

血管内超声诊断左主干严重痉挛1例及文献复习

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收稿日期: 2022年1月14日; 录用日期: 2022年2月8日; 发布日期: 2022年2月17日

摘要

冠状动脉痉挛是血管痉挛性心绞痛的发病机制, 左主干痉挛是冠状动脉痉挛中比较罕见的一种, 目前文献中较少报道。本文报告一例经血管内超声检查确诊为左主干痉挛的患者, 最后行左主干支架植入术治疗。血管内超声(IVUS)可有助于鉴别血管痉挛性心绞痛和非血管痉挛性心绞痛, 识别及诊断左主干痉挛, 为患者制定治疗方案提供依据。

关键词

左主干痉挛, 血管造影, 血管内超声

Intravascular Ultrasound Diagnosis of Severe Left Main Coronary Artery Spasm: A Case Report and Literature Review

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Received: Jan. 14th, 2022; accepted: Feb. 8th, 2022; published: Feb. 17th, 2022

Abstract

Coronary artery spasm is the pathogenesis of vasospasm angina pectoris. Left main coronary artery spasm is a relatively rare type of coronary artery spasm, which is rarely reported in the literature. This article reports a case of a patient who was diagnosed with spasm of the left main co-

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ronary artery by intravascular ultrasound, and was finally treated with intracoronary stenting. Intravascular ultrasound (IVUS) can help differentiate vasospasm angina and non-vasospasm angina, identify and diagnose left main coronary spasm, providing evidence for treatment plans.

Keywords

Left Main Coronary Artery Spasm, Angiography, Intravascular Ultrasound

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1. 病例汇报

经皮冠状动脉造影(CAG)中左主干痉挛是比较少见的现象,是导管刺激所致还是自发痉挛,与临床现象有无关系,目前还有较多的争论。本文报告一例反复胸闷、大汗、心电图正常的患者,冠状动脉造影发现严重的左主干狭窄,经血管内超声检查确诊为冠状动脉痉挛。现报告如下:

患者男性,50岁。有20余年饮酒史及“高血压病”家族史,无吸烟、高血脂、高血糖等冠心病危险因素。2005年7月因“发作性胸闷、胸痛10天”入院,初始症状发作多与劳累有关,持续时间10分钟左右,含服“速效救心丸”效果不明显,发作时心电图无缺血性改变,运动平板试验阴性,超声心动图提示左室舒张功能减退。入院前症状发作较频繁(间隔时间约30分钟左右),伴心悸、出汗、濒死感,活动或静息时均发作。入院以后动态观察心电图、心肌酶均无异常发现,症状仍反复发作,严格按不稳定型心绞痛治疗效果不佳,遂行冠脉造影检查。经右侧股动脉造影, JL4 造影导管造影时见左主干中远段向心性狭窄90%,其它冠状动脉管壁光滑,未见明显狭窄。但当时患者无任何症状,心电图正常。冠脉内注射硝酸甘油100 μg后复查造影见狭窄已减轻,残余狭窄约50%。即行血管内超声(IVUS)检查。6FXB指引导管到位后, BMW导丝穿过狭窄,经导丝插入血管内超声导管(波士顿科学公司),显示左主干全程“三层结构”清晰可见,内膜轻度增厚(0.3 mm),远段及近段血管内径分别为4.5 mm及4.8 mm,管腔面积分别为6.0 mm²及6.3 mm²,中段狭窄最重处可见轻度偏心性纤维斑块(斑块最大厚度0.6 mm,面积狭窄率23.6%)。此时患者出现明显胸痛,造影剂“冒烟”见左主干严重狭窄,即刻冠状动脉内注射硝酸甘油200 μg,仍决定行左主干支架植入术,迅速用Maverick 2.5 × 20球囊扩张,后在左主干植入TAXUS 5.0 × 16 mm支架,复查IVUS见支架膨胀及贴壁好(见图1),无并发症。

2. 讨论

冠状动脉痉挛是指在心脏表面走行的相对较大的冠状动脉短暂出现异常收缩的情况。目前被广泛认可的是,吸烟是冠状动脉痉挛的主要危险因素,其余的危险因素包括了血脂异常、糖尿病和糖耐量异常。冠状动脉痉挛是变异型心绞痛的重要发病机制,其最早于1959年由Prinzmetal首先提出。除了导致心绞痛,冠状动脉痉挛同时也可引起心肌梗死、心律失常等心血管疾病[1] [2] [3],因此冠状动脉痉挛在血管造影中逐渐受到关注与研究。冠状动脉痉挛在血管造影中并不罕见,其中以右冠状动脉痉挛最为多见[4],而左主干痉挛在文献中虽偶有报道,但在造影中极为罕见,尤其是本例患者左主干痉挛造影显示为严重狭窄。

目前诊断冠状动脉痉挛的辅助检查主要包括了心电图、血管造影、冠状动脉痉挛激发试验、血管内超声(IVUS)及光学相干断层成像(OCT)。冠状动脉痉挛性心绞痛发作时典型的心电图改变包括病变部位对

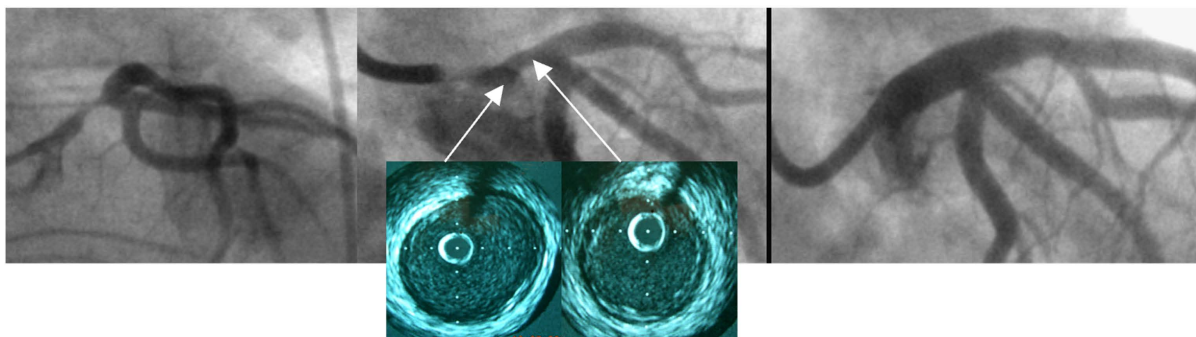


Figure 1. Angiography: Left: left main stenosis (before nitroglycerin); Middle: the stenosis of the left main trunk was reduced (after nitroglycerin); Right: after left main stent implantation. IVUS: Left: no stenosis and no obvious plaque on the left trunk; Right: the most severe stenosis of the left trunk, intimal thickening

图 1. 造影: 左: 左主干狭窄(使用硝酸甘油前); 中: 左主干狭窄减轻(使用硝酸甘油后); 造影右: 左主干支架植入术后。IVUS: 左: 左主干无狭窄处, 未见明显斑块; 右: 左主干狭窄最重处, 内膜增厚

应的导联 ST 段抬高, 对侧导联 ST 段压低, 在服用速效硝酸盐类药物后恢复正常。但部分血管痉挛性心绞痛患者发作时, 心电图可无明显变化[5]。在既往的报道中, 左主干痉挛发生的原因被认为可能是自发的[6]或者是导管诱导的[7]。血管造影导管诱导的冠脉痉挛的一般具有顽固性和持续性的特点, 对重复的大剂量的冠状动脉内血管扩张剂治疗反应并不敏感。而左主干痉挛往往也对硝酸甘油反应不敏感, 甚至表现为硝酸甘油抵抗, 这为明确左主干痉挛的诊断及分析其发生原因造成了困难。有报道一例左主干狭窄患者半小时内使用硝酸甘油 600 μg 进行药物治疗[8]。但尽管如此, 左主干痉挛仍持续存在, 这使诊断和治疗进入了两难的境地, 使最终治疗方案的选择变得困难。一周后, 患者拟择期介入治疗再次行冠状动脉造影时发现狭窄消失, 此时才诊断为冠状动脉痉挛。如果缺乏第二次冠状动脉造影检查, 往往会诊断为左主干动脉粥样硬化性狭窄。因此, 仅仅依靠单一冠状动脉造影及硝酸甘油后造影诊断左主干痉挛并不是非常可靠的方法。而在未确定冠脉痉挛的情况下, 对血流动力学不稳定的患者立即使用硝酸甘油可能会带来低血压等不良反应[9]。而药物诱导的冠状动脉痉挛激发试验, 是通过向冠状动脉内注射乙酰胆碱或麦角新碱[10]来进行的。有研究曾对 1185 名患者通过麦角新碱诱发冠脉痉挛[4], 结果发现麦角新碱激发试验与乙酰胆碱激发试验的阳性率无明显差异, 其中未发现发生左主干痉挛患者。由于药物诱导的冠状动脉痉挛激发试验尤其在多支血管中进行时, 可能导致包括心绞痛、呼吸困难、呕吐和心律失常等并发症, 因此不适用于有左主干严重病变征象的患者[11]。因此, 其对于诊断左主干痉挛仍有待进一步的研究。

目前, IVUS 及 OCT 可为诊断冠状动脉痉挛提供重要线索。IVUS 是第一种广泛应用的导管成像技术, 其可提供关于血管的狭窄严重程度、病变长度、血管大小和斑块特征的有价值的信息[12]。OCT 是一种基于近红外光的发射和反射的光学技术, 与超声相比, OCT 的分辨率大约高 10 倍[13]。但是, 更高的分辨率(轴向 10 至 15 μm , 横向 20 至 25 μm)是以较差的穿透血液和组织(1 至 3 mm)为代价的[14], 因此在主动脉开口处及左主干病变应用上有一定限制[15]。相比之下, IVUS 图像采集则不需要注射造影剂进行血液清除, IVUS 更适合于评估较大的血管, 如主动脉口和左主干[16] [17]。因此, 有专家提出使用 IVUS 评估左主干病变的重要性, 并采用 6.0 mm^2 的最小管腔面积(MLA)临界值来评估是否需要进一步介入治疗[13]。分析左主干痉挛发生的原因应紧密结合临床特征, 但多数与左主干本身有轻度狭窄有关。通过 IVUS 研究发现, 局灶性血管痉挛在 IVUS 中主要表现为低回声、钙化较少而胶原纤维较多的斑块[11] [18], 甚至痉挛局部可无钙化斑块, 血管痉挛与病变的严重程度及累及范围无关[19]。而严重冠脉痉挛则可能与弥漫性内膜增厚有关[1]。本例患者行 IVUS 后可见左主干存在轻度的内膜增生和纤维斑块, 无钙化, 结

合患者的临床表现, 考虑左主干管腔的改变是导致左主干痉挛的原因, 且自发痉挛的可能性大。

冠状动脉痉挛的治疗主要包括了日常生活管理、药物治疗和手术治疗。治疗冠状动脉痉挛的药物主要包括硝酸盐类药物和钙通道阻滞剂[20] [21] [22]。 β -受体阻滞剂的使用可能导致 α -肾上腺素能刺激和冠状动脉血管张力的增加, 因此 β -受体阻滞剂在冠状动脉痉挛中不建议单独使用, 可联合硝酸盐类药物和钙通道阻滞剂用于冠状动脉明显狭窄的血管痉挛性心绞痛患者。手术治疗包括了冠状动脉旁路移植术(CABG)和介入治疗。CABG术后的复发性心绞痛和移植血管血栓是其主要限制[23], CABG术后仍出现左主干痉挛也曾有报道[24], 因此CABG在左主干痉挛中的应用有待进一步研究。介入治疗可应用于存在伴严重器质性狭窄的血管痉挛性心绞痛的患者[21]。有研究发现IVUS指导的药物洗脱支架植入后12个月避免支架血栓形成的重要预测因素[25], 可以降低术后左主干狭窄的长期死亡率[26]。本例患者临床表现为典型心绞痛, 发作时心电图未见明显改变。由于存在反复胸闷、心绞痛, 造影中可见左主干痉挛引起严重的狭窄。行IVUS后可见左主干存在轻度的内膜增生和纤维斑块而无钙化, 考虑为左主干自发性痉挛。基于患者左主干痉挛存在严重狭窄, 结合IVUS检查结果, 我们最终制定了支架植入的治疗方案。

3. 结论

尽管左主干痉挛与临床表现的关系尚不确定, 但是由于其硝酸甘油抵抗性以及持续时间长等特点, 我们认为应引起临床高度重视, 严重左主干痉挛可以引起猝死等严重后果。本例报告的意义在于: 严重左主干狭窄使用硝酸甘油后造影复查很容易将左主干痉挛误诊为粥样硬化性狭窄, 此时IVUS成像可帮助显示斑块负荷, 有助于左主干痉挛的识别, 减少误诊、漏诊, 同时指导制定进一步的治疗方案; 严重反复发作的左主干痉挛, 仍建议支架治疗。

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