diagnosis for S-beta-Thalassemia. **Results:** The 1st male twin, FPS, 34 years old, black, works as a freelancer, has a mild to moderate clinical condition and does not need transfusions and/or hospitalizations. He does not use hydroxyurea. Her last hemoglobin profile showed a concentration of 13.7% HbFetal and 5.1% for HbA2. The 2nd male twin, VPS, 34-years old, black ethnicity, farmer, has had painful crises since childhood, with a history of several hospitalizations, mainly due to splenomegaly and splenectomy performed at 5-years of age. He has cholelithiasis and underwent cholecystectomy at the age of 22. He started using Hydroxyurea only at the age of 23, with significant improvement in symptoms and in the intensity and frequency of vaso-occlusive events. Her last hemoglobin profile showed a concentration of 22.6% to HbFetal and 4.9% for HbA2. The sister, CPS, 41-years old, black, works as a housemaid, was only diagnosed when she was 17-years old. She reported three miscarriages and one high-risk pregnancy at age 24 (with the HbAS trait). She currently has several painful crises/year, mainly pain in the lower limbs, aseptic necrosis of the femoral head, malleolar ulcer for over 15-years, with two blood transfusions/year. She has been using Hydroxyurea for 10-years. Her last hemoglobin profile showed a concentration of 16.9% for HbFetal and 5.3% for HbA2. **Discussion/conclusion:** The clinical variability of SCD, even in twins, has very different clinical manifestations, with several direct and indirect factors that modulate the manifestations, whether hematological, biochemical, inflammatory or genetic biomarkers, in addition to socioeconomic and demographic characteristics.

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TWO SICKLE CELL ANEMIA SIBLINGS: CASE REPORT

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Introduction/objective: Sickle Cell Anemia (SCA) causes different multisystemic manifestations with high morbidity and mortality, mainly characterized by vaso-occlusion, hemolytic anemia and vasculopathy. **Materials and methods:** We aimed to report a case of two Sickle Cell Anemia Siblings, with different clinical behavior, from the city of Atalaia do Norte, Amazonas, Brazil, assisted and monitored by the HEMOAM Foundation. **Results:** ASI*, the oldest brother, currently 19-years old, was diagnosed with Sickle Cell Anemia at the five years old, with few symptoms, no physical abnormalities, stature, and height normal, no hospitalization and without need for blood transfusion. He started using hydroxyurea only at 12-years old and remains stable and with mild comorbidities, needing only sporadic analgesia administration. Her last hemoglobin genotype showed a concentration of 16.7% for Fetal Hemoglobin (HbF). The younger brother, ASI**, aged 17, was diagnosed at the age of 4, with evolution to exuberant symptoms, with an average of eight painful crises/year, mainly in the lower limbs and with several hospitalizations and infectious intercurrences. He had splenomegaly and splenic sequestration, which resulted in splenectomy 1-year after his diagnosis (5-years old). He started using hydroxyurea at the 10-years old, and today he still presents with intense symptoms, cholelithiasis and cholecystectomy at the age of 15 and severe aseptic necrosis of the femoral head, resulting in monthly follow-up with an orthopedist. Her last hemoglobin profile showed a concentration of 7.10% for HbF. **Discussion/conclusion:** The symptomatological difference between siblings demonstrates that SCA has genetic and non-genetic factors involved in the severity of the disease and its clinical manifestations, which contribute to the clinical heterogeneity of the disease. Currently, at HEMOAM, we are researching candidate genes for sequencing in an attempt to identify possible genetic variants existing in the family and to be able to associate phenotypic complications of the disease with the differences and concomitantly investigate possible hematological, biochemical, immunological and environmental predictors that may influence this clinical diversity.

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LINFOMA DE HODGKIN E SÍNDROME DE MEDIASTINAL EM PACIENTE PEDIÁTRICO: ABORDAGEM CORRETA E PRECOCE DO PACIENTE VISANDO UM MELHOR PROGNÓSTICO

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Introdução: A Síndrome da Veia Cava Superior (SVCS) decorre de uma condição que leve à obstrução do fluxo sanguíneo na Veia Cava Superior (VCS). Ocorre por obstrução endovascular ou compressão externa da VCS por um processo patológico adjacente envolvendo pulmão