Megakaryocytes were hypolobulated. The patient presented with a left subdural hematoma following a mild traumatic brain injury in October 2022, requiring neurosurgical intervention. At that time, coagulation profile and platelet levels were normal. In December 2022, she experienced headaches again, with a subdural hematoma on the right side without any history of local trauma. The hematoma was drained once more, and laboratory tests continued to show normal platelet levels and coagulation profile. After discharge, the patient presented with neurological symptoms in January 2023, and a new right-sided subdural hematoma required two additional surgical interventions. Structural abnormalities were not detected on imaging exams. Given the recurrent episodes of severe intracranial hemorrhage with a risk of herniation, the hypothesis of platelet dysfunction associated with CMML was considered, and platelet transfusion was performed. After this intervention, there were no further bleeding episodes, and subsequent platelet aggregation testing confirmed hypoaggregation with collagen, ristocetin at a concentration of 1.2 mg/mL, adrenaline, and arachidonic acid. In outpatient follow-up, the patient showed an increase in white blood cell count and anemia with thrombocytopenia, and she received the AZAVIT-ABCDEF protocol (azacitidine, erythropoietin, filgrastim associated with high doses of vitamin B1, C, and D3) after signing the informed consent. There was no recurrence of central nervous system bleeding, and the leukocytosis and monocytosis were controlled. **Discussion:** CMML is a condition characterized by persistent monocytosis associated with dysplastic morphology in the bone marrow and can exhibit features of either myelodysplastic neoplasms or chronic myeloproliferative diseases. It is associated with various mutations, including the TET2 gene mutation, which occurs in up to 60% of patients and appears to lead to mild platelet dysfunction. Platelet transfusions stopped the intracranial hemorrhage, and high doses of vitamin C possibly restored the function of the TET2 gene. **Conclusion:** Advanced in molecular studies may further elucidate the heterogeneity of these myeloid disorders with uncertain clinical behavior and assist in therapeutic decision-making. The patient in this case was diagnosed with CMML and presented with recurrent subdural hematomas in the context of platelet dysfunction, with bleeding cessation achieved through platelet transfusion.

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INVESTIGATING THE IMPACT OF EGCG ON INEFFECTIVE HEMATOPOIESIS IN A MDS MOUSE MODEL

FID Via [a], KP Ferro [a], GR Assis-Mendonça [b,c,d], P Latuf-Filho [e], I Santos [a], NG Amôr [a], FVP Souza [a], MC Alvarez [a], STO Saad [a]

[a] *Centro de Hematologia e Hemoterapia (Hemocentro), Universidade Estadual de Campinas (UNICAMP), Campinas, Brazil*
[b] *Multipat Pathology Laboratory, Campinas, Brazil*
[c] *Collaborative Researcher Program, Department of Structural and Functional Biology, Institute of Biology, Universidade Estadual de Campinas (UNICAMP), Campinas, Brazil*
[d] *National Academy of Medicine, Young Leadership Physician Program, Rio de Janeiro, Brazil*
[e] *Center for Investigation in Pediatrics - CIPED, Campinas, Brazil*

Background and aims: Green tea catechins, particularly, epigallocatechin-3-gallate (EGCG), exhibit a range of beneficial effects, like apoptosis and differentiation-promoting effect which can help to counteract the uncontrolled proliferation and impaired differentiation trait of leukemia and myelodysplastic syndrome. This study aims to evaluate the effect of EGCG in vivo in mice model of myelodysplastic syndrome (NHD13 mice). **Methods:** To assess the effects of EGCG on MDS, 10-week-old female NUP98-HOXD13 mice and their littermate WT mice were treated with EGCG (50 mg/kg/day 5x/week i.p. for 4 weeks) or vehicle. Animals were sacrificed and bone marrow and spleen were obtained to evaluate immune system by flow cytometry and histology analyses. In addition, automated hematological counts and clinical biochemistry parameter analysis were done. Moreover, a 71-year-old man diagnosed with high-risk myelodysplastic syndrome (MSD) with 9% blast presence in the bone marrow, underwent a daily treatment of 800 mg EGCG intake, administered orally. Throughout this period, the patient underwent weekly regular clinical assessments and laboratory studies for monitor any potential toxicities. Additionally, peripheral blood samples were evaluated before and after the treatment to observe any alterations in immune cell composition. **Results and discussion:** NDH13+ exhibited, before the treatment, a reduction in the number of leukocytes, platelets and hemoglobin levels, which was in line with literature. EGCG treatment ameliorates the hematological parameters, visualized by the increase in the number of leukocytes (monocytes and neutrophils) and platelets; with no signals of toxicity - not morphological features nor enzyme levels changes in the liver. The expansion of Foxp3+ regulatory T cells leads the suppression of host antitumor responses and is a characteristic of progression to aggressive subtypes of the disease. Interestingly EGCG did not alter the percentage/frequency Foxp3+ regulatory T cells (CD4+CD25+ and/or CD4+FOXP3+), as well as the percentage/frequency of immature cells (CD45*Int*CD34+), in bone marrow and spleen. Our preliminary results showed an increase in extramedullary hematopoiesis in the spleen. One patient treated by EGCG for a month, exhibit reduction of regulatory T cells CD4+CD25+ and CD4+FOXP3+), and increased CD8+ T cells (CD3+CD8+) and natural killer cells (CD16+CD56+) accompanied with an increase of mature leukocytes (monocytes and neutrophils), with no changes in hemoglobin levels and platelets number. **Conclusion:** Our initial findings indicate that EGCG treatment may reduce ineffective hematopoiesis leading to an improvement of hematological parameters. These results align with the data obtained from one patient with high-risk myelodysplastic syndrome HR-SMD who received EGCG treatment. In summary, EGCG might be a promising adjuvant compound in the treatment of MDS. Further research and clinical studies are warranted to validate and explore its therapeutic benefits fully.

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