relapsed multiple myeloma

Thrombotic microangiopathy

fiable causes, such as drugs, infections and

The two entities is rarely clear and, therefore,

Hb 86 g/L, WBC 5.1

with bone pains, hypercalcemia and renal fail-

In December 2008, the disease relapsed

was performed leading to complete response

increased platelet aggregation and fragmenta-

tion.

describe microvascular occlusive disorders

characterized by thrombocytopenia due to

knowledge is the second case of TTP/HUS

associated with bortezomib treatment.

Case Report

Introduction

A 52-year old woman was diagnosed with

disease in September 2007. The patient had

Bone marrow aspiration showed 50-100% plasma cells,

100), ionized calcium 2.07 mmol/L. Bone mar-

row was started. CR was obtained after three cycles.

CR was obtained after three cycles.

11, for a 21-day cycle) and dexamethasone (20

mg days 1-2, 4-5, 8-9, 11-12) at 3-week intervals

Details of blood counts, plasma creatinine con-

was started. CR was obtained after three cycles.

CR was obtained after three cycles.

Further, renal function and electrolytes were

normal during the treatment are shown in Table 1.

At that time, enterohaemorrhagic E. coli

Gingival biopsy stained positive for fibrin con-

uous fat staining for amyloid was negative.

confirmed the remission of myeloma, and subcuta-

neous fat staining for amyloid was negative.

Peripheral blood smear demonstrated a signifi-

cant increase of schistocytes (25% of 500 red

cells) and thrombocytopenia. Findings were

consistent with thrombotic microangiopathic

cells) and thrombocytopenia. Findings were

consistent with thrombotic microangiopathic

Thrombotic thrombocytopenic purpura (TTP)

and hemolytic-uremic syndrome (HUS)

showed: hemoglobin 78 g/L, erythrocytes

and severe orthostatic hypotension. She need-

for TTP/HUS like thrombotic microangiopathy

measure of ultra large VWF multimers that propa-

gated during the treatment and propagated during

expected but recovered before the next cycle. It

static hypotension and diarrhea were timely

Conflict of interests: the authors report no poten-

hemolytic-uremic syndrome, multiple myeloma.

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tor (VEGF) agents, i.e. bevalizumab and sunitinib. In mouse models, ablation of VEGF production in the kidney was sufficient to recapitulate the glomerular injury seen in patients. This may be the most probable mechanism by which bortezomib can cause thrombotic microangiopathy since bortezomib is known to decrease transcription of VEGF and other pro-angiogenic molecules by NF-κB inhibition.

Conclusions

In summary, we describe what to our knowledge is the second case of TTP/HUS associated with bortezomib treatment. Bortezomib is increasingly used in the treatment of multiple myeloma and clinicians should be aware of this association in patients with anemia and unexpectedly severe thrombocytopenia.

References


Table 1. Laboratory values during velcade treatment.

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<tr>
<th>Cycle</th>
<th>Day</th>
<th>Hb (g/dL)</th>
<th>Plt (×10^9/L)</th>
<th>Creat (mg/dL)</th>
<th>LDH (U/L)</th>
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