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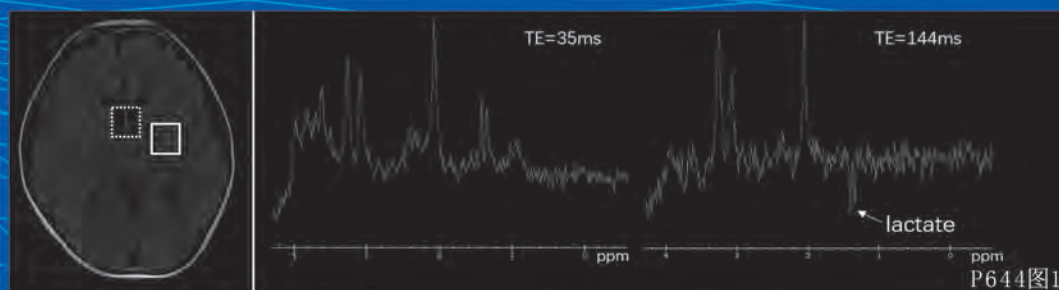
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封面文章

线粒体脑病(mitochondrial encephalopathy, ME)是一种由线粒体DNA或核基因缺陷引起的遗传代谢性疾病,临床上分为多种亚型,常被误诊为缺血性脑卒中、脑炎及脱髓鞘脑病等。脑组织¹H-MRS出现乳酸(Lac)峰是诊断ME特异性较高的征象,目前其临床应用多限于脑内异常信号区。但临床实践中存在的问题是,某些情况下乳酸检测阳性结果的特异性并不高,在很多非线粒体脑病,如脑肿瘤、炎性病变或缺血性损伤等亦可出现乳酸水平的升高;此外,脑组织异常代谢物浓度较低时,其诊断效能亦明显降低。有研究表明,脑内异常代谢所产生的乳酸主要通过脑脊液清除,而乳酸经蛛网膜颗粒回流入血的速度明显慢于其由异常脑细胞清除至脑脊液中的速度;且理论上脑脊液区无NAA、Cho等脑内代谢物的干扰,因此脑脊液中的乳酸浓度甚至高于脑内病灶区。由此我们提出:脑脊液区¹H-MRS能否提高对线粒体脑病的诊断效能呢?这能否为临床诊断提供新的线索和思路,从而降低误诊率呢?本研究收集经线粒体脑肌病标准评分系统确诊的13例线粒体脑病患者,另17例临床上疑似线粒体脑病但最终确诊非线粒体病的患者为对照组,所有患者均行常规MRI和¹H-MRS检查,选取MRI异常信号区、正常脑组织区及脑脊液区为感兴趣区,分别比较两组患者相应区域Lac峰的差异。那么,两组患者的脑脊液区¹H-MRS乳酸峰是否真的存在差异呢?这对于线粒体脑病的诊断是否有价值呢?该研究正在进行下一步的深入探索,详见内文第641~647页。

本期支持单位:中国科学院分子影像重点实验室、首都医科大学附属北京潞河医院

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About the cover

Mitochondrial encephalopathy (ME) is an inherited metabolic disease caused by defects on mitochondrial DNA or nuclear gene. It is clinically divided into multiple subtypes and is sometimes misdiagnosed as ischemic stroke, encephalitis or demyelination encephalopathy, etc. The appearance of Lac peak in ¹H-MRS of brain tissue is a sign of ME and the specificity is relatively high. At present, its clinical application is mostly limited to the abnormal signal region in brain. However, the problem in clinical practice is that the specificity of the positive result of Lac peak is not enough in some cases. In many non-mitochondrial encephalopathy, such as brain tumor, inflammatory lesion or ischemic injury, the level of lactic acid may also rise. In addition, the diagnostic efficiency significantly reduced with the concentration of abnormal metabolite. Studies have shown that the lactic acid produced by abnormal metabolism in the brain is mainly cleared by the cerebrospinal fluid, and the rate of lactic acid refluxing into the blood through the arachnoid granules is significantly slower than the rate of clearance from abnormal brain cells to the cerebrospinal fluid; and theoretically there is no NAA or Cho in the cerebrospinal fluid region. So the concentration of lactic acid in the cerebrospinal fluid is even higher than that in the brain. From this we propose: could the ¹H-MRS in the cerebrospinal fluid region improve the diagnostic efficacy of mitochondrial encephalopathy? Could this provide new clues and ideas for clinical diagnosis, thus reducing the rate of misdiagnosis? This study collected 13 patients with mitochondrial encephalopathy diagnosed by the mitochondrial myopathy standard scoring system, and 17 patients with clinically suspected mitochondrial encephalopathy but eventually diagnosed with non-mitochondrial disease as the control group. All patients have undergone routine MRI and ¹H-MRS examination. The MRI abnormal signal region, the normal brain tissue region and the cerebrospinal fluid region were selected as the ROI, and the differences of Lac peaks in corresponding regions were compared. Then, whether the Lac peak in the cerebrospinal fluid region of two groups of patients really differed? Is this valuable for the diagnosis of mitochondrial encephalopathy? This research will be go on in the near future. See text page 641-647.