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Acute Coronary Syndromes

COMPARISON OF CIRCULATING DENDRITIC CELLS AND MONOCYTE SUBSETS AT DIFFERENT STAGES OF ATHEROSCLEROSIS

Poster Contributions
Hall C
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Background: Proinflammatory dendritic cells (DCs) and monocytes are critically involved in the proceeding and destabilization of atherosclerosis. Recent studies have reported potential associations of specific patterns of circulating DCs and monocytes with the incidence of coronary artery disease (CAD) and ST-elevation myocardial infarction (STEMI); however, further information of DC and monocyte subsets on plaque morphology and vulnerability is uncertain and required.

Methods: Thirty-eight CAD patients with borderline lesions (stenosis 50%-70%) by coronary angiography (CAG) were enrolled, while 27 subjects free of luminal diameter narrowing ≥ 50% served as controls. Likewise, 32 patients with STEMI were enrolled and confirmed with the presence of thrombosis by CAG. Plaque features of 38 CAD patients were evaluated at the site of the minimum lumen area and culprit lesions by optical coherence tomography. Peripheral blood (6 ml) was collected from each patient and drawn into heparin-anticoagulated tubes at entry. Circulating myeloid DCs (mDCs), plasmacytoid DCs (pDCs) and monocyte subsets were analyzed using flow cytometry.

Results: There was no discrepancy in the peripheral total white blood cell count, differential count among three groups. Compared to control group, patients with CAD and STEMI had significantly lower proportions of mDC1, mDC2, pDC and a remarkable higher proportion of monocytes with intermediate CD16 expression (Mon2, CD14+CD16+) in peripheral blood. In the OCT subgroup, patients with thin-cap fibroatheroma (TCFA) had a lower proportion of mDC2 than those without TCFA (0.72% ± 0.41% and 1.39% ± 0.38%, P=0.025). Otherwise, Mon2 proportion retained a higher level in patients with TCFA relative to those without TCFA (17.69% ± 3.32% and 13.01% ± 2.71%, P=0.031). When proportions were converted into absolute value, as oppose to a gradual rise in the number of Mon2/ml, the number of mDC2/ml remain gradually decreased with the progress of plaque vulnerability.

Conclusion: Circulating subsets of mDC2 and Mon2 appear to be promosing markers of plaque stabilization and rupture.