mediate tumor progression, invasion, and therapeutic resistance in several cancers. Its role in ALL, however, remains largely uncharacterized. These findings highlight ADAM genes as potential biomarkers and therapeutic targets, warranting further functional and clinical investigation to elucidate their contribution to ALL pathogenesis. **Objectives:** To investigate patterns of chromosomal alterations in pediatric ALL patients, with a focus on the ADAM gene family, and to describe potential related impacts. **Material and methods: Array comparative genomic hybridization (aCGH):** Sixteen samples from pediatric patients diagnosed with ALL were analyzed. Genomic DNA was extracted using the Pure Link Genomic DNA Mini Kit (Invitrogen, California, USA). The aCGH analysis was performed using the Agilent 4x180k CGH + SNP microarray (Santa Clara, USA). Following extraction, the DNA was digested with a restriction enzyme and labeled with the Cy5 fluorochrome, employing random primers and the exo-Klenow fragment DNA polymerase. Control DNA was labeled with the Cy3 fluorochrome. Patient and control DNA samples were combined and hybridized onto the microarray. Data interpretation was carried out using Agilent's CytoGenomics v5.0 software. **Results:** Alterations were identified in at least one member of the ADAM family and on different chromosomes in all patients analyzed by aCGH. Notably, ADAM6 showed 23 occurrences (gain and amplification effects), ADAM3A showed 10 occurrences (deletion, loss, and amplification effects), and ADAM5P showed 9 occurrences (deletion, loss, and amplification effects), with emphasis on chromosomal regions 14q32.33 and 8p11.22. **Discussion and conclusion:** Amplification of ADAM6 may enhance growth factor– and cell surface receptor–dependent processes that reinforce pro-proliferative signaling pathways and anti-apoptotic mechanisms in lymphoid blasts, suggesting a stronger association with B-lineage ALL. Conversely, losses of ADAM3A and ADAM5P may impair cell adhesion and remodeling of the bone marrow microenvironment, facilitating immune evasion, expansion of aberrant clones, and migration of these cells into the peripheral blood. Although numerous reports have linked mutations in these genes to renal, lung, and breast cancers, their roles in leukemia remain poorly characterized. The findings presented here demonstrate a strong association between the ADAM gene family and ALL patients, suggesting that ADAM dysfunction contributes to the pathogenesis of this malignancy and identifies these genes as potential targets for functional studies and therapeutic interventions. While larger studies are warranted, the data obtained from this pediatric cohort underscore the need to further explore the mutational profile of this population.

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CLASSIFICAÇÃO DE RISCO SAMLS NA LEUCEMIA MIELÓIDE AGUDA: RELEVÂNCIA EM CENÁRIOS DE RECURSOS LIMITADOS

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Introdução: A estratificação de risco na Leucemia Mieloide Aguda (LMA) é crucial para a tomada de decisão terapêutica e impacta diretamente na escolha do tratamento. A classificação European LeukemiaNet (ELN2022), exige informações citogenéticas e moleculares específicas que frequentemente são de difícil acesso para pacientes vivendo em países em desenvolvimento, limitando a estratificação adequada. O Survival AML Score (SAMLS) é um sistema de pontuação que integra o Adapted Genetic Risk (AGR) e busca reproduzir a classificação do ELN2022, contornando essa limitação ao combinar informações genéticas (mesmo quando incompletas) com dados clínicos acessíveis, como idade, contagem de leucócitos e dosagem de albumina ao diagnóstico. Esse escore foi desenvolvido por Silveira et al. (Blood, 2020). **Objetivos:** Propomos avaliar a aplicabilidade e o impacto do SAMLS em uma coorte de pacientes do sul do país, bem como verificar sua capacidade de reclassificar o risco prognóstico frente à estratificação convencional. **Material e métodos:** Foram analisados dados de 29 pacientes com diagnóstico de LMA atendidos no Hospital de Clínicas de Porto Alegre entre janeiro de 2023 e maio de 2025. A coorte incluiu 18 (62.1%) pacientes do sexo masculino e 11 (37.9%) do sexo feminino. A idade dos pacientes variou de 5 a 83 anos, com mediana de 59 anos (Intervalo Interquartil: 42-67.5). Os diagnósticos mais frequentes foram LMA (n = 16), LMA de risco desfavorável (n = 4) e LMA de risco intermediário (n = 2). A classificação de risco inicial dos pacientes, quando disponível, foi comparada com a estratificação SAMLS (baixo ou alto risco). **Resultados:** A aplicação do SAMLS resultou em uma reclassificação da estratificação de risco para 12 dos 29 pacientes (41.4%). Destes, 9 pacientes classificados como risco intermediário tiveram seu risco elevado para alto risco pelo SAMLS, refletindo a identificação de fatores clínicos ou genéticos adicionais que conferem pior prognóstico. Inversamente, 3 pacientes classificados como Risco Intermediário foram reclassificados como baixo risco pelo SAMLS, indicando um prognóstico mais favorável. Pacientes inicialmente em risco favorável ou adverso mantiveram, em sua maioria, a consistência de classificação inicial. **Discussão e conclusão:** A reclassificação de risco observada neste estudo preliminar sugere que o SAMLS possa refinar a estratificação de risco, permitindo uma avaliação mais precisa do prognóstico individual. Sua potencial aplicabilidade em cenários com recursos limitados, onde classificações com a ELN2022 não é possível, destaca-se como uma de suas principais vantagens. A integração do AGR, projetado para tolerar dados genéticos incompletos, com parâmetros clínicos de fácil acesso, posiciona o SAMLS como uma proposta para o avanço da medicina. No entanto, considerando que a população de nossos pacientes possa apresentar características e perfis biológicos distintos daqueles relatados nas coortes de validação do escore, é fundamental o acompanhamento longitudinal desses pacientes para confirmar a reprodutibilidade e a validade dos resultados prognósticos do SAMLS em nosso contexto local. Assim, o SAMLS se mostra como um recurso potencialmente

promissor em contextos de recursos limitados, cuja investigação poderá representar um avanço significativo na busca por maior equidade no acesso à saúde de qualidade para pacientes com LMA no Brasil.

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CLINICAL PROFILE OF DEATHS FROM ACUTE MYELOID LEUKEMIA TREATED AT THE AMAZONAS HEMATOLOGY AND HEMOTHERAPY FOUNDATION (HEMOAM)

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Introduction: Acute Myeloid Leukemia (AML) is a heterogeneous group of clonal diseases characterized by the uncontrolled proliferation of blasts in the bone marrow. Despite advances in the fields of diagnosis and treatment, the mortality rate remains high, often influenced by factors such as age and the specific subtype of leukemia. The elevated incidence of premature mortality is predominantly attributable to delayed diagnosis, which plays a pivotal role in shaping the clinical course. A retrospective analysis of clinical data can facilitate comprehension of the factors involved in the clinical outcome of these patients. **Objectives:** To describe the clinical profile and comorbidities of patients who died from Acute Myeloid Leukemia at the Hematology and Hemotherapy Foundation of Amazonas (FHEMOAM). **Material and methods:** The present study adopted a retrospective model, utilizing a database comprising medical records collected during the period spanning from January 2012 to January 2025. The study comprised a total of 183 patients. The subsequent data analysis employed descriptive statistics, incorporating both absolute and relative frequencies. **Results:** A slightly higher prevalence was observed among male patients, accounting for 102 cases (55.73%). The mean age was 35 years (ranging from 1 to 85 years). During the study period, 88 patients (48.08%) died, of whom 58% were male. The mean age among the deceased was 46.5 years, with the following distribution by age group: 0– 19 years (18.18%), 20–39 years (22.7%), 40 –59 years (18.18%), 60–69 years (19.3%), 70–79 years (11.3%), and ≥80 years (10.2%). The most prevalent FAB subtypes among the deaths were: AML not otherwise specified (NOS) (55.7%), followed by M3 (10.2%), M5 (9.1%), M2 (6.8%), M4 (5.7%), M7 (3.4%), M0, M1, and biphenotypic leukemia (2.3%), and M6 (1.13%). Frequently observed comorbidities in these patients included: systemic arterial hypertension (18.2%), diabetes mellitus (15.9%), other neoplasms (14.7%), pulmonary disease (11.3%), cardiopathies (9%), hepatitis (7.9%), and chronic kidney disease (4.5%). **Discussion and conclusion:** The mortality rate and the higher prevalence among males align with national literature, as do the mean age and other demographic characteristics. These findings underscore the need for further investigation into environmental factors that may contribute to disease progression. Among these factors, socioeconomic conditions are particularly salient, given their direct impact on patients access to diagnosis, treatment, and overall health outcomes. The data also suggest that chronic metabolic comorbidities, such as hypertension and diabetes, are generally associated with an increased risk of death in patients with AML. These conditions are among the primary factors influencing prognosis and survival rates. It is hypothesized that the findings of this study will contribute to the identification of specific risk profiles, thereby the development of more effective care strategies for patients with AML in the state of Amazonas. Furthermore, emphasis is placed on the necessity of implementing targeted strategies for monitoring patients with comorbidities, as these conditions have a directly impact on prognosis and overall survival.

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COAGULOPATIA INTRAVASCULAR DISSEMINADA COMO MARCADOR DE MORTALIDADE PRECOCE NA LEUCEMIA MIELÓIDE AGUDA NÃO PROMIELOCÍTICA: UM RELATO DE CASO

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Introdução: A coagulopatia intravascular disseminada (CIVD) é uma complicação significativa em até 21% das leucemias mieloides agudas (LMA) não promielocíticas, configurando