Rego e colaboradores analisaram os subtipos linfocitários na medula óssea de 44 crianças de 2 meses a 15 anos sem doença auto-imune ou hematológica e compararam os resultados com 12 adultos hematologicamente saudáveis.[4] Posteriormente, publicaram uma comparação da densidade antigênica de CD10 e CD19 entre os precursores de célula B normais e linfoblastos.[5] Farahat e colaboradores,[6] utilizando citometria de fluxo quantitativa, demonstraram menor quantidade de moléculas de TdT e CD19 e maior quantidade de moléculas de CD10 em blastos leucêmicos comparados com as células linfóides B precursoras.

Nos últimos anos, o desenvolvimento da metodologia de imunofenotipagem por citometria de fluxo multiparamétrica tornou possível utilizar três a quatro cores simultaneamente. O conhecimento adquirido das expressões antigênicas na ontogênese da linhagem linfocitária B tem auxiliado na distinção entre as células linfóides B precursoras e os linfoblastos residuais leucêmicos, como pode ser lido nos artigos publicados por Wells DA,[7] Borowitz MJ[8] e Campana D.[9]

A população de hematogônias exibe um complexo espectro de expressão antigênica que define a evolução normal da linhagem precursora B; dependendo do estágio de maturação pode expressar CD10, CD19, CD34 ou TdT, CD10 e CD19 ou CD19 e CD22.

As hematogônias consistem em células B em estágio médio, baixa proporção em estágio mais precoce ou blastos e poucas células B mais maduras. Na imunofenotipagem se caracterizam por uma expressão variável do CD45, forte expressão do CD10 e CD19 e somente pouco dessas células expressam antígenos CD34, TdT ou marcadores CD20 e imunoglobulinas de superfície.[10]

A positividade das hematogônias para o CD10, CD19, HLA-DR, TdT e CD34 pode mimetizar as células leucêmicas residuais, mas a maturação incompleta, o imunofenotipo assincrônico, as expressões com marcadores de linhagem não B, as super ou subexpressões antigênicas auxiliam na caracterização definitiva da célula linfoblástica.

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Plasma concentrations of D-Dimer predict mortality?

Concentração plasmática de Dímero-D é um preditor de mortalidade?

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Atherosclerosis is characterized by a nonspecific local inflammatory process which is accompanied by a systemic response. Since such an inflammatory response is present at all stages of atherogenesis, a large number of emerging inflammatory biomarkers have been identified during the past

decade, which might aid in identifying individuals at high risk for coronary heart disease(CHD).

Fibrin D-Dimer is a major fibrin-specific degradation product that detects cross-linked fibrin resulting from endogenous fibrinolysis. D-Dimer can be considered as a global marker for the direct measurement of ongoing turnover of cross-linked fibrin and for an activation of the haemostatic system without reflecting the changes in fibrinogen and fibrin status. In addition, due to the introduction of ELISA, measurement of D-Dimer is well established in clinical practice as a screening marker of activated coagulation. D-Dimer plays an important role in the detection of hypercoagulable states such as acute symptomatic deep-vein thrombosis, pulmonary embolism or disseminated intravascular coagulation (CVD) having a high negative predictive value.

Taking into account that thrombogenesis is one of the fundamental pathological processes underlying the major complications of atherosclerotic disease, measurement of D-dimer might be useful to identify patients at high risk for future CVD events .

Indeed, in the Physician's Health Study,[1] a strong association between increased D-Dimer concentrations and future myocardial infarction was found in multivariate analysis, after controlling for nonlipid risk factors and Lp(a). However, if total and high-density lipoprotein cholesterol, as well as endogenous t-PA and its primary inhibitor, PAI-1, were included in the analysis, its association was attenuated and no longer statistically significant. Future ischemic events were predicted by D-Dimer levels in a study by Lowe *et al.*,[2] but not by other markers of procoagulant activity, such as prothrombin fragment F1+2 and thrombin-antithrombin complex (measures of thrombin generation), or by factor VII coagulant activity. Increased levels of D-Dimer also predicted future CHD events in apparently healthy middleaged participants of a large community-based prospective study.[3] Comparison of men in the top tertile with those in the bottom tertile of the baseline fibrin D- Dimer distribution resulted in an odds ratio for CHD of 1.79 (95% CL, 1.36-2.36) after adjustmente for smoking, other classical risk factors, and indicators of socioeconomic status. Subsequent meta-analysis, including this and four other population-based cohorts and two studies in patients with stable CVD, yielded almost the same results: an odds ratio of 1.7 (95% CI,1,3-2,2) was found for subjects with D-Dimer levels in the top tertile compared with those in the bottom. In the Atherosclerosis Risk in Communities study, several haemostatic parameters (t-PA, PAI-1, plasminofen and prothrombin fragment F1+2, D-Dimer) were measured in 326 incident CHD cases during a follow-up of 4.3 years and in a randomly stratified referent cohort of 720 individuals. Among these biomarkers only- D-dimer showed a powerful predicitive value for incident CHD with a RR of 4.21 (95% CI, 1.9-9.6).[4] Finally, in there more recently published reports the strength of the association between elevated levels of D-Dimer and cardiovascular events was similar to those preciously found in meta-analysis, revealing multivariate-adjusted risk estimates of 1.86(95% CI,1.24-2.80.[5]

We conclude that pulmonary embolism can be exclude in patients who have low clinical probability of pulmonary embolism and negative erythrocyte agglutination D-Dimer test results and that further diagnostic testing is not beneficial to this population. These findings are present in approximately 50% of outpatients and 20% of inpatients with suspected pulmonary embolism.

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