

doença em remissão após um transplante de medula óssea ou do tratamento inicial, a lenalidomida pode ser administrada como tratamento de manutenção para prolongar a remissão. **Objetivo:** O objetivo deste trabalho consistiu na avaliação das reduções de dose devido as reações adversas apresentadas por pacientes com Mieloma Múltiplo em tratamento com lenalidomida. **Método:** Foi realizada análise retrospectiva em prontuários em uma clínica oncológica privada, no período de janeiro de 2019 a julho de 2020. Nesta análise, foram avaliados 37 pacientes de 66 a 80 anos, sendo 23 homens e 14 mulheres, de 1ª a 8ª linha de tratamento, elegíveis para tratamento com lenalidomida. As indicações avaliadas foram MM pós transplante autólogo de células-tronco (5 pacientes), MM refratário/recidivado (24 pacientes), MM recém diagnosticado NÃO elegível à transplante (6 pacientes), MM refratário/recidivado (MM IgG Lambda) (1 paciente) e MM manutenção (1 paciente). **Resultados:** No estudo foi evidenciado que 57% dos casos analisados foi necessária a redução de dose devido as reações adversas ao medicamento (RAMs). As RAMs foram divididas em: astenia (23%), reações cutâneas (8%), mielotoxicidade (53%) e toxicidade gastrointestinal (8%) e outros (8%). Em 6% dos casos, pacientes com menos de 65 anos, 38% em pacientes acima de 75 anos e 56% em pacientes de 65 a 75 anos. **Conclusão:** As reduções de dose apresentadas no trabalho foram majoritariamente a mielotoxicidade e astenia, em pacientes de 65 a 75 anos, e nesses casos o tratamento foi temporariamente interrompido ou suspenso, conforme necessário, para controle da toxicidade.

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419

ANAPLASTIC PRIMARY PLASMA CELL LEUKEMIA WITH AMPLIFICATION OF THE CCND1/IGH GENE FUSION

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Objective: To describe a case of Anaplastic Primary Plasma Cell Leukemia (aPPCL), linking clinical presentation, morphological and cytogenetic alterations. **Materials and Methods:** Clinical laboratory evaluation of aPPCL diagnosed at Instituto do Câncer do Estado de São Paulo - Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo. **Results:** Male, 63 years old, born in Bolivia, no previous comorbidities, presented with a 6-month course of lower back and rib cage pain associated with 20 kg weight loss and significant loss of functionality. No lymphadenomegaly or abdominal visceromegaly in physical assessment. Laboratory evaluation shows Hb 6.6 g/dL, Leukocytes 9660/mm³; (Neutrophils 7000/mm³; Lymphocytes 1200/mm³; Monocytes

1200/mm³), Platelets 239000/mm³; Cr 4.5 mg/dL (eGFR CKD-EPI 13 mL/min/1.73 m²); Serum monoclonal protein of 0.1 g/dL and urinary of 440 mg/24 hours output, free kappa seen in serum and urine immunofixation. 46.4% of pleomorphic plasmacytes were visualized in marrow aspirate, usually of large size, some presenting convoluted nucleus; some binucleated, others multinucleated, with marked nucleolus; Bone marrow biopsy was hypercellular at the expense of plasma cell infiltration (80% of nucleated cells); Increased reticulogenesis - MF2/MF3; Immunophenotyping by flow cytometry shows positivity for CD38, CD138, CD117, kappa and negativity for CD45, CD20, CD56, lambda. Peripheral blood smear was reevaluated, with 15% of cells with morphology and immunophenotype similar to those found in bone marrow. Male karyotype without clonal abnormalities. Interphasic FISH in CD138-positive cells shows signs suggestive of amplification of the IGH/CCND1 gene fusion in 70% of the analyzed nuclei; amplification CKS1B and CDKN2C (chromosome 1), deletion of the RB1 gene and D13S25 region (chromosome 13q) and deletion of the P53 gene (chromosome 17p) in 50% of the analyzed nuclei. Tomography shows multiple lytic bone lesions distributed diffusely, multiple pathological fractures and absence of extra-osseous tumor component. With an established diagnosis of Free Kappa aPPCL, intensive chemotherapy treatment was not started due to poor Performance Status. Therapy was then initiated with Bortezomib IV 1.5 mg/m²/weekly; Oral Cyclophosphamide 300 mg/m²/weekly; Oral Dexamethasone 40 mg/weekly. **Discussion:** Anaplastic multiple myeloma (MM) is a morphological variant of MM that occurs rarely and historically with a very aggressive course. It is characterized by the presence of pleomorphic and immature plasma cells, sometimes resembling megakaryocytes. It is more common in young patients, associated with paraprotein IgA and with predisposition to involvement of extramedullary sites. Cytogenetic abnormalities of poor prognosis such as 1q21 amplification, 17p deletion (p53) and t(4;14) are more common. Although CCND1/IGH gene fusion is frequently found in MM and PCL, gene amplification is a very rare finding and is particularly described in PCL, and could play an important role in the pathogenesis of this disease. **Conclusion:** This case shows two interesting aspects of plasma cell dyscrasia: the involvement of peripheral blood by anaplastic plasma cells and the amplification of the CCND1/IGH gene fusion. Recent studies have shown the potential effectiveness of BCL2 inhibitors in t(11;14), requiring confirmation in new clinical studies.

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420

ASPECTOS DEMOGRÁFICOS DOS PACIENTES PORTADORES DE MIELOMA MÚLTIPLO ADMITIDOS NO HOSPITAL DE BASE DO DF ENTRE 2013-2019: ANÁLISE DE DADOS DA APAC

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