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Sommario	Introduction Mixed cryoglobulinemia (MC) is characterized by the production of monoclonal (type II MC) or polyclonal (type III MC) rheumatoid factors (RF), which form with endogenous IgG cold-precipitable immune complexes that cause small-vessel vasculitis and multi-organ damage. Hepatits C virus is the causative agent in 90% of MC patients, usually characterized by the expansion of an anergic B cell subpopulation called CD21low B cells. Only a minority of the patients has idiopathic or essential MC (EMC) and the B cell population has been scarcely investigated so far. Objective: to characterize the phenotypical and functional proprieties of B cells in EMC and compare them with those of HCV-related MC and from healthy donors. Method The B cell phenotype and function was studied in 13 patients with EMC and compared to 24 patients with HCV-MC. The proliferative response of B cells was investigated through the CFSE assay, the intracellular pERK content was measured by the BD Phos-Flow system and apoptosis was measured through annexin/7AAD staining. All the analyses were performed by flow-cytometry. Results EMC patient showed significant lower absolute numbers of circulating B cells compared to HCV-MC (mean ± SD: 185/mm3 ± 236 vs 529/mm3 ± 795). Interestingly percentages and absolute numbers of CD21low B cells were

	significantly higher in EMC compare to HD but lower than HCV-MC patients. Similarly to CD21low B cells found in HCV MC, CD21low B cells in EMC proliferated poorly in response to TLR9 stimulation, displayed dysregulated pERK signaling and were apoptosis prone. Conclusion Similar features of virus-specific exhaustion and anergy induced by continual antigenic stimulation observed in B cells expanded in HCV-MC are found in B cells EMC. Our findings open the question of a possible role of a still yet unknown antigen responsible for the development of EMC.
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