脉内膜增厚、斑块形成、管腔显著狭窄,斑块内见大量泡沫细胞、脂质丰富、纤维帽菲薄,提示为不稳定性粥样斑块,结合5-LO和LTB4、LTD4的表达变化,可以推论5-脂氧合酶通路与斑块稳定性有密切关系。5-脂氧合酶通路引起斑块稳定性下降的机制目前尚不清楚。部分研究表明,5-LO及其代谢产物LTs可增强基质金属蛋白酶2和基质金属蛋白酶9在巨噬细胞中的表达,促进基质降解,导致斑块易损[12-14]。Chakrabarti等[15]发现5-脂氧合酶通路在激活大鼠内脏脂肪组织同时也上调了单核细胞趋化蛋白-1在脂肪细胞的表达,Vidal等[16]发现5-脂氧合酶抑制剂能够减少兔血管损伤模型的内/中膜比例,减轻巨噬细胞浸润,降低病变血管的核转录因子-κB(NF-κB)活性及单核细胞趋化蛋白-1的基因表达。以上研究提示5-脂氧合酶通路致斑块易损的机制可能与基质金属蛋白酶、NF-κB和MCP-1有关,具体机制有待进一步研究明确。

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