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

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Disclosures

All of the authors have 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.

Cases of basophilic meningitis can be exceptionally 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 the obtainment of informed consent and the protection of 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 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 210 white blood cells/µL. Protein and glucose concentrations were within normal ranges. Gram stain was negative as well as bacterial culture. No virus was detected using molecular techniques. On cytological examination, CSF smear showed a majority of basophils ***(figure 2A)***. Besides, the complete blood count showed elevated white blood cells at 15 $x10^9$/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. Additionally, 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 neighbouring 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 (2000 mg/m2 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 haemorrhage: these lesions were deemed to be leukemic infiltrates[7] ***(figure 3A and 3B)***. Dasatinib was stopped on day 27 after the onset of an acneiform eruption, and switched to ponatinib 30 mg/day on a continuous schedule. After cycle 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 (2000 mg/m2 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 and 3B)***. Brain MRI was also normal after two years of follow-up ***(figure 1C)***.

In sum, this report illustrates a very unusual inaugural presentation of chronic-phase CML that, to our knowledge, was never priorly reported. Although rarely described, cases of basophilic meningitis tend to occur during blastic transformation phase of CML (also called 'blast crisis'), whose incidence is extremely low since TKI were introduced[8], and are associated with dismal prognosis. Moreover, the obtained response was rapid and deep. We collegially decided not to proceed with allogeneic transplantation although a compatible familial donor was 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]. In retrospect, the patient is in deep and sustained molecular response almost three years after diagnosis. Considering that ponatinib is the only TKI option for this patient, whether ponatinib can be stopped in this case – to allow parenthood and reduce the risk of cardiovascular events in this diabetic young patient – is a highly challenging question.

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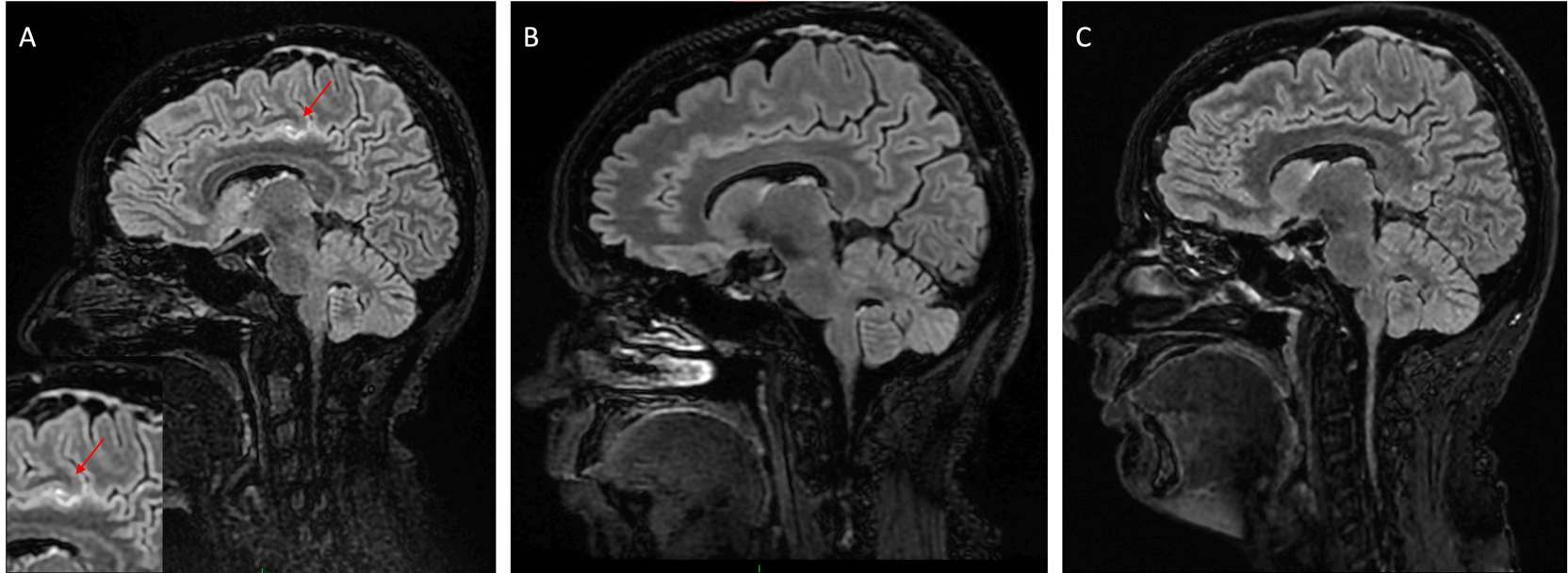
Figure legends:

Figure 1. **Brain MRI performed on diagnosis, after induction treatment and at 2 years of follow-up**. Sagittal FLAIR-weighted images on diagnosis (1A), after two months (1B) and two years (1C) of follow-up after induction therapy. Abnormal MRI findings are indicated with a red arrow in 1A (whole and enlarged image).

Figure 2. **Cytologic evaluation and fluorescence in situ hybridization (FISH) testing of cerebrospinal fluid**. (A) May-Grünwald-Giemsa staining is performed on cytological smears of cerebrospinal fluid. Results are shown with 100X magnification. (B) Interphase 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.

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 *Xpert® BCR-ABL Monitor* (developed and manufactured by *Cepheid, Sunnyvale, CA, USA*) on multiple timepoints. Levels of major molecular response (MMR/MR3) and deeper molecular responses (MR4 and MR5) are indicated.

Keywords: basophil meningitis; chronic myeloid leukemia; dasatinib; ponatinib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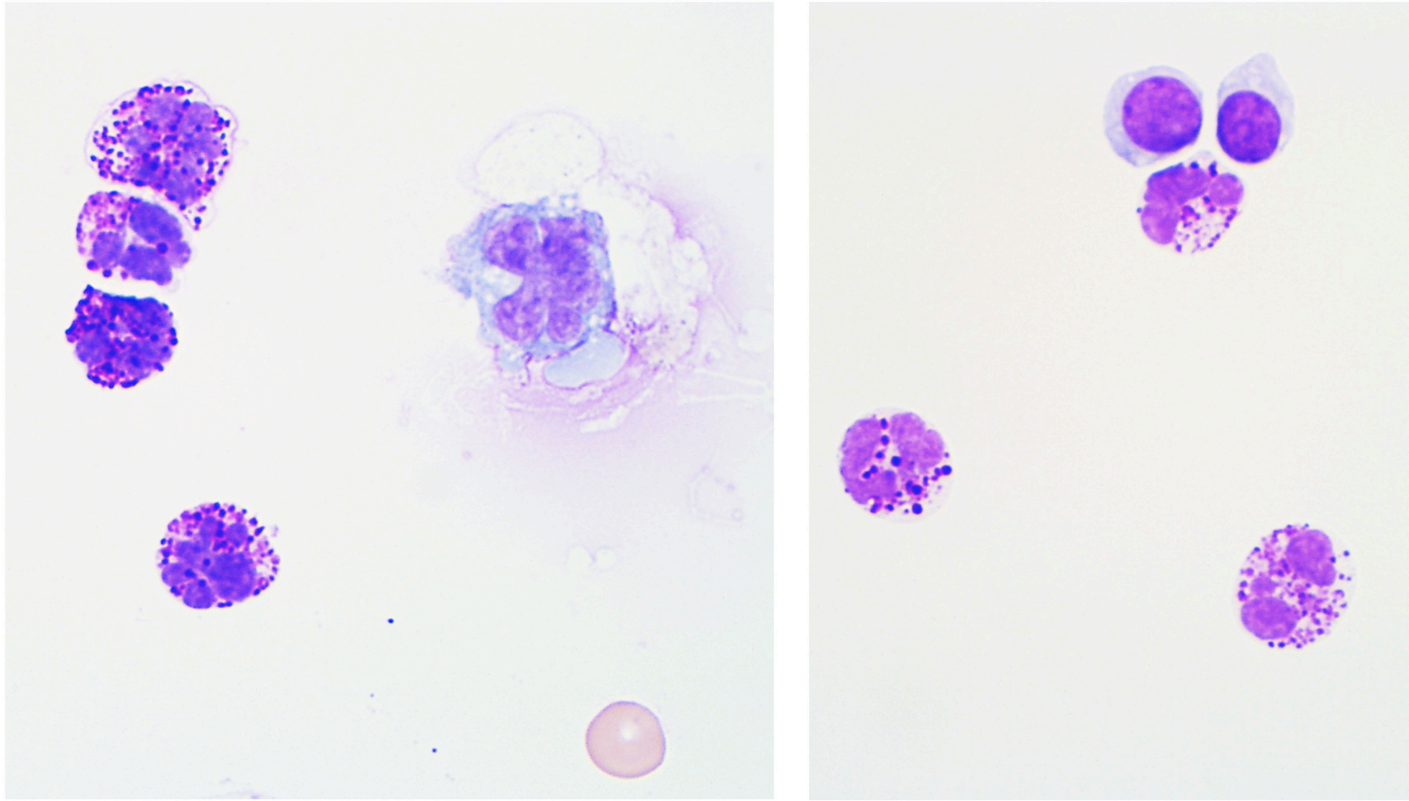

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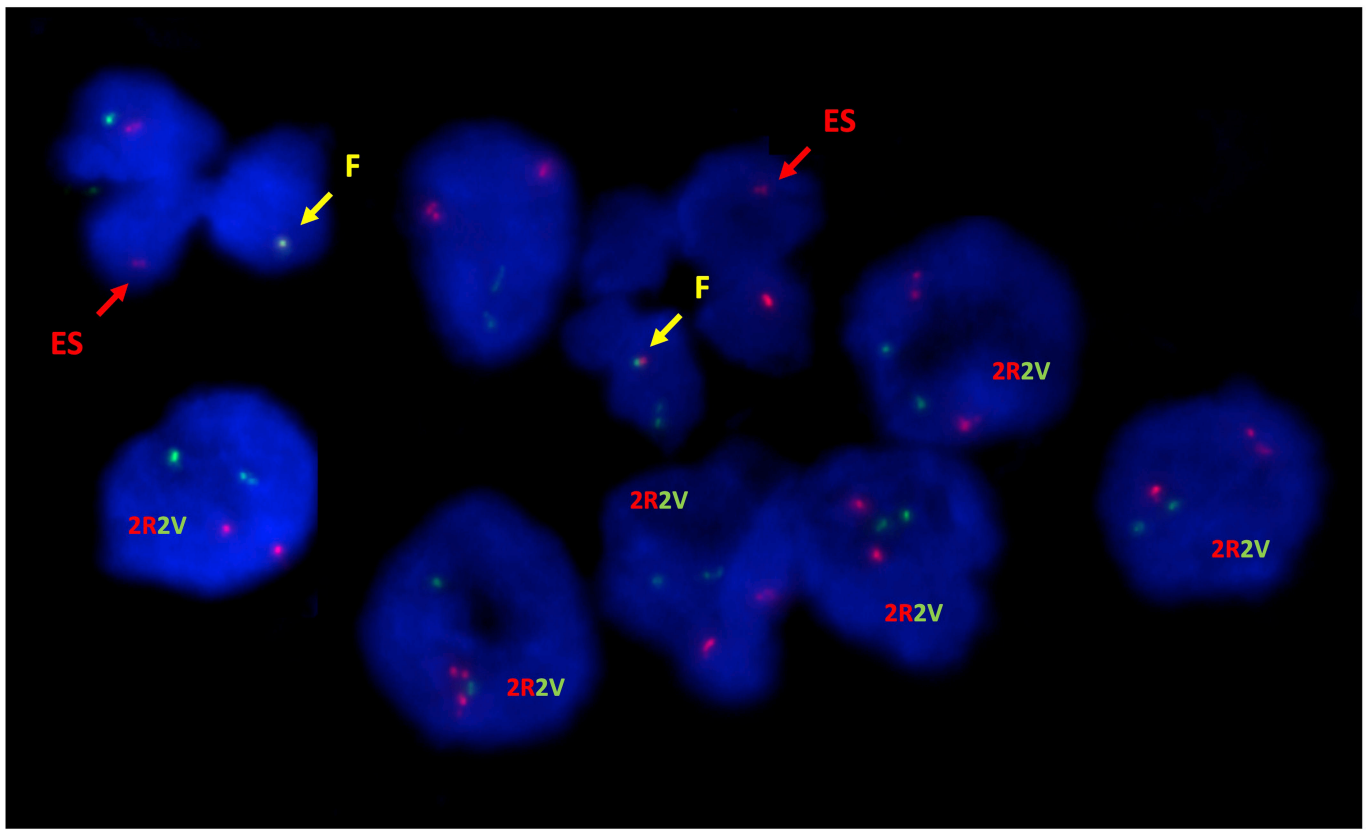

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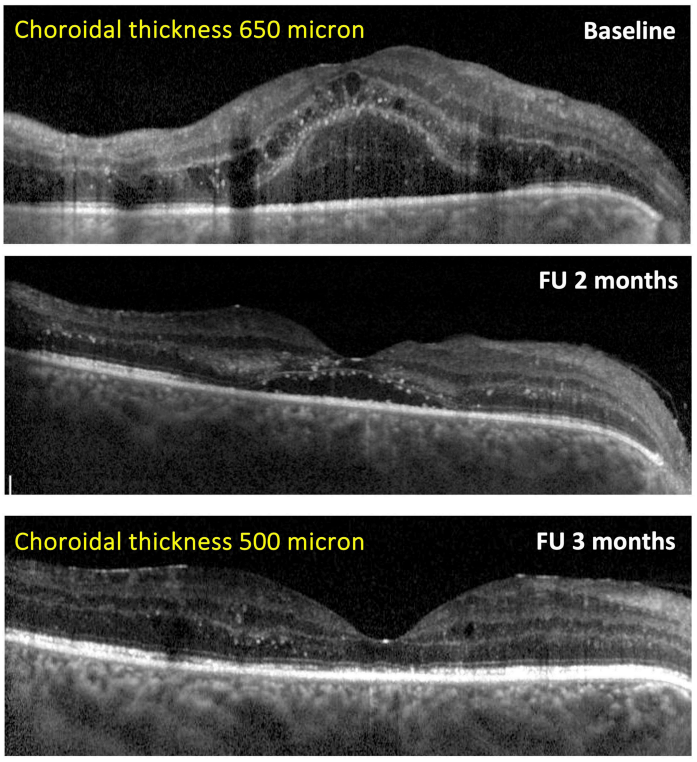
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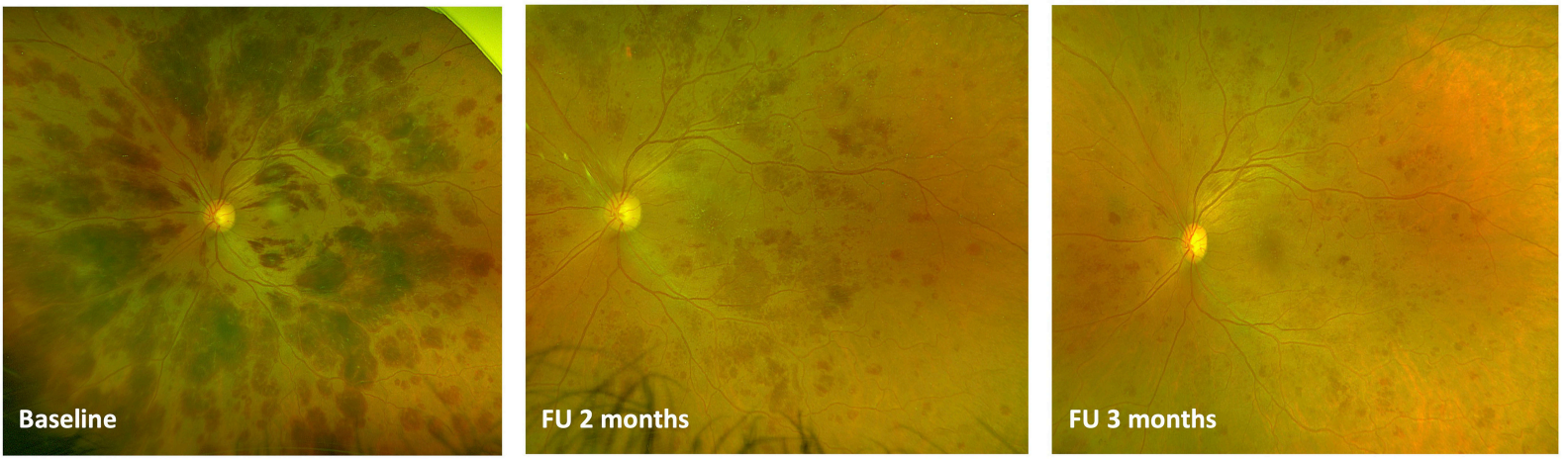
Baseline
FU 2 months
Choroidal thickness 500 micron
FU 3 months

3B


Baseline
FU 2 months
FU 3 months

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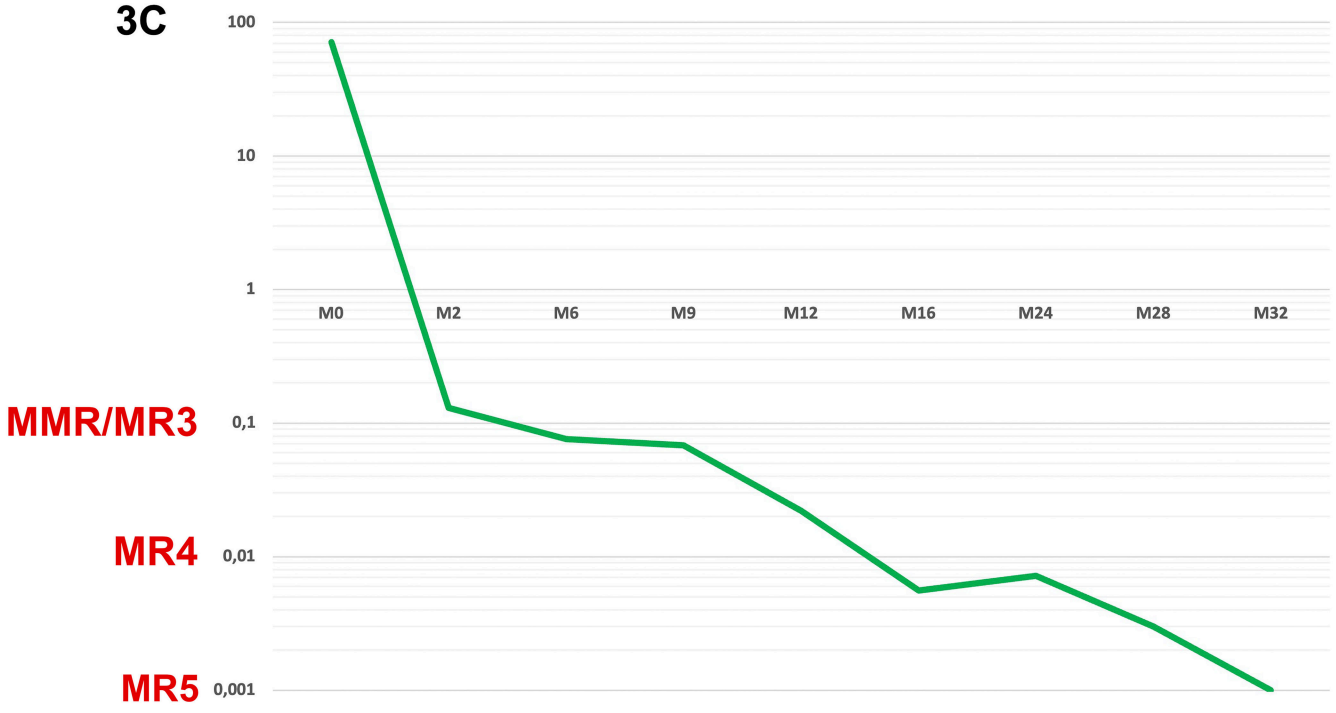
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