

Brugada Syndrome (BrS)合并ST段抬高型心肌梗死(STEMI) 1例及文献复习

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摘 要

Brugada综合征(BrS)是一种染色体显性遗传性疾病,易导致室性快速性心律失常而增加心源性猝死的风险。典型的BrS心电图表现为胸前导联V₁₋₃ST段抬高,当发生急性ST段抬高型心肌梗死(STEMI)时,两者心电图诊断会相互影响、相互干扰,早期诊断会对正确制定临床治疗方案具有重要价值。在此,我们报道1例73岁男性患者,以急性胸痛入院,其既往心电图提示Brugada综合征,此次就诊诊断为ST段抬高型心肌梗死(STEMI)。我们对该病例心电图特征及临床特征进行分析,并对相关文献做一综述。

关键词

Brugada综合征, ST段抬高型心肌梗死

A Case of Brugada Syndrome with STEMI: Case Report and Literature Review

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Abstract

Brugada syndrome (BrS) is an autosomal-dominant disease, which easily leads to ventricular tachyarrhythmia and increases the risk of sudden cardiac death. Typical BrS ECG is characterized by elevation of V₁₋₃ ST segment of precordial lead, when acute ST segment elevation myocardial infarction occurs, both ECG diagnosis will influence and interfere with each other, and early diagno-

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sis will be of great value to correctly formulate clinical treatment plan. In this article, we report a 73-year-old male patient who was admitted with acute chest pain. His previous ECG showed Brugada syndrome. This patient was diagnosed as ST segment elevation myocardial infarction. We analyzed the ECG characteristics and clinical features of this case, and reviewed the relevant literature.

Keywords

Brugada Syndrome, ST-Segment Elevation Myocardial Infraction

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1. 研究前景

Brugada 综合征(BrS)是一种遗传性心脏疾病,其特征是典型的心电图模式以及心律失常和心源性猝死(SCD)的风险增加。BrS 在诊断以及心律失常风险预测和管理方面是一个具有挑战性的实体。如今,无症状患者占新诊断 BrS 患者的大多数,由于(遗传)家庭筛查,其发病率预计将上升。然而,详细和深入的研究继续产生新的见解,包括复杂的遗传和分子基础。BrS 患者的风险分层仍然具有挑战性,尤其是无症状患者,但最近的研究表明,风险评分对于识别心律失常和 SCD 高风险患者具有潜在有用性。开发和验证包含临床和遗传因素、合并症、年龄和性别以及环境因素的模型可能有助于改进对疾病表达性和心律失常/SCD 风险的预测,并可能指导患者管理和治疗[1]。

2. 病例简介与分析

患者男性,73岁,有约30年吸烟史及“高血压病、糖尿病、高脂血症”病史2年。2021年7月21日因“间断胸痛3天,加重6小时”入院,初始症状为胸骨下段手掌范围大小的压榨样疼痛,持续十余分钟,后患者胸痛症状发作较前频繁,伴胸闷、心悸、出汗,入院6小时前出现持续胸痛。入急诊室体格检查:血压162/92 mmHg,心率92次/分。心电图(图1)示:V₁₋₅ ST段抬高0.1~0.5 mv,其中,V₁₋₃呈J点抬高,V₄₋₅为弓背向上样抬高。TnT 0.076 ug/L。初步诊断:冠心病,急性ST段抬高型心肌梗死;Brugada综合征(1型)。给予负荷剂量抗血小板药物(阿司匹林100 mg、波立维600 mg)后行急诊冠脉造影,提示左主干(LM)+3支病变(图2),介入干预梗死相关血管(IRA)前降支,术后血流TIMI3级。术后复查心电图(图3)V₁₋₆ ST段抬高较前回落,T波倒置;V₁仍呈现显著的J点抬高。追溯患者既往心电图(图4),发现2013年及2019年均表现1型BrS样心电图,其病史中无晕厥史,无家族性猝死。后予以规范的STEMI指南推荐的药物治疗(GBMT),病情平稳。术后第5天患者突发意识丧失并四肢抽搐,心电监护提示室颤,予胸外按压30s后转窦律,患者意识恢复,血流动力学逐渐稳定。复查冠脉造影排除急性支架内血栓。超声心动图显示左心室射血分数(LVEF)为43%,左室前壁节段性室壁运动障碍。继续药物治疗基础上植入体内埋置式自动除颤仪(ICD)出院。

brugada 综合征(BrS)属于遗传性原发性心律失常综合征,由brugada教授1992年首次报道[2]此类疾病,主要发生在40岁至50岁的男性,常具有遗传基础和无结构性心脏异常的共同特征,可增加心源性猝死(SCD)的风险[3],被认为可以作为解释许多过去原因不明的特发性室速或室颤的又一重要病因而受到广泛研究。

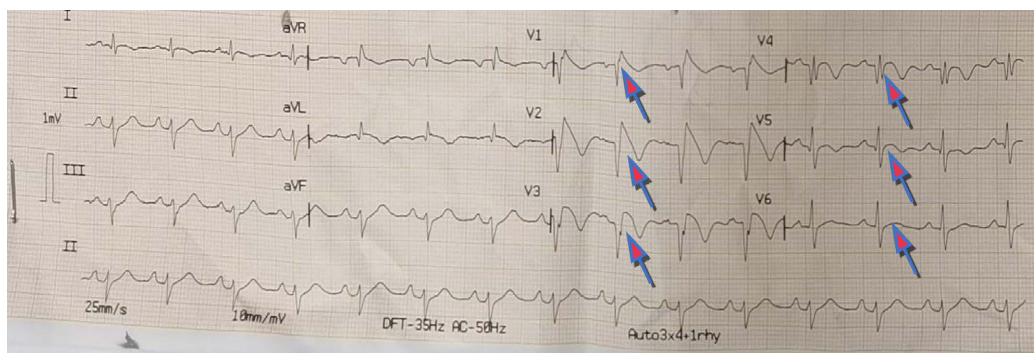


Figure 1. Emergency ECG in July 2021: V₁~V₆ ST segment elevation (as shown by the arrow), the elevation of V₁₋₃ J point is more than 2 mm, and V₃₋₆ is arcuate, with two-way or inverted T wave
图 1. 2021 年 7 月急诊心电图: V₁~V₆ ST 段抬高(箭头所示), 其中, V₁₋₃ J 点抬高>2 mm, V₃₋₆ 呈弓背样抬高, 伴 T 波双向或倒置

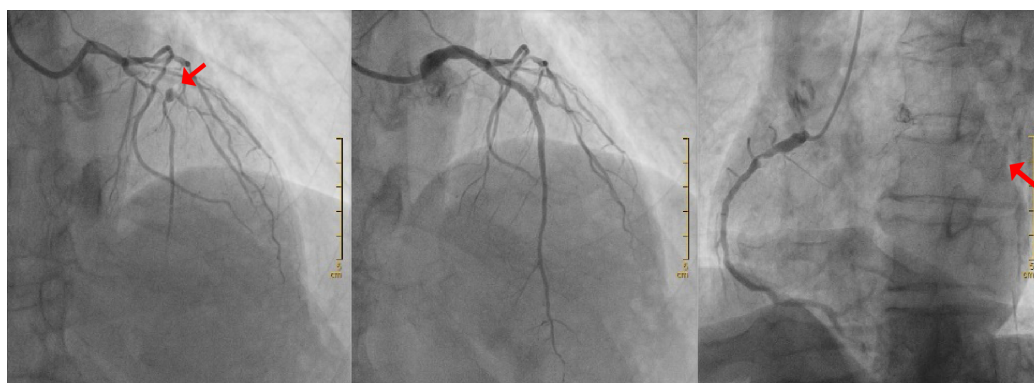


Figure 2. Coronary angiography: left: LAD (preoperative); Middle: LAD (after operation); Right: RCA (preoperative)
图 2. 冠脉造影: 左: LAD (术前); 中: LAD (术后); 右: RCA (术前)

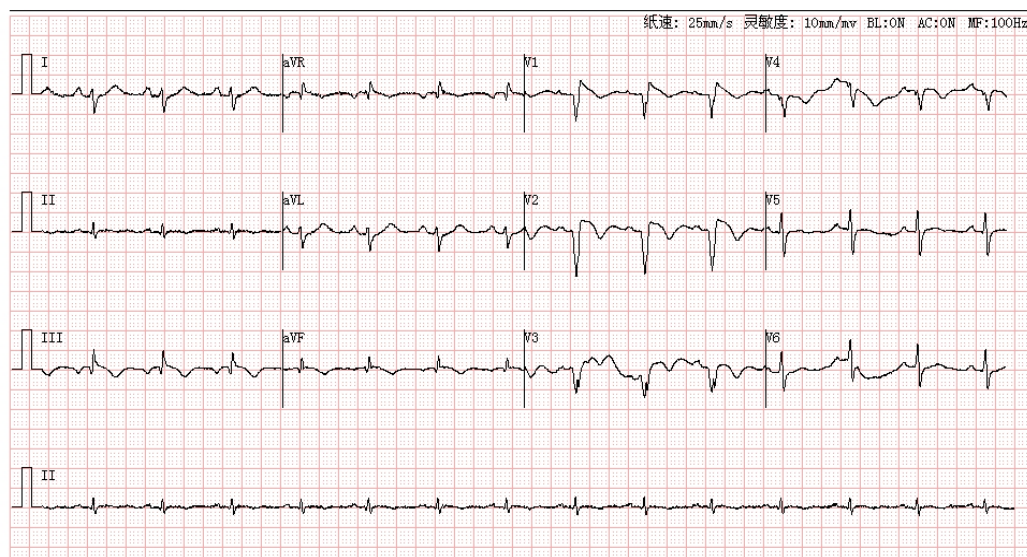


Figure 3. Postoperative ECG in July 2021: V₁₋₆ ST segment elevation fell back from the previous, and T wave inversion; V₁ still shows significant J-point elevation
图 3. 2021 年 7 月术后心电图: V₁₋₆ ST 段抬高较前回落, T 波倒置; V₁ 仍呈现显著的 J 点抬高

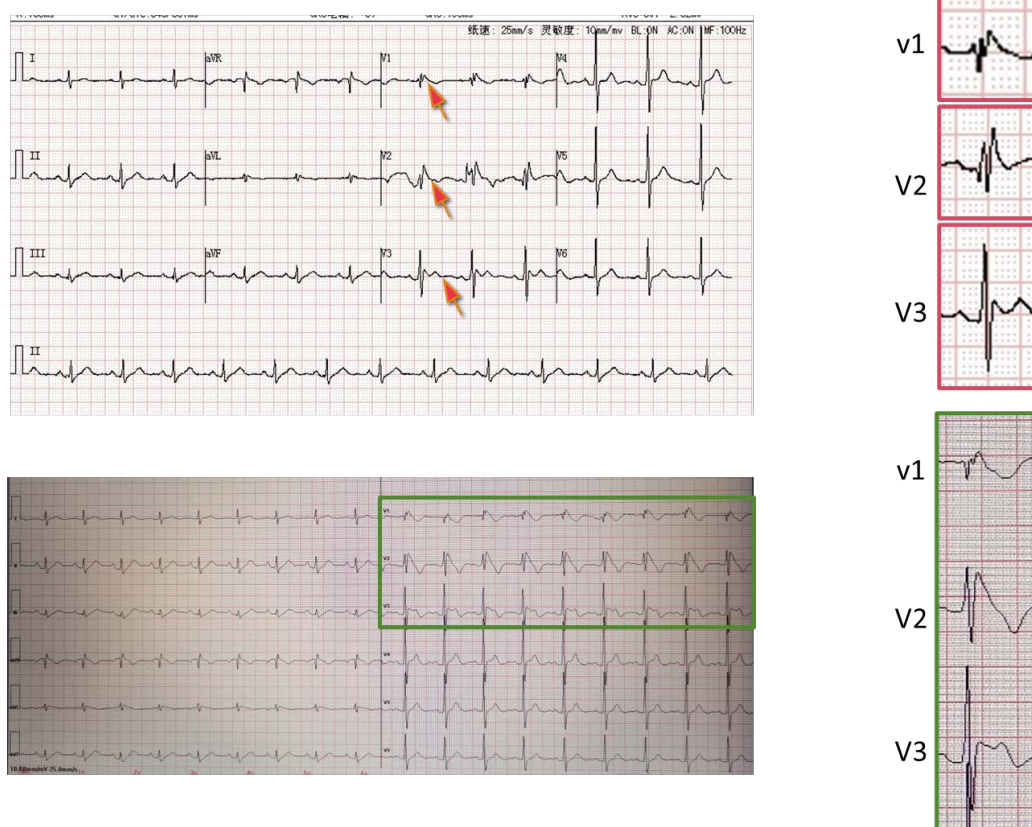


Figure 4. In the previous ECG (upper: 2013; lower: 2019), the J point of lead V₁ and V₂ was elevated (arrow), and the ST segment of lead V₃ was elevated with depression. It is a typical type I BrS-like ECG

图 4. 既往心电图(上: 2013 年; 下: 2019 年)中, V₁、V₂ 导联 J 点抬高(箭头), V₃ 导联抬高的 ST 段可见凹陷。为典型的 I 型 BrS 样心电图

目前诊断 BrS 主要依靠心电图。由于相关的去极化和复极化异常, 心电图上的胸前导联 ST 段抬高构成了 BrS 的标志[4], 其特征是右心前导联的典型 ECG 改变的短暂或持续表现[5], 具体表现为位于第二、第三或第四肋间隙的 V₁ 或 V₂ 导联 ST 抬高, 自发性或由钠通道阻断药物诱导, 无其他导致 ST 抬高的原因[6], 具有多样性、多变性、隐匿性的特点。1 型 BrS 心电图右心前导联 ST 段抬高可自发性、发热或迷走神经紧张状态或钠通道阻滞剂药物刺激后出现。只有 1 型心电图模式具有诊断性, 无论是自发的还是在药物激发后。本例患者既往 ECG 可观察到 V₁~V₃ 导联均出现前述心电图表现, 提示患者此前即有 I 型 BrS。急性 ST 段抬高型心肌梗死特征型心电图表现为相应导联 ST 段抬高, 同时具有其它急性心肌梗死的临床特征[7], 比如急性胸痛、心肌损害标志物升高等, 冠脉造影是诊断 STEMI 的金标准。本例患者存在胸痛急性加重、TnT 与 CK-MB 升高、冠脉造影提示前降支 99% 狭窄的等 STEMI 的临床表现, 急诊 ECG 提示胸前导联 V₁₋₅ ST 段抬高, 因此, 前壁 STEMI 诊断明确。对比其既往心电图, 我们发现, 此次 STEMI 时心电图表现有如下改变: 1) 之前的 V₁₋₃ J 点抬高幅度明显升高; 2) ST 段抬高的范围扩展至 V_{4,5}, 并且呈弓背样抬高, 伴 T 波双向或倒置。更加有意义的是, 我们发现该患者 II、III、avF 导联始终没有出现 ST 段压低, 典型前壁心梗的下壁镜像改变消失。另外, 再灌注治疗后心电图保持 1 型 BrS 形态是本病例的另一个特点, 尽管 J 点抬高的幅度明显下降。

文献报道显示, BrS 是可能被误诊为 ST 段抬高心肌梗死的众多疾病之一[4] [8] [9] [10], 反过来, 急性心梗也可能被误诊为 BrS [11]。事实上, 心肌缺血既可能诱发 BrS, 也可能暴露出真正的先天性 BrS,

比如 Ferrando Castagnetto 等人[12]曾经报道了一名妇女在急性前壁心肌梗死期间出现 BrS 的典型表现, Alper 等人[13]也描述了一例急性下壁心肌梗死背景下导联出现 BrP 的病例。急性缺血和 Brugada 心电图模式之间的关系及其机制目前尚不明确。因此, 鉴别 BrS 与缺血的关系, 追溯患者既往心电图具有重要的价值。文献提出, 在 BrS 基础上发生 STEMI 的心电图中, 心肌梗死部位相对导联 ST 段抬高、后伸, 同时伴有完全房室传导阻滞[14] [15]。前者在患者急诊心电图中表现较明显, 表现为 $V_1 \sim V_2$ ST 段明显凹陷, 部分掩盖了 Brugada 型心电图特征; 此时患者 ST 段抬高明显, 累及 $V_{4,5}$ 导联, 呈穹窿形改变, T 波负向。心内膜一过性外向钾电流(Ito)的存在对于全或无复极至关重要, 复极导致动作电位圆顶的损失。在缺血条件和模拟缺血成分条件下观察到的心外膜动作电位圆顶的损失几乎只发生在右心室外膜组织中, 其中 Ito 是最突出的[16], 表明心外膜动作电位圆顶的缺失可能是右心室急性心肌缺血患者 ST 段抬高的原因之一。当右冠状动脉近端也受累, 尤其是右心室流出道受累, 就会出现 brugada 样心电图表现。本例患者后续冠脉造影显示右冠全程内膜不光滑伴斑块, 近中段狭窄 80%~90% (图 2), 提示患者 brugada 样心电图可能与此相关。数据表明, Ito 介导了动作电位缺口的突出, 右心室缺血导致“真正的”ST 段抬高, 继发于心外膜动作电位圆顶的选择性降低, 复极的跨壁弥散, 以及能够加速室速/室颤的第二阶段再入。因此, Ito 调节急性缺血和 Brugada 综合征的心电图表现[17]。与 STEMI 患者的典型心电图表现相对照, 后者中 ST 段往往向上凸, 形成一个圆顶, 经常超过 QRS 的高度, 有增宽或倒置的 T 波, ST 段压低和 Q 波。如果 ST 段抬高不符合与 STEMI 相关的经典模式, 则应评估心电图改变的其他原因[18]。此外, 部分 brugada 综合征患者心电图表现在下壁导联或左胸导联 ST 段抬高, 有时还表现为 QT 间期略延长。右胸导联 QT 间期延长较左胸导联明显, 可能是右心室外膜动作电位时程延长更明显所致。本病例除了以上心电图特点, 前壁 STEMI 但未出现下壁导联镜像 ST 段压低, 文献中未见类似报道, 机制也不明确, 有待进一步观察研究。

目前 BrS 缺乏有效的药物预防 SCD 的发生, 但当合并急性心肌梗死时, 药物治疗中仍有一些特殊之处和争议。有研究显示, β 受体阻滞剂(β -blockers, BBs)和钙拮抗剂(Calcium Channel Blockers, CCBs)可增加 BrS 患者 ST 段抬高风险及心室颤动风险而建议避免使用[19] [20] [21] [22], 而 β 受体阻滞剂是 STEMI 治疗的基础药物之一。但最近又有研究表明, BrS 患者长期摄入正常剂量的 BBs 和 CCBs 与心电图参数的加重和临床结局无关, 在对 BrS 患者密切观察的前提下, 适当使用 BBs 和 CCBs 是可以接受的[23]。另外, 多项报道提及口服或静脉使用胺碘酮引起 BrS 综合征改变, 提示 BrS 患者应慎用相似抗心律失常药物[24] [25] [26]。本例患者心梗后常规使用了 BBs, 其于心梗后第 5 天发生室颤的原因除了考虑心梗后钙离子超载有关以外, 不排除与 BBs 有关, 有必要将来引起更多临床关注。无论何种机制, 按照 BrS 和 STEMI 的国际诊疗指南, 植入 ICD 是预防此类患者 SCD 的有效方法[6] [27]。

本例报告的意义在于, BrS 因其胸前导联 ST 段抬高的特点可能掩盖 STEMI 的心电图表现, 合并 STEMI 时, BrS 的特点也容易被忽略。针对这类人群, 需要结合其既往心电图以及动态观察心电图演变有助于明确诊断。STEMI 合并 BrS 时, 药物治疗避免使用 BBs 或 CCBs。ICD 植入预防 SCD 应遵循相关指南。

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