

# Commentary

Trauma is the most common cause of acute subdural hematoma. Acute nontraumatic subdural hematoma (ANTSDH) is a relatively rare, and the most common causes are vascular lesions including ruptures, malformations, and fistulas. Other causes of ANTSDH are severe hypertension, coagulation problems related with underlying diseases or anticoagulation, amyloid deposition disease, cocaine abuse, and moyamoya disease.<sup>[1]</sup>

Ichimura *et al*<sup>[2]</sup> reported in this journal an interesting case with myelodysplastic syndrome/myeloproliferative neoplasia (MDS/MPN) and ANTSDH. This older case had severe leukocytosis and thrombocytopenia related with MDS/MPN and he was treated with erythrocyte, platelet, and fresh frozen plasma transfusions. However, bleeding recurred in epidural space in spite of intensive transfusions, and authors attributed this condition to the MDS/MPN-related thrombocytopenia.

MDS/MPNs are relatively rare myeloid neoplasias exhibiting both dysplastic and proliferative properties. Diagnosis is based on the demonstration of cytopenias and cytos with dysplasia and less than 20% blastic cells in peripheral blood and/or bone marrow.<sup>[3]</sup> Patient had one severe cytopenia and one severe cytosis; his platelet and leukocyte counts were 5,700/ $\mu$ L and 1,176,000/ $\mu$ L, respectively. It is very well known that hyperleukocytosis causes leukostasis, especially when leukocyte count is more than 100,000/ $\mu$ L. Leukostasis is an oncologic emergency causing respiratory failure and intracranial hemorrhage and also early mortality. Leukocytosis is more prevalent in older cases as seen in the reported case (>65 years).<sup>[4,5]</sup> Generally it is thought that leukostasis is related with very high number of leukemic cells in microcirculation. However, direct endothelial cell damage due to adhesion molecules, cytokines, and receptors is one of the essential pathogenetic mechanisms in the pathogenesis of leukostasis.<sup>[5]</sup> For this reason, to decrease the leukocyte counts by apheresis and also to block the endothelial damage with steroids are very important in the management of cases with severe leukocytoses related with MPNs. Patient reported by Ichimura *et al* was treated by platelet, erythrocyte, and fresh frozen plasma replacements. Patient had severe thrombocytopenia and bleeding was related with this. In addition, platelet functions may be abnormal in MDS/MPN and bleeding may be due to the platelet dysfunction. However, very high leukocyte count probably contributed to the subdural and epidural hematoma in the presented case. In conclusion, neurologists and neurosurgeons must be aware that cranial

bleedings including ANTSDH may be related with very high leukocyte counts and this may be managed by using corticosteroids and leukocyte apheresis.

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