OP 24

SUCCESSFUL REDUCTION OF TRANSFUSION DEPENDENCE WITH LUSPATERCEPT IN A PATIENT WITH THALASSEMIA MAJOR: A CASE REPORT

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Objective: Thalassemia Major is a disorder of ineffective erythropoiesis with severe anemia, often requiring transfusions of RBCs throughout life. Transfusions are often required so frequently that the risk for iron overload and other complications strongly impairs the quality of life. Recently, this new erythroid maturation agent, luspatercept, has shown promise in reducing the transfusion requirements in patients with transfusion-dependent thalassemia. **Case Report:** A 40-year-old male patient with Thalassemia Major has been receiving regular erythrocyte suspensions since 2011, amounting to a total of 472 units by November 2023. The patient initially required an average of 2 units of RBCs per month to manage symptoms of fatigue and anemia. On February 17, 2023, the Luspatercept therapy was started at 75 mg every three weeks. Over the span of 22 treatments, one week after another, the need for RBC transfusions gradually diminished. The last transfusion was on November 21, 2023. The patient has since then maintained stable hemoglobin without further transfusion needs for approximately 10 months, a very impressive clinical improvement. **Discussion:** Therefore, this case offers a real-world view of the regard in which luspatercept proves effective in reducing transfusion requirements among patients suffering from Thalassemia Major. The sustained response for a period beyond 10 months really opens up possibilities for an overall better quality of life and reduction of the transfusion burden, which are important objectives in the management of transfusion-dependent patients. This report underlines early adoption of novel therapies such as luspatercept, which is considered instrumental in lessening complications resulting from chronic transfusions. This needs further studies and clinical discussions to optimize the dosing and duration of treatment in similar patients. This case adds to the growing body of evidence regarding the integration of erythroid maturation agents into standard management in patients with thalassemia.

Keywords: Thalassemia Major, Luspatercept, Transfusion Dependence, Erythrocyte Suspension, Ineffective Erythropoiesis.

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OP 25

PLEURAL EFFUSION DEVELOPING AS A CONSEQUENCE OF G-CSF ADMINISTRATION IN A PATIENT UNDERGOING AML TREATMENT

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Objective: Human granulocyte colony-stimulating factor (G-CSF) plays a vital role in boosting neutrophil production from hematopoietic progenitor cells, both in lab settings and within the human body. Beyond just raising neutrophil counts, G-CSF primes these cells, enhancing their ability to defend the body, making it a key player not only for neutropenic patients but also for those who are immunocompromised but not necessarily neutropenic. G-CSF is widely used in treating acute myeloid leukemia (AML), either alongside or following chemotherapy. One of its primary benefits is to speed up neutrophil recovery after chemotherapy, reducing both the length of hospital stays and the risk of infection. Here, we share a case involving a 17-year-old male with AML who developed pleural effusion after receiving G-CSF during his cytotoxic treatment. **Case Report:** The patient, a 17-year-old male, came to our clinic with an elevated white blood cell count and was subsequently diagnosed with acute myeloid leukemia (AML) after a bone marrow aspiration. He was immediately started on the 7+3 induction chemotherapy protocol. During the post-chemotherapy phase, when his neutrophil levels dropped, filgrastim (G-CSF) was introduced to help reduce the risk of infection and shorten the neutropenic period. For the first five days, everything seemed normal, and no side effects were noted. However, after that initial period, the patient began to experience worsening shortness of breath. Imaging revealed a growing pleural effusion on the left side. A diagnostic thoracentesis was performed, and the fluid was drained to provide relief. The analysis confirmed the fluid was transudative, with no signs of infection or malignancy. When the pleural effusion returned, G-CSF was promptly stopped, and the effusion rapidly resolved. After consolidation therapy, G-CSF was reintroduced, and once again, pleural effusion reappeared on the third day of treatment, but this too resolved spontaneously once the G-CSF was discontinued. A pleural biopsy showed no pathological findings, confirming that the G-CSF was likely responsible for the effusion. **Discussion:** In neutropenic patients, pleural effusion is typically linked to infections, but in this case, no signs of infection or AML involvement were found. The recurring pleural effusion, which resolved after stopping G-CSF, suggests a rare side effect of the treatment. Research indicates that G-CSF may trigger local inflammatory responses, including elevated cytokines like IL-6 and TNF-α, potentially leading to fluid accumulation in the pleura. This case highlights the importance of monitoring for unusual side effects during G-CSF therapy in AML patients.

Keywords: G-CSF, AML, pleural effusion, neutropenia, cytokine response.

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OP 26

MODERATE CLINICAL COURSE IN SEVERE ANAEMIA

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Introduction: Although iron deficiency anemia is more common in children and women, it can also occur in adult men, depending on their socioeconomic status and health conditions (1). Symptoms of anemia, such as dyspnea, tachypnea, tachycardia, pallor, heart failure, and cognitive dysfunction, can vary based on the severity of the anemia, its onset speed, the patient's age, and physiological condition. In young and generally healthy individuals, chronic anemia may go unnoticed until hemoglobin levels fall below a critical threshold or until physically demanding situations arise (2). We present a case of iron deficiency anemia with a hemoglobin level of 2.8 g/dL, yet the patient did not exhibit anemia symptoms that would disrupt hemodynamics. **Case Report:** A 43-year-old male patient with mental retardation secondary to meningitis during childhood presented to the emergency department in September 2024 with complaints of weakness, fatigue, and exertional dyspnea. On physical examination, blood pressure was 90/60 mmHg, heart rate was 94 bpm, respiratory rate was 18/min, skin and conjunctiva were pale; other findings were normal. Laboratory results showed WBC: $9.2 \times 10^3/\mu$L, hemoglobin: 2.8 g/dL, hematocrit: 9.1%, and platelet count: $365 \times 10^3/\mu$L. The patient was clinically admitted due to severe anemia. LDH and indirect bilirubin levels were normal. Peripheral smear revealed severe hypochromia and microcytic erythrocytes. No schistocytes or atypical cells were observed. For the etiology of anemia, serum Fe: 7 μg/dL TIBC: 294 μg/dL, ferritin: 3.6 ng/mL, folate: 6.68 ng/mL, and vitamin B12 was 375 pg/mL. Due to the hemoglobin level of 2.8 g/dL, the patient received 3 units of red blood cell suspension. After replacement, hemoglobin increased to 7.4 g/dL, and iron replacement was planned. Considering the patient's mental retardation and the difficulty in regular medication adherence, parenteral iron replacement was administered. The patient was discharged with a recommendation for follow-up in 2 weeks. **Results:** According to many anemia grading systems, a hemoglobin level dropping below 6.5 g/dL is considered life-threatening, and patients are theoretically expected to experience a range of symptoms (3). However, the literature reports cases where individuals sought medical assistance with hemoglobin levels below 3 g/dL and hematocrit levels below 10% (4,5). Despite our patient having a hemoglobin level of 2.8 g/dL at the time of admission, serious anemia symptoms such as tachycardia and tachypnea were not observed. As a result, as seen in our male patient, the activation of adaptive mechanisms in chronic anemia can allow for a mild clinical presentation. Even at critical hemoglobin levels, patients may present with moderate symptoms

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