decorrer do estudo foram analisados um total de 133 ICS, sendo 65 (49%) em portadores de leucemia aguda (49%) e 103 (77%) em pacientes submetidos a TMO. Um total de 124 ICS por agente único e nove polimicrobianas. Gram negativos (GN), Gram positivos (GP) e candidemias foram 75 (60%), 50 (40%) e 8 (6%), respectivamente. O tempo mediano de positivação foi de 28 horas (variando de 1 a 77 horas), sendo em 47 (35%) dos eventos menor que 24 horas, 71 (53%) entre 24 e 48h e em 11 (8%) entre 48 e 72h, respectivamente. A mediana do TPP nas ICS por GN foi 25 horas vs. 32h nas GP e 33h nas candidemias ($p = 0.013$). TPP < 48 horas foi identificado em 88% das ICS por BGN, 89% das ICS por CGP e 100% das candidemias. O tipo de frasco primeiro positivar sofreu influência dos diferentes tipos de ICS. Nas candidemias, o frasco de aeróbio foi o mais rápido ($p = 0.03$). Nas ICS por enterobactérias, o menor TPP foi mais frequente nos frascos de anaeróbio ($p = 0.01$). Em relação ao local de coleta (sangue periférico vs. cateter), não houve diferença. Dentre as 21 ICS por GN MDR, não houve diferença no TPP comparando GN MDR vs. GN não MDR (23h vs. 23h; p = NS) e todas ICS por GNMDR tiveram TPP <48h. Conclui-se que em cerca de 90% das ICS o TPP foi menor que 48h. Não houve ICS por GN MDR com TPP > 48h. Houve benefício na coleta pareada (aeróbio e anaeróbio), e a coleta via cateter não foi inferior em relação a periférica para o desfecho analisado. O TPP mostrou-se uma ferramenta de uso prático para adequação de condutas antimicrobianas empíricas nesta população hematológica de alto risco.

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LEISHMANIOSE VISCERAL E O PAPEL DO MIELOGRAMA NO DIAGNÓSTICO

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Introdução: Leishmaniose visceral (calazar; LV) é uma doença sistêmica crônica que acomete baço, linfonodos, fígado e medula óssea (MO). É causada por protozoários do gênero Leishmania, transmitido pela picada de mosquitos flebotomíneos infectados, sendo o cão seu principal hospedeiro reservatório. O período de incubação pode ser longo, variando de dois a seis meses. O quadro inicia-se com sintomas de mal estar, febre, perda de peso e hepatoesplenomegalia e tem curso insidioso. Sabidamente, os parasitas se replicam no sistema reticuloendotelial, podendo acarretar em citopenias importantes, devido à supressão da MO, hemólise e até sequestro esplênico. **Objetivo:** Apresentar o caso de um paciente que desenvolveu quadro de LV insidiosa, com pancitopenia e diagnóstico realizado no exame microscópico do mielograma. **Relato do caso:** Homem, 75 anos, morador do bairro Piedade no Rio de Janeiro - RJ, hipertenso e diabético, iniciou em outubro de 2020 quadro de astenia e fadiga, sem febre, com hemograma evidenciando pancitopenia leve. Na ocasião, foi diagnosticado com tumor neuroendócrino, tratado com exérese da lesão. Ficou em seguimento ambulatorial com Hematologia, devido à pancitopenia não resolvida. Em setembro de 2021, durante consulta de rotina observou-se queda importante da contagem plaquetária, com trombocitopenia grave (< 20.000/mm^3). A análise do sangue periférico evidenciou presença de leve rouleaux de hemácias e trombocitopenia importante. Realizou aspirado e biópsia de medula óssea, com mielograma revelando presença de vários parasitas extra e intracelulares, morfologicamente sugestivos de Leishmania. Paciente convocado para internação e na admissão foram observados hepatoesplenomegalia e história epidemiológica compatível com a hipótese de leishmaniose; apesar de viver em área não endêmica, possuía 7 cachorros, 6 deles falecidos sem causa conhecida. Realizou novo mielograma, para coleta de cultura para Leishmania, com resultado confirmatório. O laudo histopatológico da MO confirmou a presença de inúmeros histiócitos, por vezes agrupados, com inúmeras formas amastigotas de Leishmania, sp. O tratamento escolhido foi anfotericina B e após 10 dias houve melhora clínica, com redução das visceromegalias e citopenias, recebendo alta hospitalar. **Discussão:** A tríade típica de sintomas da LV: anemia, esplenomegalia e febre, pode não estar completa em todos os casos. As opções de métodos diagnósticos da doença incluem visualização do amastigota em esfregaços ou tecidos (geralmente medula óssea ou baço); isolamento do parasita por cultura; detecção por PCR do DNA do parasita e testes sorológicos. O aspirado de MO (para histopatologia, cultura e testes moleculares) é a amostra diagnóstica de escolha, pois o aspirado do baço está associado ao risco de hemorragia. O histopatológico requer visualização de amastigotas (corpos esféricos que medem de 1 a 5 mícrons, encontrados dentro de macrófagos, mas também podem ser visualizados no ambiente extracelular, possuem um grande núcleo e uma proeminente organela em forma de bastonete chamada cinetoplasto). **Conclusão:** O exame da medula óssea embora seja invasivo, quando realizado no paciente com citopenias importantes tem boa acurácia e pode ajudar com o diagnóstico na análise citológica inicial (mielograma).

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NEUTROPHIL COUNTS RELATED TO PRESENTATION OF COVID IN AUTOIMMUNE NEUTROPENIA?

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Objective: To describe 2 cases of autoimmune neutropenia (AIN) patients infected with Sars-Cov-2. **Design/Method:** Two subjects case report. **Results:** Case report 1: A girl with primary AIN since 1 year and 10 months old, maintaining severe neutropenia and mild recurrent infections. Presented to the emergency department in June/2020, at 3 years and 8 months old, with flu-like symptoms, afebrile, in good general condition. Physical examination was normal. The absolute neutrophil count (ANC) was 0.279×10^9/L. At hospital admission, Sars-Cov-2 (RT-PCR) tested positive and filgrastim (G-CSF) 5 μg/kg/day was initiated. Chest X-ray was also normal and blood culture resulted negative. She remained in great general

condition, afebrile, and was discharged on the 2nd day of hospitalization, with clarithromycin (15 mg/kg/day). After G-CSF, ANC: 0.494×10^9/L (1st dose), 1.431×10^9/L (2nd dose). On outpatient follow-up, she had no long-term complications from Covid-19. Case Report 2: A man with chronic immune thrombocytopenia purpura (ITP) since 2008, autoimmune hemolytic anemia since 2013, evolved with AIN on May/2020, at 42 years old, with ANC lower than 0.5×10^9/L. On 6/1/2020, he had ANC 0.170×10^9/L. On 6/25/2020, he started flu-like symptoms, had ANC of 5.118×10^9/L, and tested positive for Sars-Cov-2 (RT-PCR). He kept high fever (102,2°F) and was hospitalized for 10 days without use of G-CSF. After discharge, on outpatient follow-up, he had no long-term complications from Covid-19, and presented ANC 0.338×10^9/L (Aug/2020). **Discussion:** At beginning of Covid-19 pandemic, severity infection in children was unknown. Today is known that most of them have milder clinical course, regardless of chronic diseases. In adults, in contrast, the inflammatory response tends to exacerbation, with more severe clinical conditions. Furthermore, many case reports of patients infected by SARS-CoV-2 with comorbidities literature are published. However, to date there are no reports on the impact of COVID-19 in AIN patients. Increased neutrophil counts during infectious episodes are common in AIN, which appears to be related to the benign course of most infections. We reported 2 cases of AIN patients diagnosed with Covid-19, both with favorable clinical outcomes despite heterogenic clinical course. On the first case, she presented few symptoms and ANC increased only after using G-CSF. On the second reported patient, there was a spontaneous increase of ANC and greater inflammatory response than the first case. It could suggest a correlation between inflammatory response to COVID-19 and ANC in cases of autoimmune neutropenia. **Conclusion:** In the reported cases, clinical course of disease and neutrophil count were different between adult and pediatric patients. It is not possible to state whether this difference is due to age group, individual response to infection or other variables. It is important to assess other cases of AIN infected by COVID 19 to better understand correlation between severity of infection and neutrophil count response.

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IS GFI-1 A TRIGGER TO DEVELOPMENT OF AUTOIMMUNE NEUTROPENIA?

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Objective: To describe a child carrying a *GFI1* -variant with autoimmune neutropenia. **Design/Method:** Single subject case report. **Results:** This is a case report of a 1 year-and-10-months-old female presenting severe chronic neutropenia ($0{,}03 \times 10^9$/L) with mild infections. The frequency of infections reduced as she got older. Physical examinations outside of infections were always normal and she had expected development. She did not have monocytosis or other hematologic disorders, and lymphocytes evaluation by flow cytometry analysis resulted normal. Viral diseases were negative, immunoglobulins dosage was normal for the age group, and screening for systemic autoimmune disease (sAID) was negative. Bone marrow aspirate analysis with karyotype resulted normal, without maturation arrest or dysplasia. Inborn errors of metabolism (IRM) and immunity (IRI) were ruled out. Anti-neutrophil (anti-HNA) autoantibody was detected on two occasions: at ages of 2-years-and-9-months-old and 5-years-and-3-months-old, presenting the same intensity and the same pan-reactivity to the FCγRIIIb glycoprotein. She has no siblings, but her father presented recurrent otitis until 6-year-old and hemogram with a persistent mild neutropenia documented since childhood. Ethnic benign neutropenia was ruled out. Both expressed Fyb on erythrocytes and were heterozygotes for single nucleotide polymorphism *rs2814778* in the *ACKR1*. She did not present remission of the disease until the age of 5 years and 7 months. A whole exome sequencing, showed a heterozygotic non-pathogenic *GFI-1* variant in exon 6 (c.929G>A:p.Arg310Gln). The variant, confirmed by Sanger sequencing, was also present in her father. He has no anti-neutrophil antibodies detected to date. **Discussion:** Most cases of pAIN are diagnosed between 3 to 8 months. Spontaneous remission in about 90% could be expected in 7 to 24 months of diagnosis or up to age of 4-5 years. In this reported case, the patient had severe neutropenia demonstrated since 1 year-and-10-month-old. Even 45 months after the beginning she remains neutropenic, carrying anti-HNA autoantibody with the same pan-reactivity features. The persistence of anti-HNA with no specificity may be associated to persistent autoimmune neutropenia (AIN) as well as a characteristic of secondary AIN, but screening for both sAID and IEI were negative. Due to the persistence of neutropenia, a whole exome sequencing was performed, presenting *GFI-1* variant inherited from her father, also neutropenic. The patient does not present features of *GFI-1* -variants, clinical course and the bone marrow were not compatible with SCN. In addition to regulating myelopoiesis, *GFI-1* also plays a role in the maturation of B and T lymphocytes, and in process of antigen-mediated lymphocyte activation. Although mature B-cells do not express *GFI-1*, this loss may be correlated with the increase of some IgG fractions, and some works have correlated *GFI-1* changes with autoimmune conditions and could be the cause of AIN reported. **Conclusion:** For patients with persistent pAIN, perfoming tests to detect a genetic involvement may be useful to understand the cause of the disease. In this case, the presence of *GFI-1* variant could be the trigger of AIN reported.

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AN UNUSUAL CASE OF NEUTROPENIA

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