marrow transplantation after BEAM polychemotherapy (BCNU, etoposide, melphalan). This transplantation was performed on February 13, 2012; he underwent a second intensified chemotherapy consisting of busulfan, etoposide, melphalan (BAM), in order to prepare another transplantation (June 12, 2012). 57 days later, the patient presented with rapidly progressive motor dysfunction. In previous days, it was not described in the infectious process, especially cough or diarrhea. Neurological examination, performed at the patient's admission to the fifth day of evolution noticed a motor deficit with tetraparesis (muscular evaluation 2/5) and ataxia. Protopathic and epicritic sensibility were normal, tendon reflexes were absent, with no Babinski sign. Cranial nerves examination shows a bilateral facial palsy and lower cranial nerves impairment (palate hypoesthesia and abolition of the cough reflex). Hypophonia was noticed, and there were no oculomotor disorders. Polypnea (respiratory rate more than 30 cycles per minute), impaired respiratory amplitude and decreased peak-flow value (250 liters per minute) were observed. There were no other clinical abnormalities, and no dysautonomia (normal heart rate, and blood pressure stability). Routine blood examinations were unremarkable (no signs of inflammatory process, normal blood count, coagulation, hepatic tests, ionogramme, TSH). Cerebrospinal fluid (CSF) showed albumino-cytologic dissociation, with increased protein level (3.4 g/L), low glucose and cellularity. Cytopathology was normal

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(especially no lymphoma cells), as well as bacterial cultures. Data from the electromyogram discussed an acute polyradiculoneuropathy: motor distal latencies were diffusely delayed; motor and sensory conduction velocities were slowed while the amplitudes were reduced without proximal or distal conduction blocks. F-waves were delayed (Table 1). Needle examination recorded neurogenic muscle pain. Chest and abdominopelvic CT-scan were unremarkable with any lymphadenopathy. Microbiological and viral investigations were of no use, only demonstrating former contacts with cytomegalovirus, Epstein-Barr virus, rubella virus, and toxoplasma. Other virus or bacterial serologic tests were negative (Herpes virus type I and II, HHV-6, varicella, HIV, hepatitis (A, B, C) virus, seasonal and H1N1 influenza virus, Lyme disease, campylobacter jejuni, chlamydia, Yersinia pestis, Q-fever, haemophilus influenzae). Blood samples did not show any hepatic dysfunction, so hepatitis E was excluded. PCR assay in CSF was negative for varicella and herpes virus. Auto-immune analyses were normal, as well as anti-ganglioside antibodies, especially anti-GM1 and GM2. Onconeural antibodies were negative both in blood and CSF. The initial treatment of this polyradiculoneuropathy was based on four plasma exchanges (every two days), performed in intensive care unit (UCI). Clinical examination at week 3 showed a mild improvement of tetraparesis, ataxia, and muscular evaluation especially in the pelvic area. At the end of week 4, a dysautonomic syndrome appeared; then the patient presented an acute respiratory distress syndrome (ARDS), followed by cardiac arrest. He was resuscitated and intubated, then readmitted to UCI. CSF showed albumino-cytologic dissociation, with increased protein level (1.3 g/L). Cell count and cytopathology were normal. No signs of lymphoma reactivation were observed: chest and abdominopelvic CT-scan and PET scan were unremarkable. Anti-ganglioside and onconeural antibodies remained negative. 13 days later, after a period of hemodynamic dysfunction (requiring vasopressor and cardiotonic support), weaning from mechanical ventilation was begun firstly on laryngeal tube, then on tracheotomy (performed on September 25). Numerous infectious complications were iteratively treated, including ventilator-associated pneumonia generating two episodes of ARDS. Initial polyneuropathy was complicated by neurological impairment due to weight-loss and ICU-acquired neuromyopathy (multifactorial: curarizing drugs, corticosteroids, sepsis). Plasma exchanges were not indicated in this context, prompting to use IV immunoglobulin 0.4 g/kg/day (16 g/day during five days). Five cycles were performed monthly, from September 2012 to January 2013, with a positive effect as regards clinical and electromyographic examination. Hence, in January 2013, muscular testing reached 3/5, and the patient was beginning to recover an efficient tonus of the trunk muscles allowing chair position. The CSF control analysis, six months after the beginning, was normal. This slow improvement of neurological, respiratory and nutritional status prompted to consider a transfer to post-ICU unit. Unfortunately, an acute mechanical complication occurred: a ventilator-associated severe bilateral tension pneumothorax, complicating an iatrogenic diffuse pulmonary fibrosis. The patient died 231 days after the beginning of the disease, in a context of multi-organ failure. Diagnosis was severe GBS. The absence of preceding infectious episode and the onset concomitant with immune reconstruction following autologous bone marrow transplantation in a context of Hodgkin lymphoma, prompt to emphasize the deleterious potential effects of immune reconstruction on the pathogenesis of this polyradiculoneuropathy.

Discussion

GBS is a polyradiculoneuropathy characterized by rapidly progressive symmetrical ascending motor weakness: muscles associated to long-fibers nerves are firstly impaired, with loss of deep tendon reflexes, with mild motor deficit, but impairing variably autonomic nervous system. Weakness usually develops subacutely (up to four weeks), then reaches a plateau followed by clinical resolution. Pathogenesis remains incompletely understood, involving immuno-allergic mechanisms. Thus, GBS is usually associated with antecedent bacterial or viral infections. It can also occur concomitantly with neoplasms, blood or immune diseases [4]. GBS has previously been reported in the setting of GCSH, with isolated cases reports; multiple cases reviews are rare [1,5-9]. It occurs with a frequency of 0.3%-0.7%, more often in case of allogeneic transplantation. A complete review of literature, based on the Medline database, enhanced with two personal case-reports, was performed by Zang et al. [1]. Nevertheless, we did not take account the cases 40, 52 and 58, which rather described chronic polyneuroradiculopathies, which may occur as well [10]. Since few cases were discribed [2,11]. Thone et al. [12] described a new case that we did not take into account considering the very long interval between GBS and allogeneic transplantation (more than 10 years); the discussion was mainly focused on host-versus-graft reaction [12]. So we identified 33 cases (Tables 2 and 3) [1-3,5-9,11,13-26]: 23 GBS after allograft, 10 after autograft. Median age was just about 32 years for both allo and autograft with M/F sex-ratio 2.3. The time span, preceding the onset of neurological symptoms, range from two days [9,23] to 45 months (18), with an average of 4.3 months. Heterologous grafts were primarily indicated in the treatment of leukemia (acute lymphoid or acute/chronic myeloid). Other indications emerged: acute [1] and chronic myelodysplastic syndromes [20], Hodgkin's [8] or non-Hodgkin's lymphoma [9]. GBS after autologous graft are more rarely observed, with similar indication. Other indications of autograft causing GBS have been described: multiple myeloma [1] and metastatic breast neoplasia [24]. To our knowledge, we report the first case occurring after autologous graft for Hodgkin's lymphoma.

	Right median nerve	Right ulnar nerve	Right common fibular nerve	Right tibial nerve
Motor nerve				
Latency	8.5	4.8	11.9	9.4
Velocity	40.7	42.4	30.1	33.6
Distal Amplitude	3.1	3.5	0.7	1.4
F waves latency	31.5	98.5	30.2	86.3
	Right median nerve	**Right ulnar nerve**	**Right superficial fibular nerve**	**Right sural nerve**
Sensitive nerve				
Latency	3.1	2.6	2.8	3.1
Velocity	41.9	40.4	32.1	35.5
Amplitude	1.3	1	3.6	2.5

Table 1: Electrophysiologics datas.

References	Age/Sex	Initial disease	conditioning of chemotherapy	Pre transplant Chemotherapy	Time to onset after transplantation	GVHD	Evolution	Infection	Treatment
[13]	19/M	CML	MRD, BM	TBI/AraC	1 week		Improvement	None	None
[14]	25/F	AML	MRD, BM	CY/TBI	6 weeks	Positif	Death	None	Steroids
[5]	14/M	CML	MRD, BM	TBI/CY/AraC	2 months		Death	CMV, HSV	PE
[5]	37/F	ALL	MRD, BM	TBI/CY/Mel/VP/ AraC	1 month	Positif	Death	None	Non done
[15]	57/M	MDS	MRD, BM	BUCY	12 months	Positif	Improvement	Campylobacter	IVIg
[16]	41/M	CML	URD, BM	CY/TBI	3 months	Positif	Improvement	Campylobacter	PE, IVIg EP, IgIV
[17]	42/M	CML	BM	BUCY	4 months	Positif	Improvement	CMV	PE, ganciclovir
[18]	34/M	CML	MRD, BM	ND	45 months		Regression	None	PE, Steroids
[8]	34/F	CML	BM	CY/AraC/TBI	5 months	Positif	Regression	None	PE
[8]	27/M	HL	BM	CY/BCNU/VP	15 months		Regression	None	PE
[8]	34/M	AML	BM	CY/TBI	4 months	Positif	Improvement	None	PE
[8]	59/F	CML	BM	CY/TBI	11 months	Positif	Death	None	IVIg, PE
[19]	26/M	CML	MisMRD, PB	Fluda/BU/TBI	3 months	Positif	Death	CMV, Toxoplasma	IVIg, Foscarnet, Ganciclovir
[9]	16/M	ALL	URD, BM	Ara/CCY/TBI	6 days		Death	Parainfluenzae 1	IVIg
[9]	17/M	NHL	URD, BM	Ara/CCY/TBI	3 days		Death	None	IVIg
[9]	18/M	ALL	URD, BM	Ara/CCY/TBI	2 days		Death	None	IVIg
[20]	49/M	MDS	MRD, PB	Samarin/BU/Fluda	3 weeks		Death	EBV	IVIg, Acyclovir
[21]	23/F	AML	MisMRD, PB	CY/TBI	6 weeks		Death	CMV	IVIg, PE, Ganciclovir
[22]	24/M	AA	MRD, BM + PB	CY	11 weeks	Positif	Regression	HSV, Candida	IgIV
[3]	34/F	ALL	MisMRD, PB	BUCY	11 weeks		Regression	CMV	IVIg, Ganclovir
[1]	39/F	AML	URD, PB	AraC/ Daunorubicine/ VP	5 months		Death	None	IVIg
[11]	43/M	CML	URD, BM	Treosulfan-Fludarabine-ATG	84 jours days	Positif	Regression	HHV6	IVIg, Foscarnet
[2]	18/M	ALL	URD, PB	Dasatinib/ CsA	48 days	Positif, cutaneous	Regression	HHV6	IVIg, Foscarnet, Ganciclovir

HSCT: Hematopoietic Stem Cell Transplantation; M: Male; F: Female; CML: Chronic Myeloid Leukemia; AML: Acute Myeloid Leukemia; ALL: Acute Lymphoblastic Leukemia; MDS: Myelodysplastic Syndrome; HL: Hodgkin Lymphoma; NHL: Non- Hodgkin Lymphoma; AA: Aplastic Anemia; MRD: Match-Related Donor; BM: Bone Marrow; URD: Unrelated Donor; misMRD: Mismatched-Related Donor; PB: Peripheral Blood Stem Cell; TBI: Total Body Irradiation; AraC: Aracytine; CY: Cyclophosphamide; Mel: Melphalan; VP: Etoposide; BU: Busulfan; ND: Non Done; Fluda: Fludarabine; ATG: Antithymocyte Globulin; CsA: Cyclosporine A; GVHD: Graft Versus Host Disease; CMV; Cytomegalovirus; HSV: Herpes Simplex Virus; EBV: Epstein Barr virus; HHV6: Human Herpes Virus 6; PE: Plasma Exchange; IVIg: Intravenous Immunoglobulin

Table 2: Review of SGB cases of GBS occurring after allogenic HCST.

References	Age/Sex	Initial disease	conditioning of chemotherapy	Pre transplant Chemotherapy	Time to onset after transplantation	GVHD	Evolution	Infection	Treatment
[23]	40/M	NHL	BM	CY/BCNU/VP	7 days		Non done	None	PE, Steroids
[7]	54/F	Breast cancer	BM	CY/Thiotepa	7 weeks		Imporvement	None	PE
[7]	32/F	Breast cancer	BM	CY/Thiotepa	3 weeks		Death	None	PE
[24]	44/F	Breast cancer	BM+PB	CY/CDDP/BCNU	2 days		Non done	None	PE, IVIg
[25]	20/M	NHL	PB	BUVP	3 weeks	Positive	regression	None	Steroids
[26]	49/M	CML	PB	BU/Mel	8 weeks		Improvement	None	IVIg, PE
[6]	7/M	AML	BM	BU/Mel	2 weeks		Regression	None	IVIg
[6]	7/F	ALL	BM	None	4 weeks		Regression	None	INIg
[1]	47/M	MM	PB	Vincristine, Doxorubicin	9 months		Regression	None	IVIg
Personal case	21/M	HL	BM	CDDP/AraC, then BCNU/VP/AraC/Mel	57 days		Death	None	PE, IVIg

HSCT: Hematopoietic Stem Cell Transplantation; M: Male; F: Female; NHL: Non- Hodgkin Lymphoma; CML: Chronic Myeloid Leukemia; AML: Acute Myeloid Leukemia; ALL: Acute Lymphoblastic Leukemia; MM: Multiple Myeloma; HL: Hodgkin Lymphoma; BM: Bone Marrow; PB: Peripheral Blood Stem Cell; CY: Cyclophosphamide; CDDP: Cisplatin; BU: Busulfan; VP: Etoposide; Mel: Melphalan; PE: Plasma Exchange; IVIg: Intravenous Immunoglobulin

Table 3: Review of Cases of GBS occurring after autogenic HCST.

Etiopathogenic factors remain unclear: a rapid onset of neurological symptoms, upon one week after the first graft, is often imputed to high-doses chemotherapy, especially when cytosine arabinoside, or very similar drugs, are involved [9]. Indeed, these products may induce nervous lesions by itself, as well as a higher sensitivity to antigens activated by immunoreactive cells in case of infection. In case of GBS occurring after graft for chronic myeloid leukemia, the use of interferon-alpha was reported as being involved in neurological dysfunctions, most often peripheral [18]. Except for these precocious toxic causes, physiopathology of GBS in this context remains obscure. An infectious etiology must be suspected [1], such as viral infection (Cytomegalovirus, Epstein-Barr virus, Herpes virus, Parainfluenza virus), as well as bacterial, with two confirmed cases involving Campylobacter jejuni [15,16]. Other causes are also separately reported with the questioning

of HHV6 virus for the most recent cases [2,11]. Co-infections are also reported: Herpes virus and Candidosis [22], Cytomegalovirus and Herpes virus [6], or Cytomegalovirus and Toxoplasma [19]. However, as illustrated in our case-report, a preceding infection is most often not observed: for each of the 20 autologous grafts, no pathogenic agent could be found. Other contributing factors have been reported, such as cerebrospinal irradiation [27]. In case of allogeneic transplantations, a graft-versus-host disease should be evoked [12]: it was observed in 16 out of the 23 allogenic grafts. Graft-versus-host diseases are not reported in case of autografts, even if a graft reaction is possible, then concerning teguments but not nervous system [25]. None of these hypotheses were relevant for our patient: he presented with complete remission of Hodgkin's lymphoma and no relapse was noted despite repeated examinations, both clinical and paraclinical. A possible paraneoplastic hypothesis was also excluded. Repeated viral, bacterial or parasitic serology remained negative or showed no recent contact. Teguments were not affected, excluding graft-versus-host disease. The 57-days' time gap between the end of chemotherapy and the beginning of symptoms of polyradiculoneuropathy, does any advocate for a toxic mechanism of the Aracytine.

Therapeutic strategy is not very different from GBS treatment: it requires immunomodulatory therapy, either IV immunoglobulins or plasmapheresis, possibly alternately. The review of literature finds out nine cases of plasmapheresis used alone (six after allograft, three after autograft). IV immunoglobulins were more commonly used (24 patients), eleven times after allograft, three times after autograft. These treatments were combined six times, three times after allograft, three times after autograft. An old paper reports a single case of a patient presenting with graft-versus-host disease, who was treated with corticosteroids, following cyclosporine [14]. Hence, corticosteroids are rarely used, only indicated in the context of graft-versus-host disease after allograft [18]. For autograft, they were occasionally used, alone [25] or combined with plasmapheresis [23]. Only one patient recovered spontaneously, however incompletely [13]. Finally, in one case, the treatment was not reported [6]. If an infectious agent is identified, a specific treatment can be initiated, such as ganciclovir for cytomegalovirus [3], aciclovir for herpes simplex virus [20], or foscarnet for human herpes virus 6 [2]. Outcome is inconstant, but clinical presentation is mostly severe, with 50% mortality. Eleven patients died after allograft, and two after autograft, over a time interval ranging from two weeks [20] to seven months (personal case). Survivors presented with sequel ranging from complete recovery to motor sequelae requiring walking assistance [1].

Conclusion

This case report illustrates the possible onset of a GBS following immune restoration after autologous hematopoietic stem cell transplantation, based on clinical, paraclinical, electrophysiological or biological arguments. We emphasize that this diagnosis requires stringent elimination of other causes of GBS, including neoplastic or infectious causes. Therapeutic strategy against this neurological complication is not different from other acute polyradiculoneuropathies care, based on plasmapheresis or IV immunoglobulins. Nevertheless, the prognostic remains poor with unfavorable outcome in one case out of two.

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