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• 病例报告 •

急性心肌梗死所致室间隔穿孔自行闭合1例

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患者,男,29岁,因持续胸闷伴恶心、呕吐4h入院。查体:神清气平,心率72次/分,律齐,未闻及杂音,血压130/90mmHg,双肺呼吸音清晰,未闻及干湿罗音,双下肢无水肿。入院当日心电图为窦性心律,V1、V2呈QS型,V1~V3导联T波高耸,心肌酶谱肌红蛋白216.55ng/ml、肌钙蛋白>50ng/ml,考虑患者为病毒性心肌炎。次日患者胸骨后仍隐痛,心超发现符合急性前间壁心肌梗死表现,心电图和心肌酶谱均有动态变化。即行冠脉造影术,发现前降支中段100%闭塞,植入支架1枚,症状缓解。术后查体未闻及心脏杂音。术后第二天(即发病第三天)再次查体可闻及胸骨左缘第四肋间全收缩期3/6级杂音,急查床边心超(床边心超未录像),符合急性前间壁心肌梗死表现。二维心超未见明显室间隔连续中断,但彩色多普勒探及室间隔肌部左向右分流,分流为湍流的频谱,其峰值为2.8m/s,未见明显室壁变薄,左室射血分数(LVEF)为46%。由于患者无明显心衰表现,且心梗急性期行手术封堵危险性较大,故给予保守治疗。1周后杂音消失,复查多普勒心超未见室间隔肌部左向右分流。3个月后再次复查心超,符合前间壁心梗表现,LVEF为58%,未见室间隔穿孔表现(见下图)。

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图1 患者3个月后复查心超图像

讨论 较小的不引起明显血流动力学紊乱的先天性室间隔缺损(VSD)有自然愈合的趋势,一般在一岁半前完全闭合或缺损变小。成年人较小肌部的VSD(如Roger病)不影响血流动力学的变化,不需要手术治疗,也不影响生活。

急性心肌梗死引起室间隔穿孔较游离壁穿孔少见,其发生率为0.5%~2%,一般发生在心肌梗死后3~7d,本例为发病第三天。室间隔的膜部由后心内膜垫纤维组织所组成,不含心肌组织,故梗死通常只累及室间隔心肌部。本例累及室间隔肌部即,证实此说。心肌梗死后数小时即可出现心室重塑,推测本例未受累及的室间隔肌部出现反应性增厚,促使穿孔部位闭合。

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