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Dynamical histological observation on 1, 2-DMH induced coloectal carcinoma in rats

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Abstract The carcinoma of the large intestine induced by 1,2-DMH was studied in rats. It was found that hyperplasia of the colorectal mucosa was found in the 8th week after 1,2-DMH administration, mild or moderate dysplasia of the colorectal mucosa in the period from 10th to 15th week, and colorectal adenomas or carcinomas besides mocosal dysplasia in the period from 18th to 32nd week. Altogether, 14 adenomas, 10 carcinomas in situ and 35 invasive carcinomas were seen. These findings suggest that the histogenesis of colorectal carcinoma is a developmental process from mucosal hyperplasia, through dysplasiaor adenoma and carcinoma in situ, to invasive carcinoma, and mucosal dysplasia is an important precancerous lesion.

Key words 1,2-DMH; colorectal neoplasms; mucosal dysplasia; rat

论文摘要

定量张力对家兔正中神经急性损伤的实验研究

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本文通过建立在体神经瞬时应力和应变定量的单纯性牵拉伤动物模型,采用弦式加载法和直线式加载法,分别对游离神经、非游离神经受到急性牵拉后的力学表现,定量张力与神经的形态结构、内部微循环、传导功能变化的相互关系和各级损伤的临界点进行了研究。

实验结果表明:①牵拉伤是机械性牵拉、持续性压迫和缺血三种主要致伤因素共同作用形成的;②以束中心病变为主,Ranvier's 结为薄弱部位,髓鞘为敏感结构成份的纵行广泛分布的特征性损伤,是急性牵拉伤的主要标记;③游离的家兔正中神经延长的生理极限是2.85%±1.44%,应力为12.63±4.78g·(mm²)⁻¹;损伤的临界点为:延长8.57%±1.14%,应力为22.56±3.28g·(mm²)⁻¹;神经纤维的断裂点为延长32.85%±5.11%,应力为182.08±26.11g(mm²)⁻¹;神经干断裂点为延长72.5%±9.4%,应力为361.04±85.6g·(mm²)⁻¹。非游离神经生理极限的延长是1.51±0.28cm,载荷为18.28±3.09g;损伤临界点为延长4.49±1.63mm,载荷为58.07±2.26g;神经干断裂点为延长15.65±3.10mm,载荷为266.74±84.27g。④神经承受急性牵拉时,最先影响的是神经内静脉回流,以后髓鞘发生肿胀、变性,轴突拉长、受压,轴浆内细胞器聚集。随着牵拉的持续,髓鞘发生节段性脱失,轴突呈串珠样改变,并在 Ranvier's 结区锥状变细,此时动作电位开始下降,毛细血管内皮细胞间连接破坏,血管通透性增加,蛋白质外渗,有效循环血量明显减少。进一步牵拉,则发生神经内血栓形成,供血中断,动作电位持续下降,轴突极其细小,离开正常位置,与 Schwann's 鞘脱离,被挤向一侧。在神经干断裂前,内部微循环已完全停止,组织结构损伤严重,但动作电位大部分仍存在。文中还讨论了神经的力学表现及其生物学意义,并对急性牵拉伤可能的损伤机制和本研究的临床意义进行了综合评价。

关键词 定量张力;神经损伤;力学表现;正中神经;活体

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