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冠脉内超声诊断冠脉造影固定性狭窄的心肌桥1例并文献复习

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摘　要

心肌桥(myocardial bridge, MB)的形成是由于冠状动脉走行于心肌之间的一种先天性解剖变异。资料显示，心肌桥在尸检病例中检出率高达40%~80%，但冠脉造影发现率仅为0.5%~12%。近年来随着冠脉内超声(IVUS)的发展，对于心肌桥的诊断准确率大大提升。本文报导一例因“发作性胸闷、胸痛1年，加重2天”疑诊“冠心病、不稳定性心绞痛”患者行冠脉造影，可见前降支中段固定性狭窄，管腔内径不随心动周期发生明显变化，并且与投照体位无关，随后行IVUS检查时，发现为典型的心肌桥改变，并及时调整治疗方案为例，提高对冠脉造影下表现为固定性狭窄性病变的诊治思路以及强调IVUS在介入领域的独特优势，指导治疗。

关键词

血管内超声(IVUS)，心肌桥，冠脉造影，固定性狭窄

Diagnosis of Myocardial Bridge in Coronary Angiography Fixed Stenosis by Intracoronary Ultrasound: A Case Report and Literature Review

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Abstract

Myocardial bridge (MB) is a congenital anatomical variation of coronary artery which runs along the myocardium. The data showed that the detection rate of myocardial bridge in autopsy cases was as high as 40%~80%, but the detection rate of coronary angiography was only 0.5%~12%. In recent years, with the development of intracoronary ultrasound (IVUS), the diagnostic accuracy of myocardial bridge has been greatly improved. This paper reports a patient who suspected of "coronary heart disease and unstable angina pectoris" due to "paroxysmal chest tightness and chest pain for 1 year and aggravated for 2 days". Coronary angiography showed fixed stenosis of the anterior descending artery. The lumen diameter did not change significantly in cardiac cycle, and had nothing to do with the projection position. Later, we used IVUS for further examination and we found that it is typical myocardial bridge and timely adjusted the treatment. We aim to improve the diagnosis and treatment of fixed stenotic lesions under coronary angiography, emphasize the unique advantages of IVUS in the field of intervention, and guide the treatment.

Keywords

Intracoronary Ultrasound (IVUS), Myocardial Bridge, Coronary Angiography, Fixed Stenosis

1. 引言

冠状动脉造影的心肌桥发现率仅为 0.5%~12% [1]。严重的心肌桥可以引起典型的心绞痛发作，甚至心肌梗死和心源性猝死。冠脉造影时血管内径随心动周期变化是心肌桥的特征性改变，但近来我们对 1 例冠脉造影表现前降支中段固定性狭窄的患者行冠脉内超声(IVUS)检查时，发现为典型的心肌桥改变。现报告如下：

患者男性，40 岁。因“发作性胸闷、胸痛 1 年，加重 2 天”疑诊“冠心病、不稳定性心绞痛”收住入院。患者症状发作与情绪激动、劳累有关，持续 1~2 分钟，休息后可自行缓解。既往吸烟 20 年，40 支/天，长期饮酒，无其它冠心病危险因素。心电图表现“II、III、avF 病理性 Q 波”。冠状动脉造影见前降支中段可见长约 1.5 cm 的 80%狭窄病变，管腔内径不随心动周期发生明显变化，并且与投照体位无关。即行 IVUS 检查，见狭窄远端(见图 1(d))血管内膜光滑，管腔内径 2.85 mm，狭窄处(见图 1(b)、图 1(c))血管内径于心脏收缩期明显变细(直径：2.3 mm；面积：4.0 mm^2)，舒张期增粗(直径：2.8 mm；面积：9.4 mm^2)，面积增大率为 56.5%，但血管内膜光滑，收缩期可见“半月征”，狭窄近端(见图 1(a))血管相对扩张(直径：5.0 mm)，可见管壁“三层结构”，内膜轻度增生。冠脉内注射硝酸甘油 200 μg 后造影仍表现固定性狭窄，但 IVUS 上述征象更加明显，面积变化率达 57.9%。根据 IVUS 的特征性表现，诊断前降支中段“心肌桥”，伴冠脉轻度粥样硬化。

2. 讨论

通常认为心肌桥是心脏中良性且常见的解剖变异，尸体解剖检出率可达 40%~80% [2]。1960 年 Porstmann 和 Iwig 首次在血管造影上描述了这一解剖异常的现象[3]。据估计，成人中心肌桥患病率约为

25% [1]，但心肌桥在冠脉造影术中发现率仅为 0.5%~12%，说明至少有一半以上心肌桥未被准确诊断。由于心肌桥走形于心肌纤维下方，因此也称为隧道动脉(tunneled artery)，前降支中段是心肌桥的高发部位，其次为回旋支、右冠状动脉[4]。大多数患者无明显临床症状，有症状的心肌桥患者多表现为劳力性心绞痛和(或)呼吸困难，严重者可引起急性冠状动脉综合征[2]。其他少见的临床表现包括房室传导阻滞[5]、应激性心肌病(Takotsubo 综合征) [6]、快速型室性心律失常[7]、晕厥甚至猝死[8] [9]。心肌桥在肥厚型心肌病中很常见，发病率可达 25% [10]，它是年轻人和运动员心源性猝死的重要潜在病因之一[10] [11]。虽然心肌桥与多种心脏不良事件有关，但其预后良好，症状和预后与肌桥狭窄程度无关[12]。严重的心肌桥临床表现与动脉粥样硬化性心脏病(冠心病)导致的固定狭窄相一致，因此，很难从临床表现方面鉴别二者。然而，同样是导致冠脉狭窄的两种疾病，其药物治疗和介入治疗却有着非常大的区别。对于有冠脉内血流动力学改变的伴缺血性体征的心肌桥患者，低剂量 β 受体阻滞剂及钙通道阻滞剂是目前首选的药物治疗方案，目的是减慢心率，降低心肌收缩力，改善舒张期血流及减少心肌收缩期的压迫[8]。需要注意的是，单独使用 β 受体阻滞剂可能会加重原本已有心肌缺血症状的心肌桥患者和冠状动脉痉挛患者的胸部症状[9]，因此对部分病人可能需联合药物治疗。减慢心率的药物如窦房结 If 电流选择性抑制剂伊伐布雷定可考虑替代或联合上述药物改善心肌桥患者的症状[8]。对伴有动脉粥样硬化的心肌桥患者需行调脂稳定斑块、抗血小板等治疗。药物难以缓解症状的患者，非药物治疗包括经皮支架植入及外科冠状动脉旁路移植术(CABG)和去顶肌切除术(unroofing) [8]。与冠心病患者支架植入改善狭窄血管血供不同，对于心肌桥患者，经皮支架植入的选择应十分谨慎。据报道，心肌桥段支架植入与冠状动脉穿孔(约为 6.3%) [13]、支架断裂及短期再狭窄率(其中裸支架及药物洗脱支架 1 年狭窄率分别为 75%和 25%)有关[8] [14]。光学相干断层扫描(OCT)和 IVUS 估心肌桥段发现隧道动脉比临近冠脉血管管腔更小，管壁更薄[15]。为了对抗心肌桥的挤压，进行支架植入时往往需要较高的压力获得支架的理想扩张状态，因此可能导致管壁菲薄的冠脉穿孔；另一方面，支架植入后可出现斑块突出或斑块脱落，可引起局部炎症和血栓形成的增加，

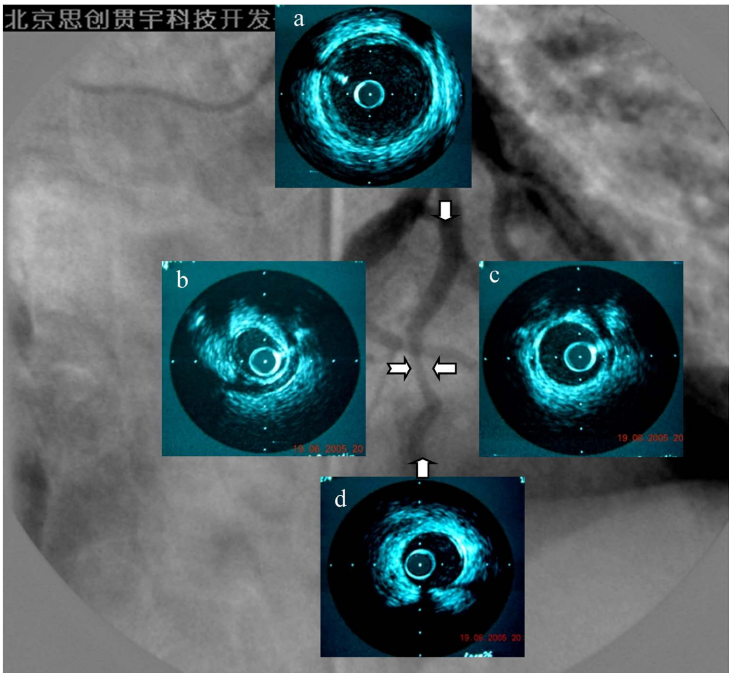

Figure 1. Fixed stenosis of the middle segment of the anterior descending branch. (a)~(d) are IVUS images of fixed stenosis and its proximal and distal ends
图 1. 冠脉造影前降支中段固定狭窄。(a)~(d)为固定性狭窄处及其近端、远端 IVUS 图像

发生急性支架血栓形成，从而导致支架失效[16]。因此对于心肌桥介入治疗的安全性和有效性仍然是未知且可能有潜在风险的。

心肌桥的冠状动脉造影典型表现为“挤牛奶效应(milking effects)”，即冠状动脉节段收缩期受压，表现为心脏收缩期血管明显狭窄变细，舒张末期(收缩早期)恢复正常。冠脉内注射硝酸甘油可使收缩期受压现象更加明显以利于发现心肌桥的存在[2] [8]。对于仅有轻度压迫或位置表浅的心肌桥冠脉造影术缺乏敏感性，常规血管造影中通过视觉评估仅能识别出收缩压缩超过 20%的节段[17]，因而导致了对心肌桥检出率的巨大差异[18]。除此之外，当心肌桥位于冠脉远端或走行于心肌内节段较短或合并肌桥近端狭窄以及左心室射血分数较低时，心肌桥均不容易在常规冠脉造影术中被发现[19]。冠状动脉造影仍然是目前侵入性检查中评估心肌桥最常用手段，但只能对肌桥解剖及心动周期中压迫程度进行评估，无法评估肌桥内血流动力学情况，对患者的治疗及预后指导意见有限。国外学者曾利用 IVUS 对造影已经诊断的心肌桥患者进行了研究，发现正常阶段冠脉管腔截面积的变化率为 9% ± 7%，而心肌桥节段的血管其截面积变化率为 40% ± 25%，并发现半月征(half-moon)在 IVUS 诊断心肌桥时其特异性达到 100%，表现为围绕心肌桥节段可见收缩期和舒张期呈半月形的低回声区，88%的心肌桥近端血管存在轻度内膜增生并血管扩张，认为与肌桥近端和远端存在压力阶差有关[2] [3] [8]。如图 1 造影结果显示，前降支中段狭窄约 75%~80%，管腔内径不随心动周期及冠脉内注射硝酸甘油发生明显变化，结合患者典型心绞痛表现，可能具备介入治疗指征，但经 IVUS 检查可见：图 1(b)、图 1(c)为典型“半月征”，面积变化率达 57.9%，肌桥远端及近段血管内膜光滑，并无动脉粥样硬化形成，予以 β 受体阻滞剂或钙通道阻滞剂治疗即可，避免了植入支架和抗血小板等治疗。因此，准确鉴别冠心病和心肌桥具有重要意义。

该例患者多角度投照冠脉造影检查均未提示明显的心肌桥表现，其原因尚不清楚。肌桥周围心肌及组织严重的纤维化潜在病因。有报道称[20]，当心外膜组织发生纤维化，纤维粘液样组织和肉芽肿组织侵袭至左前降支时，在冠脉造影上表现为收缩期狭窄闭塞、舒张期血流正常的“挤奶”样心肌桥表现；Bassim [21]及其同事也报道了一个类似的案例：一位疑诊急性心肌梗死的患者进行冠脉造影时，观察到左前降支中段 80%固定性狭窄且冠脉内注射 200 μg 硝酸甘油未见管腔直径的变化，由于可见狭窄段血管有明显心肌内行程，冠脉造影诊断为心肌桥后送至手术室行外科去顶肌切除术，术中可见严重纤维化组织包绕着心肌桥，并最终选择行 CABG 术进行治疗。这与我们的病例很相似。但事实上，心肌桥周围出现纤维化似乎并不罕见。Ami [22]等人对 180 颗在左前降支有心肌桥的尸体心脏进行尸检发现，136/180 例(75.6%)心肌桥上心肌出现不同程度的纤维化，在肌桥筋膜下的血管周围间隙存在脂肪组织。而隧道动脉周围间隙包含脂肪组织，实际上可以作为一个“冠状动脉垫(coronary cushion)”，在心脏收缩期抵抗心肌的压迫[23]。当局部出现炎症性疾病时，导致心肌桥周围间隙出现纤维化时，可能会造成心肌桥在冠脉造影时表现为严重的固定性狭窄，硝酸甘油难以或不能诱发收缩期受压现象加重的情况，因此与冠状动脉粥样硬化固定性狭窄难以区分。除此之外，本例患者还出现收缩期管腔呈不规则圆形，提示可能存在血管部分受压情况。考虑到心肌桥多出现在肥厚型心肌病的患者，局部心肌肥大后出现瘢痕修复导致压迫可能是其潜在病因。本例患者虽无明显心肌肥厚的表现，但据报道，心肌瘢痕修复多发生在肥大区域，可发生在无症状或轻度症状的肥厚性心肌病患者中[24]，而肌桥由于长期受心肌压迫，受其血液供应区域的心肌可能会出现心肌代偿性肥大，并随后在某些病理因素下出现心肌瘢痕修复并由此导致肌桥壁不规则压迫。

近年来多层螺旋计算机断层扫描(MSCT)、单光子发射计算机断层成像术(SPECT)和应力超声心动图等非侵入性检查手段已广泛被用于心肌桥的诊断[2]。其中冠状动脉 CTA 在确定心肌桥的长度及深度具有较好的相关性，但对于肌桥附近斑块的检出仍缺乏特异性，尤其是非钙化斑块[25]。IVUS 能直接观测及精确测量心肌桥附近的血液流速及管壁压力，直接清晰显示管腔内结构，且可进一步分析斑块组织形态

学特征，进行粥样斑块定量测量和定性分析，指导支架的合理及准确植入。本病例报告说明 IVUS 在冠脉介入领域的另一重要意义。

声　明

该病案已获得患者知情同意，签署知情同意书。

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