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Case Report

Multiple Myeloma and Chronic Myelogenous Leukemia; an Uncommon Coexistence in 2 Patients, with Literature Review

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Abstract

The coexistence of Multiple Myeloma (MM) and Chronic Myelogenous Leukemia (CML) is an extremely uncommon event that has only been reported in very few cases. Here, we present 2 new cases of concurrent MM and CML. The case of 63 year old woman with CML, who was treated with imatinib mesylate and subsequently developed MM 6 years after the diagnosis of CML, and the case of a 68 year old man with MM, who was treated with radiation therapy and chemotherapy and subsequently developed CML 4 years and 7 months after the diagnosis of MM. The relationship between CML and MM, is also discussed.

Keywords: Multiple myeloma; Chronic myeloid leukemia; Cell transformation

Introduction

Multiple Myeloma (MM) is a malignancy of lymphoid origin, characterized by monoclonal proliferation of malignant plasma cells in the bone marrow microenvironment, monoclonal protein in serum and/or urine and associated organ dysfunction [1]. The diagnosis is based on the presence of at least 10 % bone marrow infiltration by clonal plasma cells that are usually secreting a monoclonal immunoglobulin in addition with or without end organ damage manifestations [1-3]. Chronic Myeloid Leukemia (CML) is a clonal disorder of myeloid origin characterized by a chromosomal reciprocal t(9;22) (q34;q11) translocation, the Philadelphia (Ph) chromosome [4]. The crucial pathogenetic consequence of this translocation is the creation of a chimeric BCR/ABL fusion protein, resulting in a constitutively active tyrosine kinase with high proliferative potential [5,6].

Therefore, the respective malignant cells in MM and CML are completely different, being lymphoplasmacytic and myeloid cells, accordingly. The occurrence of MM and CML in the same patient, either at diagnosis or with one or the other disease pre-existing, is a rare event that has previously been reported in a limited number of case reports in the literature [7–25], and the question of the origin of these 2 malignancies in such patients, remains unanswered.

In this report, we reviewed previously published cases, and presented 2 more patients with concurrent MM and CML.

Patient 1

A 63 year old woman was referred to our hospital in October 2002 because of leukocytosis. She had a history of hepatits A, hysterectomy for benign fibroids (1996), and colon cancer (1999) in complete remission after surgical resection and chemotherapy. She had no symptoms and no clinical findings.

Peripheral blood analysis revealed white blood cell count of $17.8x10^9$/l, (64% segmented neutrophils, 7% basophils, 4% myelocytes, 2% metamyelocytes, 3% band forms, 18% lymphocytes, and 2% monocytes). Haemoglobin was 12.8gr/dl, and platelets were $339x10^9$/l. Neutrophil Alkaline Phosphatase (NAP) was 128, and Lactate Dehydrogonase (LDH) elevated (765U/L). Bone Marrow (BM) aspirate and biopsy were typical of CML; BM karyotype showed Philadelphia (Ph) chromosome in 18 out of 20 metaphases. Molecular analysis by quantitave reverse transcription-polymerase chain-reaction (qRT-PCR) detected the chimeric BCR-ABL messenger RNA, confirming the diagnosis of CML.

The patient was started on imatinib mesylate at standard dose (400mg/day) and achieved complete hematologic and cytogenetic response at 3 months and complete molecular response at 9 months after treatment initiation. Two years after, and although she was still under imatinib treatment, qRT-PCR became positive for BCR-ABL transcripts, and BM karyotype revealed Ph chromosome in 1 out of 20 metaphases; BM biopsy showed additionally 5-8% κ light chain restricted plasma cell infiltration, while a small M-spike was present on serum protein electrophoresis, serum immunofixation showed an IgGκ monoclonality and quantitative Immunoglobulin (Ig) measurements were within normal range; Monoclonal Gammopathy of Undetermined Significance (MGUS) was diagnosed. Imatinib mesylate dosage was increased to 600mg/day, resulting again in a complete molecular response.

While under follow-up and regular CML evaluation with BM karyotypes and BCR-ABL quantification, and although she remained in complete molecular CML remission, her karyotype presented in 2005 an additional abnormality del (7)(q32) and two more in 2006 [t(1;7)(q32;q21) and t(2;7)(q33-35;q21)]. At that time BM examination showed 12% infiltration by clonal plasma cells (Table 1). As she had no MM-related symptoms, the diagnosis of asymptomatic MM (AMM) was made.

In October 2012, BM biopsy revealed 80% monoclonal plasma cell infiltration, IgG and serum FLC-κ quantification were steadily increasing (Table 1) and 6 months later she presented severe bone

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Table 1: Laboratory follow up tests (of patient 1), concerning CML & MM.

Date	Karyotype	% BCR-ABL	BM PC infiltration	IgG (mg/dl)	FLCκ (g/L)	FLCR	Diagnosis	
10/15/2002	46,XX [2] 46,XX, t(9;22)(q34;q11)	yes	0%				CML	Imatinib 400mg
2/20/2003	46,XX [20]	yes					CML	Imatinib 400mg
7/15/2003	46,XX [20]	yes					CML	Imatinib 400mg
10/7/2003		0.0005	no clonal plasmacytes				CML	Imatinib 400mg
11/30/2004	46,XX [19] 46,XX, t(9;22)(q34;q11)	yes	5%	980	50	7	CML+MGUS	Imatinib 600mg
4/5/2005	46,XX [19] 46,XX del(7)(q22) [1]						CML+MGUS	Imatinib 600mg
6/6/2006	46,XX [16] 46,XXt(1;7)(q32;q21) 46, XX t(2;7)(q33-35;q21)	yes	12%				CML+AMM	Imatinib 600mg
3/29/2007	46,XX [17] 46,XX t (1;7)(q32;q21) [3]	not detected	10%	1310	296	21.9	CML+AMM	Imatinib 600mg
9/30/2008	46,XX [19] 46,XX t (1;7)(q32;q21) [1]	not detected	50%	1570	349	24.5	CML+AMM	Imatinib 600mg
10/20/2009	46,XX [20]	not detected	15%	1520	427	33.8	CML+AMM	Imatinib 600mg
4/15/2011	46,XX [20]	0.02%	30%				CML+AMM	Imatinib 600mg
6/20/2011		0.03%		1910	337	33.7	CML+AMM	Imatinib 600mg
2/24/2012		not detected		2100	422	40	CML+AMM	Imatinib 600mg
6/26/2012		not detected		1880	889	330.48		Imatinib 600mg
10/24/2012	46,XX [19] 46,XX t (1;7)(q32;q21) [1]	not detected	80%	1760	1130	634.83	CML+SyMM	Vel-Dex
5/3/2013		not detected		2090	1180	1296	CML+SyMM	
10/9/2013	46,XX [23] 46,XX del(7)(q11.2q32) [2]	not detected	40%	1620	686	264	CML+SyMM	Rev-Dex
10/10/2014		not detected	2%	1050	20	1.05	CML+SyMM	Rev-Dex

BM PC: Bone Marrow Plasma Cells; FLCκ: Free Light Chains κ; FLCR: Free Light Chains Ratio; CML: Chronic Myeloid Leukemia; MGUS: Monoclonal Gammopathy of Undetermined Significance; AMM: Asymptomatic Multiple Myeloma; SyMM: Symptomatic Multiple Myeloma; Vel-Dex: Velcade-Dexamethazone; Rev-Dex: Revlimid-Dexamethazone

pains with spontaneous lumbar fractures. Her MM was Symptomatic (SyMM) and she received treatment with Velcade (bortezomib) and Dexamethazone, while imatinib continued. After 4 cycles, MM was in very good partial response but treatment had to be stopped due to severe peripheral neuropathy. The patient deteriorated 3 months after, and 2nd line treatment with Revlimid (lenalidomide) and Dexamethazone was started, additional radiation therapy was also performed for a new fracture. The patient is stable since then. With regard to combined treatment toxicities, it should be reported that after six months of treatment with lenalidomide 25 mg per day and imatinib, the patient presented neutropenia and lenalidomide dosage was reduced to 15 mg per day, while she still sporadically needs granulocyte colony stimulating factor injections in order to keep safe her neutrophils counts.

Patient 2

In April 2008, a 68 year old man with history of hypertension (2003), diabetes (2003), hypothyroidism (2007), and diverticulosis (2005), presented with mild anaemia and spinal bone pains and an elevated monoclonal spike on serum protein electrophoresis. Serum immunofixation showed IgGκ monoclonality, Urine total protein was 1005mg/24h. IgG and serum FLC-λ levels were increased (2400mg/dl and 97mg/L respectively). BM biopsy revealed 40-45% plasma cell infiltration, with λ light chain monoclonality. Fluorescence In Situ Hybridization (FISH) analysis was negative for IgH rearrangements, chromosome 13q and 17p deletion. Lytic lesions and a L5 bone plasmacytoma were present in lumbar spine. The patient had IgGλ SyMM, He was treated with radiation therapy followed by VAD (vincristine, doxorubicin and dexamethazone) resulting in very good partial response.

From September 2008 until October 2012, the disease remained in plateau. He was re-evaluated in October 2012 because of a slight paraprotein increase. BM trephine biopsy and smears revealed limited 10% plasma cell infiltration; however BM karyotype detected the presence of Philadelphia chromosome in 24 of 28 metaphases. BCR-ABL transcripts were also detected confirming the diagnosis of CML. At that time, blood analysis exhibited white blood cell count of $11810x10^9$/l (78% neutrophils, 2% myelocytes, 12% lymphocytes, 5% monocytes, 3% basophils). Haemoglobin level was 11.3gr/dl, and platelet count $417x10^9$/l.

The patient was placed on imatinib mesylate at standard dose, and achieved major molecular response at 9 months after treatment initiation. He is thereafter asymptomatic and clinically stable as concerning both diseases. It should however be mentioned that at the time of imatinib mesylate administration, the patient was still under zoledronic acid bi-monthly adjuvant administration for the prevention of bony manifestations but as serum creatinine increased, we were obliged to discontinue zoledronic acid and renal function returned to normal thereafter.

Literature Review

There are only 19 cases in the literature, in which coexisting MM

Table 2: Coexisting MM and CML cases.

							MM			CML		
	Patient	Reference	Year	Age/Sex	Diagnosis Interval	1st disease treatment	Type	Marrow PC	Bone lesions	WBC count ($x10^9$/L)	Spleno-megaly	Ph+
Diagnosed simultaneously	1	Boots MA et al. [7]	1982	58/M			IgG-k	numerous	yes	140	yes	yes
	2	Tanaka M et al. [8]	1998	72/F			IgG-k	3%	yes	162.4	yes	yes
	3	Alvarez-Larran A et al. [9]	2001	81/M			IgA-k	25%	yes	28.7	NS	yes
	4	Schwarzmeier JD et al. [10]	2003	66/M			IgG-k	3%	yes	171	yes	yes
	5	Wakayama T et al. [11]	2005	85/F			IgG-λ	32.8%	NS	8.1	no	yes
	6	Offiah C et al. [12]	2012	71/F			IgG-k	30%	no	12.7	no	yes
	7	Romanenko NA et al. [13]	2013	64/F			IgA-k	12.4%	no	27.2	NS	yes
MM/+CML	8	Macsween JM et al. [14].	1972	77/M	33 months	no	BJP	9-12%	no	145	yes	yes
	9	Klenn PJ et al. [15]	1993	71/M	24 months	yes	IgG-k	cluster	yes	40.8	NS	yes
	10	Nitta M et al. [16]	1999	70/M	33 months	no	IgG-k	25%	no	25.2	yes	yes
	11	Nakagawa M et al. [17]	2003	47/M	33 months	yes	BJP-k	9.8%	yes	23.9	NS	yes
	12.	Ragupathi L et al. [18]	2013	62/F	17 months	yes	IgG-k	70%	no			yes
CML/+MM	13	Derghazarian C et al. [19]	1974	65/F	113 months	yes	IgG-k	9%	yes	43.	NS	yes
	14	Zoumbos NC et al. [20]	1987	57/M	60 months	yes	light-k	NS	NS	NS	NS	no
	15	Yokota A et al.[21]	2005	71/M	38 months	yes	BJP-λ	74.4%	NS	NS	NS	yes
	16	Garipidou V et al. [22]	2005	68/M	20 months	yes	IgG-λ	25%	no	NS	NS	yes
	17	Galanopoulos A et al. [23]	2009	76/M	14 months	yes	IgA-k	60%	no	NS	NS	yes
	18	Michael M et al. [24]	2009	57/F	65 months	yes	IgA-k	95%	NS	52.38	NS	yes
	19	Ide M et al. [25]	2010	72/F	3 months	yes	IgG-k	21.6%	no	31.3	no	yes

MM: Multiple Myeloma; CML: Chronic Myeloid Leukemia; Marrow PC: Marrow Plasma Cells; Ph+: Philadelphia Positive; WBC: White Blood Cells

and CML have been reported (Table 2) [7-25]. In 7 of the 20 cases, MM and CML were diagnosed simultaneously [7-13]. In 5 of the 20 cases, MM diagnosis preceded that of CML [14-18], while in the rest 7, CML diagnosis preceded that of MM [19-25]. Regarding the interval between the diagnosis of each disease, MM preceded CML from 17 to 33 months, while CML preceded MM from 3 to 113 months. As for treatment after diagnosis of the first disease, anti-neoplastic therapy or radiotherapy was performed in 3 out of 5 and in 7 out of 7 cases, in the MM preceding CML group and in the CML preceding MM group, respectively. No particular disease characteristics were found in literature that could be connected somehow with increased or decreased possibility of these 2 diseases coexisting.

Discussion

The coexistence of MM and CML is an extremely uncommon event. However, there are reports of MM coexisting with a variety of myeloproliferative disorders, including polycythemia vera [26-28], myelofibrosis [28,29], essential thrombocytosis [30,31], and chronic neutrophilic leukemia (CNL) [32-35]. In addition MGUS, a pre-neoplastic plasma cell disorder, has also been reported to develop in pre-existing myeloproliferative neoplasms, while myeloid malignancies have been reported to develop in MGUS patients with accumulative incidence risk of <2% [36-38]. As for CML, in most cases the patient's chronic disease eventually changes to a more aggressive disease, usually towards acute myelogenous leukemia or acute lymphoblastic leukemia [39]. In addition, lymphoma [40-42], monoclonal gammopathy [34,43], or waldenstrom macroglobulinemia [44] could also occur in association with CML.

All the above suggest the existence of a common malignant pluripotent progenitor stem cell, capable of differentiating into both myeloid and lymphoid cell lineages that could lead to the development of CML and MM in the same patient [15,18,33,34]. In favour of this theory, it has been shown that Ph+ B lymphoblastoid cells may be observed in patients with CML even during the chronic phase, and that they may arise from the CML stem cell alone [45]. Furthermore, despite the fact that MM unlike leukemias and lymphomas, typically has no specific chromosomal abnormalities and is characterized by genomic instability [46,47], there have been cases of MM patients with a Philadelphia chromosome [48-50].

Another potential theory is that when CML develops before MM and vice versa, the secondary disease may be caused by the cytotoxic drugs or irradiation used to treat the first disease. In the literature several clinical observations suggest that Imatinib mesylate treatment for CML, could promote the development of MM [18,21-24]. This is further supported by a study in which Imatinib was shown to stimulate MM cell proliferation through activation of the Erk1 and Erk2 Mitogen-Activated Protein Kinases (MAPKs) [51]. Neverthelsess, Imatinib has also been shown to inhibit proliferation of MM cells *in vitro* by arresting cell-cycle progression [51]. Therefore, development

of MM in Imatinib treated CML backround can neither be ruled out, nor can be taken for granted, and since there are only a few reports on this issue, long-term studies are needed. Furthermore, exposure to radiation has been reported to induce t(9;22) translocation [52]. Therefore, CML development as a secondary disease in MM patients having received radiation therapy in the past also cannot be excluded.

Regarding MM patients, the chronic immunological deficiency of the disease, could lead to the formation of secondary malignancies including CML [53].

Lastly, it is notable that despite allogeneic stem cell transplantation is the only potentially curative treatment both for CML and MM separately, no report could be found in the literature in the setting of these two entities coexisting. This is probably due to the fact that most coexisting CML and MM cases seen in the literature are patients older than 65 years old.

In conclusion, coexistence of MM and CML suggests either a different clonal evolution from a common pluripotent malignant stem cell, or exposure to previous chemotherapy and radiation, or existence of a pro-carcinogenic environment, or a coincidence, or every each one of the above possible factors. Further investigation of associated possible causes is needed to get to definitive conclusions.

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