Sub dural hematoma due to long term carbamazepine therapy

Sir,

Epilepsy is a common chronic neurological disorder characterized by recurrent unprovoked seizures. Epilepsy is more common in young children and people over the age of 65 years; however any age group can be affected.[1] We are reporting a case of 40-year-old female patient with loss of consciousness following fall from bed. She was a known case of epilepsy for last 14 years, for which she was taking Carbamazepine. The patient arrived in an emergency room with Glasgow coma score 3 (GCS 3) (E1V1M1). Pulse rate 70/min, blood pressure 114/70 mmHg, respiratory rate 8/min and euthermia. Her right pupil was dilated and sluggish reacting to light and left pupil was dilated and nonreacting. The patient's trachea was intubated, and mechanical ventilation was initiated. Her routine blood investigations were sent, and noncontrast computed tomography (CT) scan of the head was done. CT the scan has revealed large right sided temporo-parietal subdural hematoma (SDH) [Figure 1]. Routine blood investigation revealed hemoglobin of 8.5 g/dL, total leucocyte count 3400/mm^3, differential leukocytes count $P_{20}L_{78}E_2M_0$ and platelet count 30,000/mm^3 only. Peripheral blood smear showed large erythrocytes and paucity of platelets and granulocytes and few reticulocytes. International normalized ratio of the patient was 1.56. Patient was taken up for emergency burr hole SDH evacuation after administration of platelets and fresh frozen plasma. Further bone marrow aspiration and biopsy were done which showed a hypocellular bone marrow, with normal myeloid, erythroid ratio and megaloblastic red cell precursors. The patient's GCS gradually improved and was further shifted to ward. Carbamazepine is an established medication for seizures and is well tolerated by most of the patients, though the side-effects associated with carbamazepine therapy is not uncommon, but they are rarely serious even with long-term therapy. Aplastic anemia is a very rare side effect.[2] Carbamazepine therapy is associated with a nine fold increased risk of aplastic anemia.[3] Acute bone marrow suppression with leukopenia and thrombocytopenia is associated with carbamazepine therapy and it most often occurs within 3-4 months of initiation of treatment. We have ruled out most of the known causes of acute bone marrow suppression in our patient and drug induced myelo-suppression came to us as the most likely cause of pancytopenia, leading to SDH. The mechanism of bone marrow suppression due to carbamazepine therapy has not been clearly established. Aplastic anemia is an idiosyncratic, nondose-related side-effect of carbamazepine.[2] Aplastic anemia occurs rarely but is potentially fatal, and therefore diligent monitoring of hematologic function is indicated. Once seizures are controlled, plasma levels of carbamazepine should be measured to establish optimum levels for individual patients being treated with this drug.[2] US Food and Drug Administration recommends complete hematological evaluation as a baseline in patients who are planned for carbamazepine therapy.[4] If a patient in the course of carbamazepine therapy exhibits low or decreased white blood cell or platelet counts, the patient should be monitored closely, or drugs should be switched over to other class of antiepileptic drugs. Discontinuation of the drug should be considered if any evidence of significant bone marrow suppression develops.[5] The patients who are on carbamazepine therapy should have baseline hematological investigations and must be repeated on regular follow-ups.

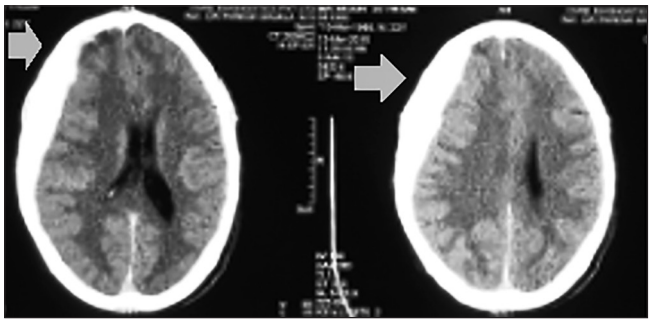

Figure 1: Computed tomography scan of patient with arrow showing location of sub dural hematoma

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Conflicts of interest

There are no conflicts of interest.

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