# Spontaneous acute subdural hematoma in a patient with multiple myeloma

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# **ABSTRACT**

Acute spontaneous subdural hematoma in a patient of multiple myeloma receiving chemotherapy is an unknown event, needing an urgent neurosurgical management. We report this patient who presented with progressive neurological deterioration and a low platelet count. She was successfully managed by craniotomy and evacuation of subdural hematoma with intraoperative transfusion of platelets. The acute spontaneous subdural hematoma in her was probably related to the bleeding diathesis due to thrombocytopenia associated with chemotherapy.

Key words: Acute spontaneous subdural hematoma, multiple myeloma, thrombocytopenia

### INTRODUCTION

Hemorrhagic strokes in cancer patients are neurological emergencies. Intraparenchymal hemorrhages are the most common type, followed by subdural hematomas (SDHs), subarachnoid hemorrhages, and epidural hematomas, in the listed order. The most common causes for symptomatic hemorrhages are coagulopathy and tumor-associated hemorrhages. Coagulopathy arises from multiple mechanisms in cancer patients and can be divided into dysfunction of platelets, coagulation factors, or both. Cancer-related platelet dysfunction or deficiency may occur from tumor infiltration of bone marrow, intrinsic bone marrow failure caused by hematologic malignancies (i.e. leukemia, lymphoma, and multiple myeloma (MM)), or chemotherapy or radiation therapy toxicity. [1] In 1934, Munro [2] was the first to describe a patient with SDH without history of head trauma. The reported incidences of spontaneous acute SDHs relative to total acute SDH have ranged from 2 to 6.7%.[3,4] The etiological spectrum includes: arteriovenous malformation (AVM), [5] cocaine

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abuse, [6] dural metastasis, [7] coagulopathy, [8,9] falx meningioma, [10] moyamoya disease, [8] and aneurysm rupture. [11] We report a patient of MM having low platelet count who presented with spontaneous acute SDH. The case is important as the condition reported is very rare and, to our knowledge, is nowhere mentioned in literature.

## **CASE REPORT**

The patient was a 34-year-old woman who had an acute episode of excruciating headache with vomiting and was later found obtunded. She had history of receiving chemotherapy (bortezomib and dexamethasone) as she was recently diagnosed as a case of MM. She had no history of any bleeding disorder or recent head trauma. On examination, she was disoriented and drowsy. She had anisochoria. Computerized tomography of the head [Figure 1] showed a right hemispheric SDH causing significant midline shift and uncal herniation. Blood investigations revealed isolated platelet count of 21,000/mm<sup>3</sup> with normal prothrombin time and activated partial thromboplastin time. The patient was started with platelets transfusion and simultaneously taken to the operating room for emergency decompression via frontotemporoparietal craniotomy. A thick, clotted SDH was removed. No active source of bleeding could be identified. Macroscopic inspection revealed no obvious vascular or neoplastic lesion. Patient had no neurological deficits after surgery and was discharged after 6 days.

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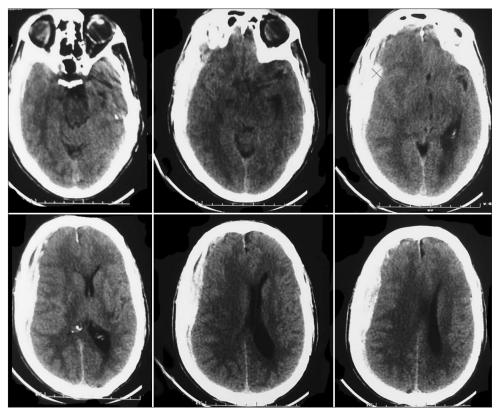


Figure 1: CT scan showing right fronto-temporo-parietal acute subdural hematoma

### **DISCUSSION**

MM is a neoplastic disease of plasma cells, characterized by infiltration of bone marrow, bone destruction, infiltration of soft tissues with plasma cells, and suppression of normal hematopoiesis The usual presenting features of MM include bone pain, weakness, fatigue, fever, and infection.

Bleeding diathesis is commonly seen in this disease and it has been estimated that as many as onethird of the patients with plasma cell myeloma may have this complication.[12,13] The common abnormalities contributing to bleeding diathesis include thrombocytopenia, platelet dysfunction related to uremia, hyperviscosity with bleeding tendency, disorders of coagulation factors, etc. The possible mechanisms by which coagulopathy could develop in patients with dysproteinemias in MM include: paraprotein interference with the normal function of coagulation factor(s) (i.e. by complexing with specific clotting factors), enhancement of the clearance of coagulation factor(s) by the reticuloendothelial system, anticoagulant activity of paraproteins, impaired normal platelet function, excessive fibrinolysis, and hyperviscosity. [2-5,12] Patients on bortezomib chemotherapy also often develop low platelet counts,

but these usually recover rapidly.[14] During melphalan, prednisolone, and thalidomide chemotherapy, periodic blood tests are needed to ensure that an individual has adequate levels of white blood cells and platelets. The dose of melphalan must be adjusted based on these findings. Paul et al. reported one death due to SDH in the dexamethasone group in a study comparing bortezomib and high-dose dexamethasone for relapsed MM.[15] Thrombocytopenia does not usually result in any symptoms, but if the platelet count is very low there is an increased risk of bleeding. Transfusion of platelets may be required temporarily. Keith et al., in their review of 23 cases of MM, described one patient with unilateral large subdural collection looking like acute SDH on imageology, but subsequently it turned out to be a malignant effusion.[16]

Spontaneous acute SDH is a rare but serious condition. This term is based simply on the absence of a traumatic history. [8,17,18] Mortality rate has been reported to be between 60 and 76.5%. [19] Early surgical intervention is often imperative for hematomas with significant mass effect. The survival rate for surgery within 4 h of acute onset compared to surgery after 4 h is 50 to 0%, respectively. Other favorable variables include a high glasgow coma score score upon admission, appropriate pupillary reactivity, and young age. [20]

### CONCLUSION

Although coagulopathy-associated spontaneous acute SDH has been well documented, spontaneous acute SDH related to coagulopathy, particularly in MM, has never been recognized.

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