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心肌标志物在评估特发性炎症性肌病心脏损伤中的价值

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【摘要】 心脏损伤作为特发性炎症性肌病的严重并发症,其检出率不断增加,早期诊断和干预有助于改善患者的临床结局。心肌标志物(肌钙蛋白和传统的肌酸激酶)作为一种便捷和无创的心肌损伤敏感的生物学指标,在评估特发性炎症性肌病患者心脏损伤中的价值尚不明确。现就心肌标志物在特发性炎症性肌病评估中的作用做一综述。

【关键词】 心肌标志物;特发性炎症性肌病;心脏受累
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Value of Myocardial Markers in Evaluating Cardiac Injury in Idiopathic Inflammatory Myopathies

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【**Abstract**】Cardiac injury is a serious complication of idiopathic inflammatory myopathies (IIM) and its detection rate continues to increase. Early diagnosis and intervention can help improve the clinical outcome of patients. Cardiac markers (troponin and traditional creatine kinase) are convenient, sensitive and non-invasive indicators of myocardial damage, however, the value of assessing cardiac injury in patients with IIM remains unclear. This article will review the role of myocardial markers in the evaluation of cardiac involvement in IIM.

【**Key words**】Myocardial markers; Idiopathic inflammatory myopathies; Cardiac injury

特发性炎症性肌病(idiopathic inflammatory myopathies, IIM)是一组以肌肉慢性炎症为特征的系统性自身免疫性疾病,包括多发性肌炎、皮肌炎、包涵体肌炎、非特导性肌炎和免疫介导的坏死性肌病[1-2]。Oppenheim[3]于 1899 年首次报道多发性肌炎和皮肌炎中的心脏损伤病例后,随后逐渐认识到心脏损伤在 IIM 中常见,且预后不良[4-5]。2010 年公布的《多发性肌炎和皮肌炎诊断及治疗指南》[6]显示多发性肌炎/皮肌炎中心脏损伤的发生率为 6% ~75%,已成为 IIM 患者死亡的最常见原因之一[7-8]。

在 IIM 中,引起心脏损伤的病理机制主要包括心肌炎、心脏传导系统的炎症浸润、心肌细胞和传导系统的纤维化,以及通过组织病理学证实的供应心肌的小血管炎症甚至冠状动脉病变[4,9-10]。心脏损伤的表现通常包括心肌炎、心包炎、心肌病、充血性心力衰竭、心律失常、心脏性猝死、心绞痛,或仅仅是心脏生物标志物与心脏影像学的异常[11],最常见的心脏并发症是充血性心力衰竭。大多数患者仅表现为心电图轻度异常的亚临床症状,如心脏传导阻滞、PR 间期延长、Q 波和非特异性 ST-T 波的变化。多发性肌炎患者最常见的心电图改变是左前束支传导阻滞和右束支传导阻滞[5]。这些临床或亚临床的心脏受累表现可能由于原本自身的心脏疾病引起,也可能继发于 IIM 免疫介导所致,也可能上述两者情况同时存在,如何区分目前不十分明确。心脏损伤客观的诊断与评估,尤其在亚临床表现的心脏损伤患者中是困难的,目前缺乏可靠的实验室指标,心肌标志物作为有效和便捷的血清学生物标志物在 IIM 心脏损伤筛查评估中可能发挥作用,现就心肌标志物在 IIM 评估中的作用做一综述。

1 心肌标志物

心肌标志物主要包括心肌酶谱和心肌肌钙蛋白,是临床上用于诊断急性心肌梗死的重要依据。2000 年,欧洲心脏病学会和美国心脏病学会(ESC/ACC)发布生物标志物是诊断急性心肌梗死的基本标准[12-13],心肌肌钙蛋白取代心肌型肌酸激酶同工酶(MB isoenzyme of creatine kinase, CK-MB),成为检测心脏损伤更敏感的生物标志物[14]。

肌钙蛋白复合物位于收缩装置的细丝系统上,由三个亚基组成:肌钙蛋白 T(TnT)、肌钙蛋白 I(TnI)和肌钙蛋白 C(TnC)。TnT 和 TnI 各以三种不同的亚型存在:快反应骨骼肌型、慢反应骨骼肌型和心肌型。这三种亚型由不同的基因编码,可通过免疫学技术加以区分。心肌肌钙蛋白 I(cardiac troponin I, cTnI)在胚胎或胎儿发育过程中不表达,仅在成人心肌组织中检测到[15]。心肌肌钙蛋白 T(cardiac troponin T, cTnT)在发育中的胎儿[16]、健康成人和再生的成人骨骼肌组织(包括 IIM 患者)中都可检测到。TnC 只有两个基因编码的亚型:一个特定于快反应的骨骼肌,另一个在心肌和慢反应的骨骼肌中共同表达,TnC 不是心脏特异性的检测,不能作为心肌损伤的实验室诊断[17]。

2 心肌标志物与 IIM

2.1 肌酸激酶

肌酸激酶(creatine kinase, CK)是一种用来携带和释放能量的酶,存在于身体的各种组织中。它有 3 种不同的同工酶:CK-BB(脑)、CK-MM(骨骼肌)和 CK-MB(心肌)。CK-MB 在骨骼肌和心肌中的含量分别为 l% ~2% 和 15% ~25%, IIM 由于持续的肌肉破坏和再生,CK 持续增加[18],随着骨骼肌开始再生,胚胎基因的重新表达,新骨骼肌中的 CK-MB 有所增加。相关研究发现 CK 和 CK-MB 的升高与反复骨骼肌再生存在显著相关性,且 CK 与 cTnT、CK-MB 和 cTnT 有很强的相关性[19]。通常用 CK-MB/总 CK 比值 >3% 来判断是否心肌受累,但由于骨骼肌病变再生也可导致 CK-MB 水平升高,因此 CK-MB/总 CK 比值 >3% 反映心肌受累的特异性仍有争议[20]。而且,多发性肌炎/皮肌炎中的心脏受累形式多样,即使临床有明确心脏损害时,CK-MB 值也可在正常范围[21]。

2.2 肌钙蛋白

骨骼肌特定肌钙蛋白组有:sTnT、sTnI 和 sTnC,当骨骼肌病变时会释放;心肌肌钙蛋白组有:cTnT、cTnI 和 cTnC,当心肌损伤发生时会释放[22]。41% ~78% IIM 患者 cTnT 升高,而 cTnI 升高患者只有 2% ~2.5%[19,23-24]。在 IIM 患者中血清 cTnI 和 cTnT 水平可帮助鉴别心脏损伤,但 cTnT 可能缺乏特异性,在无心脏损伤的患者中常常也会升高[25]。目前 cTnT 的来源仍存在争议,有研究表明 IIM 患者血清升高的 cTnT 与急性冠脉综合征患者的 cTnT 性质相同,来源于心脏[26]。也有研究显示外周血中 cTnT 水平升高可能来源于心肌损伤,也可能来源于骨骼肌再生,在骨骼肌疾病中 cTnT 升高除了存在真正的心脏受累,还可能

存在隐匿的心血管疾病,也可能是骨骼肌病变引起骨骼肌重新表达,cTnT 形式[27],用于检测 cTnT 的抗体与 TnT 的其他亚型存在交叉反应,这些蛋白质水平的升高也常会被误解为 cTnT。怀疑有心肌损害的患者,cTnT 及 CK-MB 的升高显示有心肌受损可能[22]。无心脏受累的 IIM 患者中,cTnT 和 CK-MB 的升高也很常见[23,28-29],但 cTnT 水平与 IIM 疾病活动密切相关。

外周血中检测到的 cTnI 仅来源于心肌,且 cTnI 含量比 CK-MB 大,对心肌损伤有较高的敏感性和特异性[20],能检测心肌微小的损伤,因此 cTnI 是检测 IIM 患者心肌细胞损伤更具特异性的生物标志物。但 cTnI 升高也不能完全确定心脏是否受累,因为在其他情况下 cTnI 也会升高,如:甲状腺功能亢进、肾功能不全、脓毒症及危重症严重急性神经系统疾病(如卒中、蛛网膜下腔出血)等[30]。

2.3 新型心肌标志物

尽管在临床中未被常规应用,现新一代的生物标志物越来越受到关注。半乳凝集素-3(galectin-3,Gal-3),是一个 β-半乳糖苷结合凝集素家族成员[31],由活化的巨噬细胞分泌,可诱导心脏成纤维细胞增殖、胶原沉积和心室功能障碍[32]。Gal-3 是一种具有免疫调节作用的多功能蛋白,参与细胞凋亡、细胞活化、分化和迁移等生物过程[33],目前作为一种反映心力衰竭时心脏炎症、纤维化和心脏重构的新型生物标志物[34]。Gal-3 在心肌纤维化的发生和发展中起重要作用。在一般人群中,更高的血浆浓度与全因死亡率和心力衰竭风险相关[35-36]。

在多发性肌炎/皮肌炎患者血清中,抗 Gal-3 抗体水平升高,并且高于其他自身免疫性疾病[37],提示在 IIM 患者中 Gal-3 可用来评估心脏受累情况,但 Gal-3 在多个器官中表达,任何器官损伤都能影响血浆中 Gal-3 的水平,所以循环中的 Gal-3 水平能否可靠地反映心肌损伤尚待进一步研究。

3 基于生物学标志物筛查炎症性肌病心脏受累策略

由于大部分 IIM 患者缺乏临床相关的心脏表现,尽管无症状,心脏受累也可能存在,因此需推荐一些便捷的方法来筛查心脏损伤。TnT 可作为严重疾病的预后指标[26],但鉴别心脏酶的升高是由于炎症本身还是心脏受累,仍需鉴定 TnI[38]。目前尚无公认的 IIM 相关心脏受累的临床评估策略,现推荐两种筛选策略:(1)对没有已知心脏疾病的无症状患者,疑似亚临床心脏受累,先行 cTnT 筛查,然后行验证性 cTnI 测量,当 cTnI 阳性时,提示患者可能有心脏受累[39]。(2)建议所有 IIM 患者均应进行 cTnI 检测(首选高敏感性分析),再结合心电图和超声心动图评估,随后根据这些评估中的异常结果,排查其他非心脏性的常见原因。如果 cTnI 正常,且在心电图和超声心动图评估中无异常表现,则患者有心脏受累的可能性极低。如果 cTnI 正常,但在心电图和超声心动图上有异常表现,则需考虑进一步评估心脏磁共振和/或心内膜活检。如果 cTnI 高于正常值上限,但有可解释 cTnI 升高的其他情况,心脏受累可能性较小,进一步的评估仍需根据情况选择进行。如无其他解释,则心脏受累可能性大,须进行下一步评估[40]。

4 结论

总之,心肌标志物是一种广泛而廉价的检测心肌损伤的生物学指标,具有较高的敏感性和特异性。在心肌损伤的评估中,cTnI 比 cTnT 和 CK-MB 的特异性更高,所以优先选择 cTnI 来筛选 IIM 患者的心脏损伤情况,同时结合其他评估心脏受累的辅助检查,如心电图、超声心动图、心脏磁共振和心内膜活检等,有助于对 IIM 患者中的心脏受累的早期诊断和评估,并改善 IIM 患者的预后。

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斑块侵蚀发病机制的最新研究进展

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【摘要】斑块侵蚀是导致急性冠脉综合征的另一重要原因,目前针对斑块侵蚀病变的发生和发展过程尚不清楚。血流紊乱会激活内皮细胞 Toll 样受体 2,介导内皮细胞凋亡、剥脱,作为斑块侵蚀病变发生的启动因素。透明质酸和多功能蛋白聚糖代谢障碍、细胞外基质发生重构,在斑块侵蚀病变进程中发挥核心作用。中性粒细胞形成胞外诱捕网,释放细胞活性成分,进一步损伤内皮细胞,促进血栓形成。现主要从以上三方面对斑块侵蚀病变形成的可能机制及假说进行综述。

【关键词】急性冠脉综合征;斑块侵蚀;血流紊乱;透明质酸;中性粒细胞胞外诱捕网

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