Rapid Resolution of Acute Subdural Hematoma in a Coagulopathic Patient

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Acute subdural hematoma (aSDH) is associated with a 60-80% mortality rate and is considered a neurosurgical emergency. Although it is most often treated with emergent surgical decompression, patients may be managed conservatively when they are neurologically intact or the hematoma is small. Typical progression of aSDH resolution occurs over weeks, and is characterized by corresponding changes on radiographic imaging where bright aSDH becomes first isointense at about two weeks and then hypointense on noncontrast computed tomogram (CT) head imaging. If there is continued bleeding acutely, however, the SDH may increase in size leading to transtentorial herniation and subsequent clinical deterioration of the patient.

Interestingly, there are a number of case reports in the literature describing the event of spontaneous aSDH resolution. Several hypotheses have been put forward to explain this phenomenon including redistribution of subdural blood and dilution by cerebral spinal fluid (CSF). Cerebral atrophy, as well as cerebral swelling were both identified to facilitate aSDH resolution. Here we describe a patient with an unexpected resolution of an acute SDH in the setting of a bleeding diathesis, and propose that in this patient, coagulopathy contributed to the spontaneous resolution of the hematoma.

CASE PRESENTATION

Our patient is a 73-year-old gentleman who had acute onset of left-sided weakness. His strength was 4-/5 in the left upper and lower extremities. He was alert and oriented, with stable vital signs. His past medical history was significant for myelodysplastic syndrome with chronic pancytopenia, and atrial fibrillation on warfarin therapy.

On admission, significant findings on bloodwork included platelet count of 17 x 10^9/L and an international normalized ratio (INR) of 1.6. Non-contrast CT head scan performed at the initial evaluation demonstrated a moderate-sized acute subdural hemorrhage over the right convexity (Figure 1A). He received vitamin K and platelets, and was admitted for observation. Five hours later, his level of consciousness decreased, and he required intubation. A follow-up non-contrast CT head demonstrated dramatic enlargement of the acute SDH with uncal herniation (Figure 1B). He received a platelet transfusion and the prothrombin complex concentrate on formulary, Octaplex, in anticipation of emergent surgical evacuation. However, the patient's family instead opted for comfort measures.

Over the next two days, he started to open his eyes and localize to central pain. A follow-up CT performed on Day 2 revealed an unexpected significant resolution of the acute SDH.
The unique aspect of the case presented above is that the patient had a known double coagulopathy: thrombocytopenia in the setting of myelodysplastic syndrome, and warfarin-induced coagulopathy. To achieve hemostasis, two important components must be intact. Primary hemostasis, or formation of a “platelet plug”, requires sufficient quantity of functional platelets working in an intact blood vessel. Secondary hemostasis, or production of a “fibrin clot”, requires adequate and functional clotting factors. In this patient, both primary hemostasis, including thrombocytopenia and likely dysfunctional platelets from his myelodysplasia, as well as secondary hemostasis, indicated by the abnormal INR, were involved. While both of these conditions were potential culprits for developing an atraumatic aSDH in our patient, they may have also contributed to the rapid resolution of the hematoma by preventing the formation of a well-organized blood clot and thus be more susceptible to the redistribution via the CSF dilution route. This is the first case report to our knowledge to describe this mechanism of rapid spontaneous aSDH resolution.

SUMMARY

Spontaneous resolution of aSDH is a rare event that occurs via CSF dilution of the hematoma and subsequent redistribution of blood. Cerebral atrophy and cerebral edema facilitate rapid aSDH resolution. The presented case identifies coagulopathy as a new factor that promotes rapid aSDH resolution.

REFERENCES