CASE REPORT

Chronic subdural hematoma in a patient with acute myeloid leukemia and dural metastatic infiltration

ALEXANDRA COMĂNESCU1, ELENA ROSCA1, MARCELA BOTA2, GH. NINULESCU1

1) Faculty of Medicine, University of Oradea
2) Emergency County Hospital of Oradea

Abstract
Dural metastasis (pachimeningeal carcinomatosis) refers to involvement of dura mater and subdural or epidural spaces in systemic cancer. Although dural metastasis are relatively frequent in malignancies, (8–9% of patients with advanced systemic cancer in autopsy), they are rarely associated with subdural hematoma. We present the case of a 66-year-old male, referred to the hematological unit for severe anemia of unknown cause and diagnosed as acute myeloid leukemia, developed in the 10th day from admission progressive gait disorders with marked imbalance and mental status change. A CT head scan showed a large, chronic subdural hematoma, which was surgically removed. Pathological examination of the dura and citological examination of the subdural fluid revealed metastatic involvement of the dura, demonstrating the association of dural metastasis and chronic subdural hematoma.

Keywords: dural metastasis, subdural hematoma.

Introduction
Subdural hematoma has a reported incidence of 0.5–4% of all intracranial metastatic tumors [1].

Studies on large series of cancer patients concluded there were two main groups of subdural hematoma in these patients: (1) subdural hematoma related to predisposing factors (such as previous head trauma, alcoholism, or anticoagulation) and (2) spontaneous nontraumatic subdural hematoma. The two groups were found both in solid tumors and in hematological malignancies, but the first group was more frequently associated with solid tumors, while spontaneous hematoma was mainly associated with hematological malignancies [2].

Malignant subdural hematoma was more frequently found in acute versus chronic leukemias or lymphoma [3, 4]. Malignant subdural hematoma may occur secondary to multiple factors, including:

• dural blood vessel occlusion by tumoral cells, that may rupture within the subdural compartment [5];
• tumor necrosis [6];
• chemotherapy induced thrombocytopenia or disseminated intravascular coagulation secondary to underlying malignancy [7].

Patient and methods
We present the case of a 66-year-old male, who was admitted for increasing fatigue, which he developed in the last 45 days, and recurrent upper respiratory tract infection signs.

Routine laboratory tests in outpatient clinic revealed a severe anemia, leading to admission in the hematological clinic.

The peripheral blood count was: platelets 16000/µL, reticulocytes 0.1%, hemoglobin 7.9 g/L, red blood cells 2700000/µL, white blood cells 5000/cmc, 28% lymphocytes and 72% myeloblasts, ESR = 132/146.

The bone marrow biopsy shows a cellular population consisting entirely in myeloblastocytes, while red blood cell and platelet series were absent. A diagnosis of acute myeloid leukemia was made (FAB M1), and induction therapy with Citarabine 200 mg/day × 5 days was made and complete remission occurred, with peripheral blood count of: 1000 platelets/µL, 800 white blood cells/µL, 5% neutrophils, 87% lymphocytes (Figure 1).

Blood and platelet transfusion, antimicrobial and antifungal antibiotics, and hepatotrophics were also administered. The clinical evolution was initially favorable, but in the 10th day from admission, the patient developed a rapidly progressive gait disorders with marked imbalance and a mild confusion state.

On neurological examination, the patient was alert, but became intermittently confused and had difficulty following complex commands. No focal sensorimotor signs were noted. He had a remarkable unsteadiness with lateral deviation and titubation during vertical posture and could walk only with assistance.

He did not have headache. He had no meningeal signs, nystagmus was absent, plantar cutaneous response was flexion.
Emergent CT of the head revealed a large heterogenous subdural fronto-temporo-parietal collection, with mass effect. The collection was predominantly hypodense mixed with some hiperdense foci of fresh blood. Diagnosis of a chronic subdural hematoma with foci of rebleeding was made (Figure 2).

By left parietal craniotomy were evacuated 20 ml of fluid content having the aspect of crankcase oil and the membranes of the hematoma were also removed. The clinical course was unfavorable, with persistent coma, and CT head scan showed subdural rebleeding in the same area (Figure 3).

A second surgical procedure was done, but the patient remained unconscious and died by cardiac arrest upon supported ventilation.

**Results**

The histopathological examination of the dura and of the outer membrane of the hematoma confirmed neoplastic dural invasion (Figure 4, a–c).

The pathological aspect of the neoplastic cellular spread is the same with the neoplastic cells in the peripheral blood smear.

The same morphology was found in the malignant infiltrate in the portal spaces in the liver (Figure 5, a and b).

Capsula of the newly formed hematoma by rebleeding, had the same histopathological aspect (Figure 6, a and b).

**Discussions**

The clinical presentation of the CSH with imbalance, without headache and without sensorimotor focal signs, was atypical for CSH.

The mental status change was, nonetheless, an important sign, which led to emergent CT-scan and diagnosis.

As most hemorrhagic complications in acute leukemia consist in subarachnoid or lobar hemorrhages, the CT diagnosis of CSH in this case, in the absence of previous head injury, was initially surprising.

Regarding the non-traumatic occurrence of the hematoma, thrombocytopenia is an important, but not the single etiologic factor, and the presumed association with malignant invasion of the dura was confirmed histopathologically.
Conclusions

This case report suggests that dural metastasis should be considered in patients with hematological malignancies and chronic subdural hematoma.

Whenever possible, surgical removal is the most appropriate treatment.

Prognosis for patients with CSH and acute leukemia may be poor, because of coagulation abnormalities and rebleeding.
References


Corresponding author
Alexandra Comănescu, Assistant, MD, Faculty of Medicine, University of Oradea, 1 University Street, 410 087 Oradea, Romania; Phone +40259–440 700, Fax +40259–437 814, E-mail: alexandra_comanescu@yahoo.com

Received: June 25th, 2007

Accepted: April 20th, 2008