An unusual case of basophilic meningitis revealing a chronic-phase chronic myeloid leukemia: a report after almost three years of follow-up

Cases of basophilic meningitis are rarely found in the literature. One case was reported in a patient with a blastic transformation of chronic myeloid leukemia (CML) after two years of therapy.[1] Other cases were described in viral meningitis.[2] Here we report the case of a 35-year-old woman with a basophilic meningitis revealing a chronic-phase CML. This patient was successfully treated with intensive chemotherapy combined with tyrosine kinase inhibitors (TKI) that are known to cross the blood-brain barrier (i.e., dasatinib and ponatinib[3-5]), and is still in complete remission after a follow-up of almost three years. This case report complies with the ethical and regulatory standards in force in France, including obtaining informed consent and protecting patient confidentiality.

In January 2023, this young patient with a history of type 1 diabetes and psychogenic non-epileptic seizure presented to the Emergency Department with a generalized tonic-clonic seizure confirmed on electroencephalogram. She also complained of bilateral blurred vision. The brain magnetic resonance imaging (MRI) showed subtle cortical FLAIR hyperintense punctiform images at the level of the right temporal and insular cortex (Figure 1A). An atraumatic lumbar puncture was thus performed. Cerebrospinal fluid (CSF) examination showed pleocytosis with white blood cells (WBC) 210 cells/μL. Protein and glucose concentrations were within normal ranges. Gram stain and bacterial culture were both negative. No virus was detected using molecular techniques. On cytological examination, cerebrospinal fluid (CSF) smear showed a majority of basophils (Figure 2A). In addition, the complete blood count showed elevated WBC ($15x10^9$/L) with 68% neutrophils and 5% basophils. Hemoglobin level and platelet count were within normal ranges. Morphological examination of bone marrow aspiration showed normal cellularity with neither blast nor basophil excess. Cytogenetics revealed an isolated Philadelphia chromosome. In addition, *BCR::ABL1* fusion gene was confirmed (i.e., major transcript b3a2/e14a2) and quantified at 71% using RT-qPCR. Ultimately, BCR::ABL1 was detected in CSF basophils, but not in neighboring lymphocytes, confirming their clonal origin (Figure 2B). In all, those findings were consistent with the diagnosis of a chronic-phase CML according to the fifth edition of the World Health Classification[6] associated with basophilic meningitis.

Before treatment with intensive chemotherapy, the patient received two intrathecal injections of chemotherapy (methotrexate 15 mg, cytarabine 40 mg, methylprednisolone 40 mg). CSF was clear after the first therapeutic lumbar puncture. A first induction course with intravenous (IV) high-dose cytarabine (2,000 mg/m^2 b.i.d. on days 1, 3 and 5) was then started in combination with oral dasatinib at 140 mg/day from day 1. On day 24, the patient reported a significant bilateral worsening of her vision. Ophthalmic assessment was consistent with bilateral exudative retinal detachment and choroidal thickening with extensive retinal hemorrhage; these lesions were deemed to be leukemic infiltrates[7] (Figure 3A, B). Dasatinib was stopped on day 27 after the onset of an acneiform eruption, and the patient was switched to ponatinib 30 mg/day on a continuous schedule. After cycle

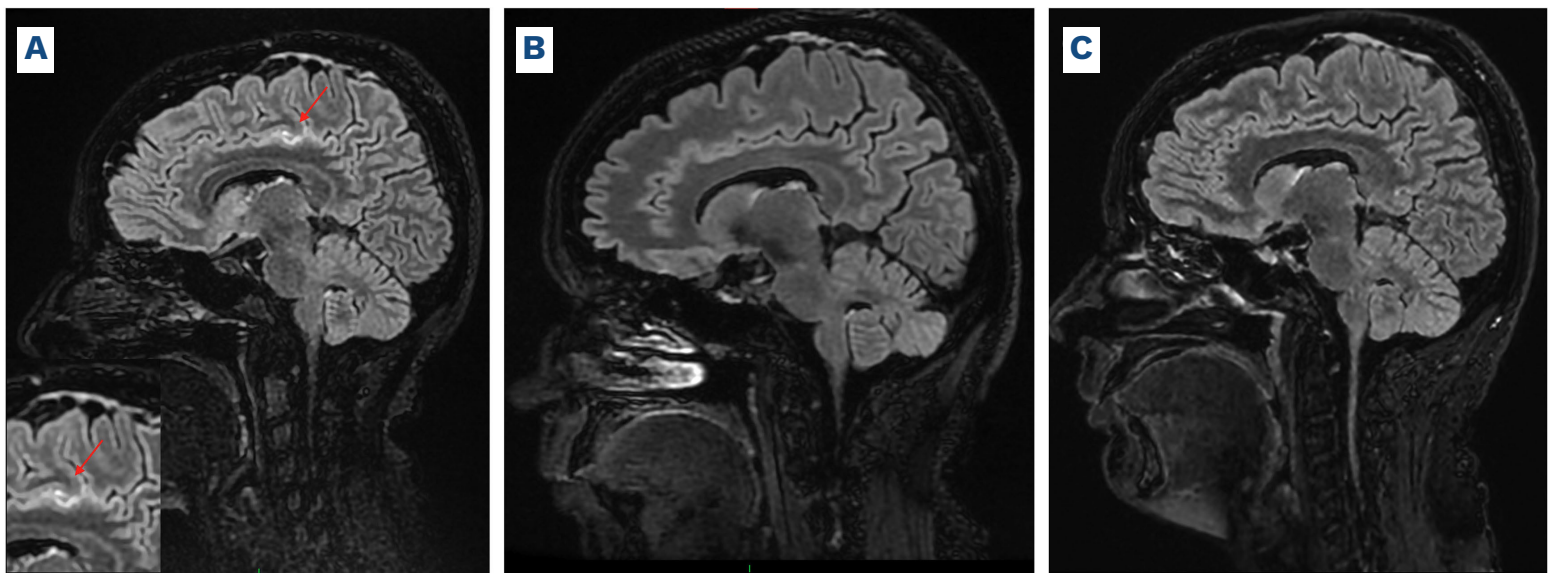


Figure 1. Brain magnetic resonance imaging performed on diagnosis, after induction treatment and at two years of follow-up. Sagittal FLAIR-weighted images on diagnosis (A), after two months (B), and after two years (C) of follow-up after induction therapy. Abnormal magnetic resonance imaging (MRI) findings are indicated with a red arrow in (A) (whole and enlarged image).

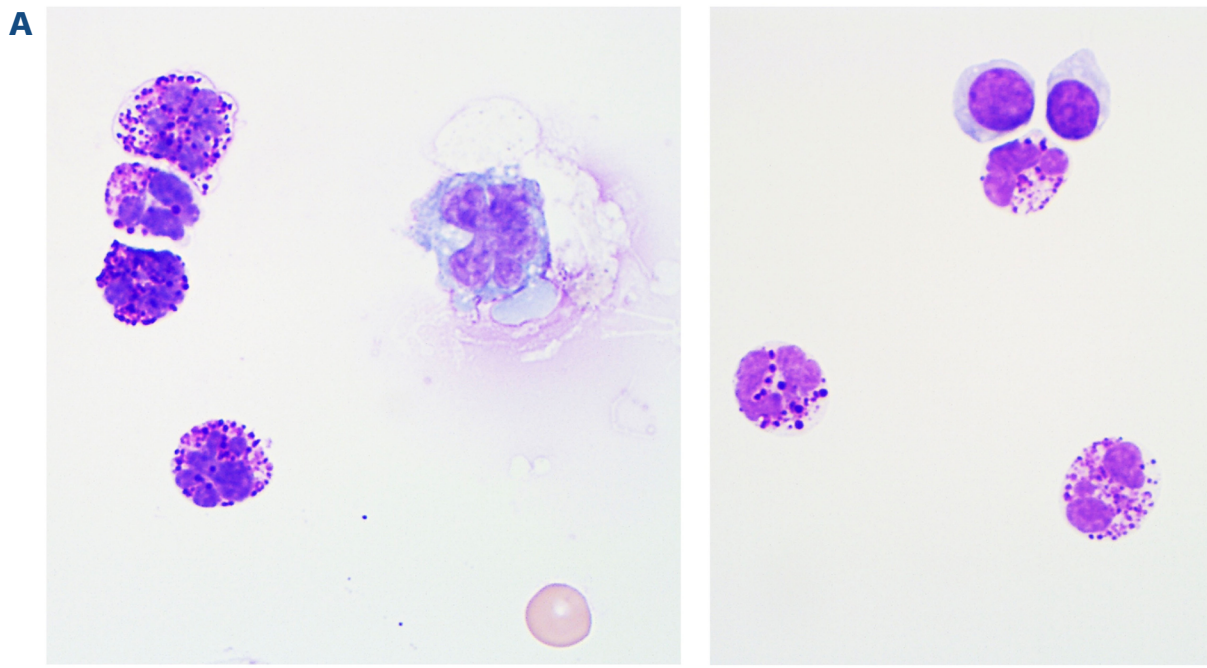


Figure 2. Cytologic evaluation and fluorescence *in situ* hybridization testing of cerebrospinal fluid. (A) May-Grünwald-Giemsa staining performed on cytological smears of cerebrospinal fluid. Results are shown with 100X magnification. (B) Interphase fluorescence *in situ* hybridization (FISH) testing for t(9;22) translocation is performed using the LSI-BCR-ABL dual color extra-signal probe (Vysis)[9]: LSI ASSI-ABL1 SpectrumOrange probe for the 9q34 region; LSI BCR SpectrumGreen probe for the 22q11.2 region. Normal pattern is found in lymphocytes (2R2V). BCR-ABL1 fusion pattern 1R1V1F 1ES (F) and extra signals (ES) are detected in basophils.

1, no visible lesion was seen on brain MRI (Figure 1B). Bone marrow aspiration was normocellular and BCR::ABL1 was quantified at 0.13% (MR2.9) (Figure 3C). Since the patient achieved remission in central nervous system on brain MRI and in CSF, no further intrathecal chemotherapy was administered. On day 60, the patient received a second induction course of high-dose cytarabine with ponatinib. On day 110, a first consolidation course of IV reduced-schedule cytarabine (2,000 mg/m^2 b.i.d. on days 1 and 2) plus ponatinib was started, followed by three additional consolidations given every 21 days. Figure 3C shows BCR::ABL1 levels during follow-up. At the end of the consolidation courses, ponatinib was continued at 30 mg/d for two years and was recently reduced to 15 mg/d after a molecular response superior to MR4.5 was achieved. In parallel, the patient's vision gradually recovered and reached previous visual acuity after one year of follow-up with resolution of the retinal infiltrates over time (Figure 3A, B). Brain MRI was also normal after two years of follow-up (Figure 1C).

To sum up, this report illustrates a very unusual inaugural presentation of chronic-phase CML that, to our knowledge, has never previously been reported. Although rarely described, cases of basophilic meningitis tend to occur during blastic transformation phase of CML (also called 'blast crisis'), the incidence of which has been extremely low since the introduction of TKI,[8] and are associated with dismal prognosis. Moreover, the obtained response was rapid and deep. After discussion with colleagues, together we decided not to proceed with allogeneic transplantation even though a compatible family donor had been identified. This decision was, by analogy, motivated by the high efficacy of ponatinib in cases of Philadelphia-positive acute lymphoblastic leukemia with CSF involvement.[5] The patient currently maintains deep and sustained molecular

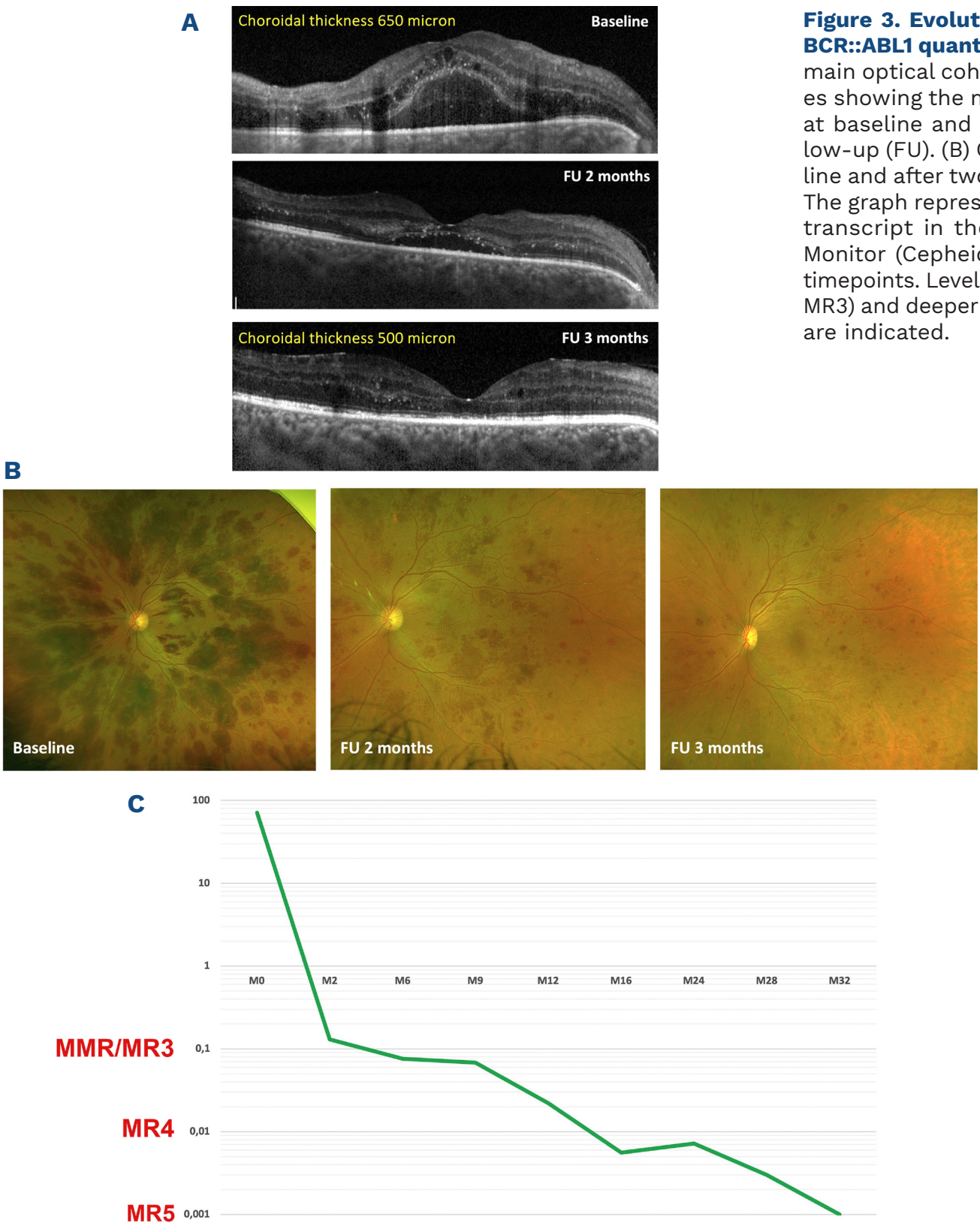


Figure 3. Evolution of ophthalmologic lesions and BCR::ABL1 quantification over time. (A) Spectral domain optical coherence tomography (SD-OCT) images showing the measurement of choroidal thickness at baseline and after two and three months of follow-up (FU). (B) Colour fundus photographs at baseline and after two and three months of follow-up. (C) The graph represents the quantification of BCR::ABL1 transcript in the blood using an Xpert® BCR-ABL Monitor (Cepheid, Sunnyvale, CA, USA) on multiple timepoints. Levels of major molecular response (MMR/MR3) and deeper molecular responses (MR4 and MR5) are indicated.

response almost three years after diagnosis. Considering that ponatinib is the only TKI option for this patient, whether ponatinib can be stopped – to allow parenthood and reduce the risk of cardiovascular events in this young diabetic patient – is a highly challenging question.

Authors

Ramy Rahmé,[1,2] Matteo Dragani,[1,2] Carole Fleury,[2,3] Giulia Tueur,[2,3] Paule Moussounda Bamba,[1,2] Héloïse Torres-Villaros,[2,4] Audrey Giocanti-Aurégan,[2,4] Roland Bangbotche,[5] Gregory Lazarian,[2,3] Fanny Baran-Marszak,[2,3] Claude Gardin[1,2] and Thorsten Braun[1,2]

[1]Service Hématologie Clinique et Thérapie Cellulaire, Hôpital Universitaire Avicenne, Assistance Publique, Hôpitaux de Paris; [2]UFR de Santé Médecine et Biologie Humaine (SMBH), Université Sorbonne Paris Nord; [3]Service Hématologie Biologique, Hôpital Universitaire Avicenne, Assistance Publique, Hôpitaux de Paris; [4]Service Ophtalmologie, Hôpital Universitaire Avicenne, Assistance Publique, Hôpitaux de Paris and [5]Service de Neurologie et Unité Neurovasculaire, Centre Hospitalier de Saint-Denis, Paris, France

Correspondence:
R. RAHMÉ - ramy.rahme@aphp.fr

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Disclosures

No conflicts of interest to disclose.

Contributions

RR and TB treated the patient, collected data, and wrote the manuscript. CF, GT, GL and FB-M performed the biological analyses. MD, PMB, HT-V, AG-A, RB and CG treated the patient.

Data-sharing statement

The data that support the findings of this study are available on request from the corresponding author.

References

1. Ding H, Zhang L. Basophils in the cerebrospinal fluid from a case of central nervous system leukemia. Int J Lab Hematol. 2022;44(1):17-18.
2. Izquierdo Alvarez C, Fores R. Basophilic meningitis. Diagn Cytopathol. 2023;51(3):196-197.
3. Ravi K, Franson A, Homan MJ, et al. Comparative pharmacokinetic analysis of the blood-brain barrier penetration of dasatinib and ponatinib in mice. Leuk Lymphoma. 2021;62(8):1990-1994.
4. Gong X, Li L, Wei H, et al. A higher dose of dasatinib may increase the possibility of crossing the blood-brain barrier in the treatment of patients with Philadelphia chromosome-positive acute lymphoblastic leukemia. Clin Ther. 2021;43(7):1265-1271.e1.
5. Zhu Y, Zhu Y, Miao L, et al. Comparison of the efficacy and safety of ponatinib and dasatinib in Philadelphia chromosome positive acute lymphoblastic leukemia with central nervous system relapse: a retrospective study. Technol Cancer Res Treat. 2023;22:15330338231165866.
6. Loghavi S, Kanagal-Shamanna R, Khoury JD, et al. Fifth edition of the World Health Classification of Tumors of the Hematopoietic and Lymphoid Tissue: Myeloid Neoplasms. Mod Pathol. 2024;37(2):100397.
7. Adaniya A, Luciano AD, Alvarado-Villacorta R, et al. Serous retinal detachment as a sign of leukemic choroidopathy: a systematic review. Surv Ophthalmol. 2022;67(1):149-167.
8. Perrotti D, Jamieson C, Goldman J, Skorski T. Chronic myeloid leukemia: mechanisms of blastic transformation. J Clin Invest. 2010;120(7):2254-2264.
9. Primo D, Tabernero MD, Rasillo A, et al. Patterns of BCR/ABL gene rearrangements by interphase fluorescence in situ hybridization (FISH) in BCR/ABL+ leukemias: incidence and underlying genetic abnormalities. Leukemia. 2003;17(6):1124-1129.