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Study of 356 cases of rheumatic valvular disease with valve replacement

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Abstract Valve replacement was performed for 356 cases of rheumatic valvular disease in our institute in the period from January 1980 to July 1995. The 356 patients consisted of 120 male and 236 female with an age range from 14 to 63 and an average of 30.58±8.49 years. Preperative NYHA classification categorized 20 cases in class Ⅰ (5.6%), 200 cases in class Ⅱ (56.2%), and 136 cases in class Ⅳ (38.2%). Mitral valve was replaced in 286 cases, aortic in 17 and both mitral and aortic in 53. 27 porcine valves and 382 mechanical ones vere employed. Associated procedures were tricuspid annuloplasty in 30 cases and thrombectomy of the left atrium in 14. The early mortality rate (within 30 days after surgery) was 7.6% (27/356). Low cardiac output, ventricular fibrillation, respiratory failure and cardiac tamponade were the main causes of death. The cases were followed cases of late death (1.8% patient-year). The causes of late death were ventricular arrhythmia, bacterial endocarditis, respiratory infection, severe bleeding due to anticoagulation and failure of porcine valves. The problems concerning the factors affecting the operative results, the second operation, the management of postoperative complications and the efficacy of anticoagulation were discussed.

Key words rheumatic valvular disease; valve replacement; mechanical valve; porcine valve

幽门螺杆菌感染与胃癌的关系

Investigation on relationship between helicobacter pylori infection and gastric carcinoma

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众多研究表明,幽门螺杆菌(Hp)感染是慢性胃炎的主要原因,但与胃癌的关系尚不清楚。本工作从临床、动物实验和细胞体外试验三个层次,探讨Hp感染在胃癌发生中的作用。

临床研究表明,Hp阳性患者慢性萎缩性胃炎和肠化生的发生率显著高于Hp阴性患者,对Hp相关性胃炎经平均4～8年的随访证实,Hp感染确可增加萎缩性胃炎和肠化生的形成与发展;Hp阳性胃粘膜细胞增殖指数、增殖细胞核抗原标记指数和AgNORs显著增加,表皮生长因子受体、C-myc和ras P^{21}蛋白表达增强,胃粘膜细胞DNA-EB荧光强度显著降低,而过氧化脂质含量显著增加,且与胃粘膜组织DNA损伤程度呈显著负相关。经治疗Hp被清除者,上述指标均明显改善,而Hp仍为阳性者,上述指标则无明显变化。上述结果提示Hp感染的胃粘膜的确具有较高的癌变易感性,Hp感染可能是胃癌发生的危险因素之一。

体外试验发现,Hp培养滤液可诱导胃粘膜细胞非程度DNA合成,导致DNA链的断裂和二级结构的变化。转化试验显示,在Hp培养滤液的作用下,胃粘膜细胞增殖加速,饱和密度增加,失去生长的接触性抑制,ras癌基因表达增高,ConA凝集试验阳性,能在软琼脂中集落生长,并有细胞排列紊乱、重叠生长、核增大、核形态不规则,细胞表面微绒毛增多等超微结构的改变,说明受Hp培养滤液作用的细胞,已获得恶性转化的部分表型特征。这一结果不仅首次证实了Hp毒性产物的体外致突致癌作用,而且为胃癌病因学和癌变机理的研究提供了一个较为理想的体外模型。

为进一步验证体外试验的结果,我们又进行了动物诱癌和促癌实验。以0.25%和0.5%两种浓度Hp培养滤液喂饲大鼠,实验第5周,0.5% Hp培养滤液组大鼠发生明显的腺体萎缩,壁细胞减少,PAS染色阳性层变薄,粘膜表面粘液含量减少,DNA合成速率加快,且随着时间的延长,上述变化逐渐加重。实验第10周,0.25% Hp培养滤液组大鼠也出现类似变化。至52周实验结束时,0.5% Hp培养滤液组大鼠腺胃粘膜肠化生和硫酸粘液阳性肠化生的发生率(57.1%,39.3%)显著高于0.25% Hp培养滤液组(27.6%,10.3%)和正常对照组(6.9%,0.0%),且有6例(17.%)出现异型增生。促癌实验表明,Hp培养滤液对MNNG诱发的大鼠实验性胃癌具有明显的促癌作用。上述结果提示,Hp的毒性产物在萎缩性胃炎、肠化生、异型增生乃至胃癌的发生中起着重要作用。

关键词 幽门螺杆菌;胃肿瘤;胃粘膜

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