Case Report

Dementia Syndrome Revealing Acute Lymphoblastic Leukemia in Department of Neurology at Fann National Teaching Hospital in Dakar (Senegal)

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Abstract: Dementia is a serious disease due to its morbidity and mortality. The etiology is dominated by Alzheimer's disease. However, there are dementia syndromes related to treatable and potentially curable cause. It's rarely reported in clinical practice in our context. We report a case of dementia associated with lymphoblastic acute leukemia complicated by lymphocytic meningitis. F. ND aged 66, widow, unschooled and unemployed was hospitalized for gait disturbance and progressive appearance of memory disorders. On physical examination, there was a dementia syndrome, bilateral pyramidal syndrome. The rest of the clinical examination associated with additional tests (thoraco-abdominal-pelvic CT scan, lumbar puncture, and myelogram) was in favor of lymphocytic meningitis and acute lymphocytic leukemia. Dementia is a disease in which the search for the cause is crucial for considering an etiological support. Blood diseases such as LAL are treatable causes. The clinic provides a guidance element of research. Also, multidisciplinary care is necessary in these kinds of patients for a better development. This scientific cooperation must be done with, hematologists, internal medicine specialists and other colleagues.

Keywords: Dementia, Acute Lymphoblastic Leukemia, Lymphocytic Meningitis, Senegal

1. Introduction

Dementia is a serious disease due to its morbidity and mortality [1]. The etiology is dominated by Alzheimer's disease; she is responsible for half of dementia [2]. However, about 1.5% of dementia syndromes are related to curable causes [3] or potentially curable [2] with uncommon etiologies raising the risk of late diagnosis, hence the importance of knowing and seeking for them systematically [2]. They are a heterogeneous group of non-degenerative pathologies, potentially reversible hence the usefulness of early diagnosis which can help improve prognosis by proper management [3]. It’s rarely reported in clinical practice in our context. We report a case of dementia associated with lymphoblastic acute leukemia complicated by lymphocytic meningitis.

2. Observation

F. ND 66-year-old, widow, unschooled, unemployed was hospitalized on the 19/02/2015 for gait disturbance and progressive appearance of memory disorders. The beginning goes back to the month of December 2012 marked by the onset of gait disturbance of hesitant walking
slowly type, associated with memory disorders made of recent events omissions and urinary and fecal disorders. This symptomatology evolves in a context of unquantified vespers-night fever, alteration of general condition with non-selective anorexia and unquantified weight loss. Faced with the worsening of the picture with the walking increasingly difficult, the patient is taken to the Fann University Hospital for better Support. She was known hypertensive for 1 year, wrongly followed, had no concept of diabetes and there were no similar cases in the family. On physical examination, there was:

Blood pressure: 110/90 mmHg, temperature: 37.8°C, pale conjunctival mucous, no edema of lower limbs, folds of dehydration presence and discrete malnutrition. Neurologically the patient was awake but did not execute any simple order. The osteotendinous reflexes was sharp to four members; the Babinski sign was present at both lower limbs. Also, we noted oppositional hypertonia, a grasping reflex, a nasoorbicular reflex present and inexhaustible, a palm-chin reflex, no motor deficit target. She was silent. Neuropsychological tests showed an MMSE score of 16 OUT OF 30. Elsewhere, diffuse scratching lesions were noted on the whole body, multiple adenopathies: cervical, sub-clavicular, axillary, inguinal, bilateral, movable relative to the superficial and deep planes, pressure sensitive, measuring about 0.5 to 1 cm in diameter, skin look normal. The spleen was not palpated and not impacted. Other clinical data were unremarkable.

The brain CT SCAN performed showed a cortico-subcortical atrophy. The check-up on the 27.02.2014 had found: the blood count of white blood cells at 6.14.10^3/mm^3, red blood cells at 3.58.10^3/mm, platelets at 281.10^3/mm^2 and hemoglobin 9.6 g/dl with VGM 82.4 fl and MCH 26.8 pg. Reticulocytes were not assayed. CRP was positive at 48 mg/l. Sedimentation rate at 20 in the first hour and 40 in the second hour. The rest of the blood tests were normal with (Fasting glucose, serum creatinine, urea blood, lipid profile, serum protein, serum calcium level, serum magnesium).

Analysis of the cerebrospinal fluid (CSF) on the 25.02.2012 had found 125 items including 70 lymphocytes and 55 unaltered polymenular. The cerebrospinal fluid proteins were 0.80 g/l. Other elements of CSF study were normal with (parasito-mycological examination, bacteriological examination). Note that the syphilitic, borrellien and brucellen serology in blood and CSF was negative. The search for mycobacteria of complex Tuberculosis in blood by PCR (Polymerase Chain Reaction) in blood, in CSF and in urine was negative.

The myelogram showed an aspect compatible to a medullary invasion of a non-Hodgkin lymphoma with infiltration of lymphoid cells of medium size to the coarse chromatin, sometimes nucleolated with a cytoplasm sparsely extended and basophil. Ganglionic biopsy showed an aspect suggesting non-Hodgkin malignant lymphoma of Burkitt-like type.

The check-up highlighted axillary adenopathy bilaterally retroperitoneal and mesenteric, and two biliary cysts of segments II and V of the liver on the thoraco-abdominal-pelvic scan. Leukemia was assessed in Stage IV of Ann Harbor's classification and Measurement of LDH would give a better idea of the prognosis.

Overall, the diagnosis of dementia on lymphoblastic leukemia complicated by acute lymphocytic meningitis was made.

The patient was put under treatment, after discussions with hematologists, with the protocol including Cyclophosphamide, Methotrexate, and Adriblastine. After a first cure, the check-up on the 12.03.2012 had found: a blood count of white blood cells at 2.65.10^3/mm^3, red cells at 2.79.10^3/mm^3, platelets at 35.10^3/mm^3 and hemoglobin at 3.2 g/dl with VGM 82.3 fl and MCH 26.5 pg. CRP was positive at 12 mg/l. The sedimentation rate was 16 mm the first hour and 32 mm the second hour. CSF analysis found 45 items including 22 lymphocytes and 8 unaltered polymenular. The cerebrospinal fluid proteins were 0.90 g/l.

The evolution has been marked by an increase in the volume of adenopathies. Unfortunately, death occurred after the first course of chemotherapy.

3. Discussion

The number of patients treated for hematological malignancies or solid tumors is expanding [4], their survival is increasing thanks to new drugs and treatment strategies [5]. If the respiratory, cardiovascular and renal failures have been the subject of epidemiological, diagnostic and prognostic research [6, 7], neurological failure has not been the subject of a descriptive or analytical work [8].

Our observation reports a case of subsequent dementia neurological complications (meningitis) of acute lymphoblastic leukemia. A dementia case related to hematologic disease was reported by Colin et al. [3], it was acute promyelocytic leukemia with neurological complications like recurrent meningitis. Acute, lymphoid and myeloid leukemia represent 10 to 15% of hematological malignancies and are often of poor prognosis [9]. Acute lymphoblastic leukemia is most common in children their prognosis is better in the latter with a 5-year survival to 90% of cases, while for adults it is only 35 to 40% [9]. The occurrence of neurological complications of acute lymphoblastic leukemia is a reality. Indeed according to Tattevin P et al. blood disorders are one of the three leading causes of aseptic meningitis [10]. Boumanhi reports a case of meningitis consecutive to acute lymphoblastic leukemia [11]. This was noted in our patient. The presence of a Senegalese study 43.5% of meningitis whose cause had not been found by Soumare et al. [12] raises the question of the importance of research of so-called rare causes. The occurrence of post-meningitis dementia shows a slow type, associated with memory disorders that would come from a totally curable cause, while
9.3% would be linked to a cause partially curable [4, 5]. The most common causes of dementia are a treatable neurosurgical cause (hydrocephalus, tumor, chronic subdural hematoma), metabolic and toxic disorders (including alcoholism) and, much more rarely, epilepsy and infectious causes [13]. Acute lymphoblastic leukemia may be in the potentially curable causes. Curable dementia syndromes are rare and their frequency is estimated to be 1.5% [14]. These are a heterogeneous group of diseases degenerative, potentially reversible after etiological treatment, where the interest to identify them early. The clinician is often steered by the presence of other anomaly no neurological. When absent, diagnosis then mainly based on comprehensiveness and rigor of the diagnostic process [15]. In the presence of a crazy syndrome, paraclinical examinations should be directed according to the interrogation, neuropsychological and neurological signs [15].

In the presence of dementia syndrome and or aseptic meningeal syndrome, the orientation of the etiologic investigation is based on an accurate analysis of the clinic such as medical history, the patient's age, the establishment mode and associated signs [2, 3]. This was performed on our patient with the clinical hematological malignancy events. It's important to following this approach because if the etiology was detected it will possible to made the remedial treatment of this dementia type.

4. Conclusion

Dementia is a condition in which the search for the cause is crucial for considering an etiological care if possible. The blood diseases such as acute lymphoblastic leukemia comprised of treatable causes. The clinic constitutes an element directing the research. Also, multidisciplinary care is necessary in these kinds of patients for a better development. This scientific cooperation must be done with, hematologists, internal medicine specialists and other colleagues.

Conflict of Interest

Authors declared no conflicts of interest.

References


