·个案报道·

脑出血微创术后无症状性脑脓肿误诊为包膜性血肿1例

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收稿日期 2022-01-19

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关键词 脑出血；脑脓肿；误诊；包膜性血肿

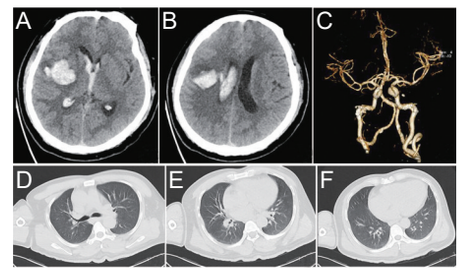
中图分类号 R741；R741.05；R743.34 **文献标识码** A **DOI** 10.16780/j.cnki.sjssgncj.20210238

本文引用格式：徐振熙, 贺峰, 陈旺, 宫健. 脑出血微创术后无症状性脑脓肿误诊为包膜性血肿1例[J]. 神经损伤与功能重建, 2022, 17(12): 822-824.

隐源性脑脓肿因其感染征象不显著，易漏诊[1]。脑出血吸收期包膜形成致持续性脑水肿与脑脓肿具相似影像征象，易误诊。本文将回顾性分析1例基底节出血微创术后形成无症状性脑脓肿被误诊为包膜性血肿再次实施微创引流的患者资料，增加临床医师对2种疾病的认知，减少漏诊，避免误诊。

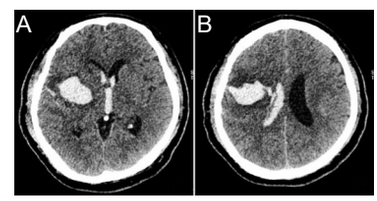
1 病例资料

患者，男，39岁。2020年5月13日因“头痛伴言语不清、左侧肢体无力2 h”第1次入院。既往高血压病史5年，血压管控差。个人酗酒史，吸烟指数150。体格检查：体温36.3℃，心率100次/分，呼吸20次/分，血压201/100 mmHg（1 mmHg=0.133 kPa）；格拉斯哥昏迷量表评分（Glasgow Coma Scale，GCS）13分，嗜睡，构音障碍，双侧眼球同向右侧凝视，双侧瞳孔直径为3 mm，对光反射灵敏，左侧中枢性面舌瘫，颈项强直3横指，左侧肢体肌力0级，右侧肢体肌力Ⅴ级，左侧Babinski征阳性。实验室检查：白细胞14.42×10^9/L，中性粒细胞百分比84.7%，淋巴细胞百分比6.8%，中性粒细胞绝对值12.22×10^9/L，淋巴细胞绝对值0.98×10^9/L；生化检查大致正常。颈部血管、心脏、双下肢深静脉超声未见显著异常。头颅CT检查：右侧基底节出血破入脑室；CT血管成像（computed tomography angiography，CTA）未见显著异常；双肺CT检查未见显著异常（图1）。初步诊断：①基底节出血；②继发性脑室出血；③高血压病Ⅲ级（极高危）。给予控制血压、适度降低颅内压、适度镇痛镇静与对症支持治疗。发病24 h头颅CT未见活动性出血（图2）。发病48 h头颅CT无再出血影像表现，双肺CT示右肺下叶炎性改变（图3）；降钙素原为0.81 ng/mL，结合患者中等度热，留取痰培养，启用头孢哌酮舒巴坦钠抗感染。发病72 h，患者意识状态恶化为昏睡，头颅CT证实脑水肿加重、中线显著移位（图4）；实施脑出血立体定向微创穿刺引流。术后第1天，颅脑CT示血肿大部已被清除，拔除引流管（图5）。术后第2天，腰椎穿刺示颅内压80 mmH_2O，脑脊液为红色，白细胞数55×10^6/L，单核细胞百分比23.6%，分叶核细胞百分比76.4%，生化正常，脑脊液细菌培养阴性，二代宏基因组测序亦未检出致病菌。术后第3天，痰培养与药敏实验结果证实为敏感抗生素型金黄色葡萄球菌，降钙素原为0.16 ng/mL。住院第13天，患者神志清，构音障碍，左侧肢体肌力0级。转往当地医院进行言语与肢体康复功能锻炼，期间未再发热，已停用抗生素。



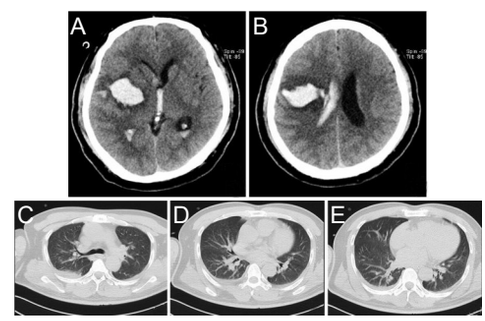
注：（A-B）头颅CT：右侧基底节团、片状高密度影，脑室系统高密度影；（C）头颅CTA未见异常；（D-F）双肺CT未见异常

图1 患者入院影像学检查



注：（A-B）头颅CT：出血量较前无增大

图2 患者发病24 h影像学检查

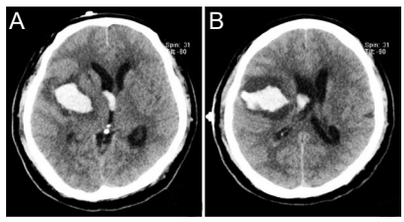


注：（A-B）头颅CT：出血量较前无增大；（C-E）双肺CT：右肺下叶炎性渗出

图3 患者发病48 h影像学检查

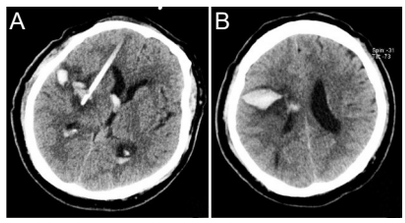
出院后1个月，于我院门诊随诊，颅脑CT示右侧基底节出血部位包膜形成（图6）。患者因“脑出血术区包裹性占位”第2次入我科。患者精神萎靡；双肺呼吸音清，未闻及干、湿性啰音，左侧肢体肌力Ⅰ级，右侧肢体肌力Ⅴ级。再次入院双肺CT未见

显著异常(图6)。实验室检查:白细胞11.56×10⁹/L,中性粒细胞百分比81%,淋巴细胞百分比12.4%,中性粒细胞绝对值9.35×10⁹/L,淋巴细胞绝对值1.44×10⁹/L;生化检查正常;C反应蛋白(C reactive protein,CRP)5.94 mg/L。诊断:脑出血吸收期包膜形成。鉴于包膜功能性分泌将致脑水肿进行性加重,再次实施立体定向微创穿刺引流,术中感包膜较韧,引流出大量脓性液体(图7)、味臭,留取脓培养,启用美罗培南联合利奈唑胺抗感染治疗。修正诊断:脑脓肿。术后第1天,颅脑CT示脓液大部被清除(图7),拔除头部引流管。术后第2天,腰椎穿刺示颅内压300 mmH_2O,脑脊液清亮,白细胞数12×10⁶/L,分叶核细胞百分比0%,单个核细胞百分比100%,脑脊液糖4.99 mmol/L,蛋白质866 mg/L,氯化物128.1 mmol/L。术后第3天,脓培养与药敏实验结果证实为耐甲氧西林金黄色葡萄球菌,利奈唑胺与万古霉素均为敏感型抗生素;调整抗生素方案:利奈唑胺抗感染。术后第5天,白细胞8.34×10⁹/L,中性粒细胞百分比66.6%,淋巴细胞百分比24.9%,中性粒细胞绝对值5.57×10⁹/L,淋巴细胞绝对值2.07×10⁹/L;CRP 2.01 mg/L;降钙素原0.04 ng/mL。术后第6天颅脑磁共振(图8)证实为脑脓肿术后影像。住院11 d,患者神志清,轻度构音障碍,左侧肢体肌力Ⅰ级,右侧肢体肌力Ⅴ级;再次转入当地医院行肢体康复功能锻炼。3个月门诊随访,期间无发热,未应用抗生素,言语清,左侧上肢肌力Ⅲ级,左侧下肢肌力Ⅳ级,颅脑CT示脓肿灶已吸收(图9)。



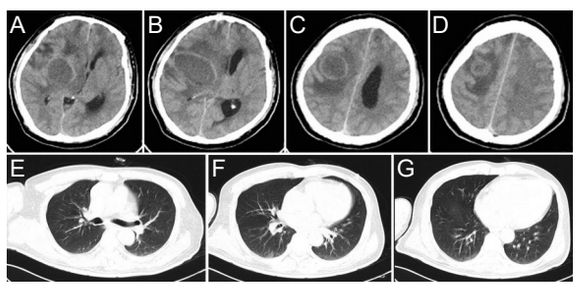
注:(A-B)头颅CT:出血量较前无增大,脑肿胀较前加重,中线显著移位

图4 患者发病72 h影像学检查



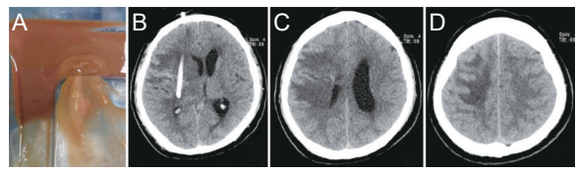
注:(A-B)头颅CT:血肿较前显著减少

图5 患者术后第1天影像学检查



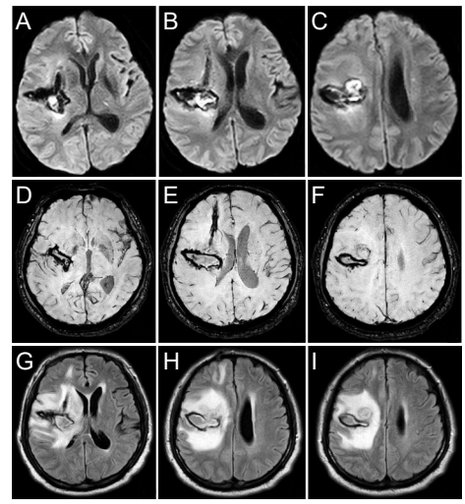
注:(A-D)头颅CT:微创引流区环状包膜性占位,周边白质低密度环绕;(E-G)双肺CT:未见显著异常

图6 患者出院后1个月影像学检查



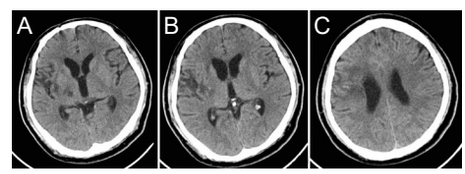
注:(A)术中抽吸出大量脓性液体;(B-D)头颅CT示脓液大部被清除,环状包膜已被破坏,残留小体积环状包膜性占位

图7 患者第2次微创引流影像



注:(A-C)DWI:引流病灶呈混杂高信号;(D-F)SWI:病灶外周存在不完整的显著低信号环;(G-I)FLAIR:病灶周边较大范围血管源性水肿

图8 患者第2次微创术后第6天颅脑MRI



注:(A-C)头颅CT:病灶已被吸收,右侧壳核与丘脑残余部分低密度灶

图9 患者第2次微创术后3个月影像学检查

2 讨论

患者术中引流出脓性液体,脓液培养为金黄色葡萄球菌,脑脓肿诊断明确。脑脓肿呈现典型三联征头痛、发热及局灶性症状的概率仅为20%,发热出现率约为53%[2]。本例患者属无热性颅内感染,脑脊液检查证实无颅内感染,致诊断困难。患者1个月前实施基底节出血微创引流术,目前处于脑出血吸收期,是包膜形成高峰期。包膜的病理属性含新生毛细血管伴袖套状淋巴细胞浸润,血肿腔内不断有血液成分经新生毛细血管渗漏致其张力逐渐增大,从而导致血肿腔内水分渗入到包膜周围形成持续性脑水肿[3]。因此包膜型血肿与包裹性脓肿具有相似的CT影像征象:环状包裹性占位伴灶周血管源性水肿;亦增加了二者相互误诊率。

化脓性细菌致脑脓肿的感染途径分为邻近组织感染、血源性感染、开放性颅脑损伤及颅脑手术感染[2,4]。该患者基底节出血实施微创穿刺引流,留置头部引流管<24 h、时间短,脑脊液检查证实无颅内感染征象,排除手术相关感染。患者发病48 h

后出现右肺下叶感染，感染菌经痰培养证实为金黄色葡萄球菌，与第2次入院时脓液培养结果一致；因此，推测该患者肺部致脓菌经血源性循环进入基底节血肿区。患者为青年男性，无消耗性疾病史，机体抵抗力强于金黄色葡萄球菌致病能力，敏感性抗生素的应用亦降低了金葡菌致病力，脓肿灶经较短时间即进入包膜期，掩盖了典型脓肿症状。

颅脑磁共振（magnetic resonance imaging，MRI）是诊断脑脓肿的最有价值影像技术，包膜期的特征性MRI表现为脓腔内T_1低信号，T_2高信号，弥散加权成像（diffusion weighted imaging，DWI）高信号；脓腔周围T_1为低信号、T_2高信号的水肿区；增强后可见完整、均匀、光滑的环形脓腔壁[2,5]。因患者无颅内感染征象，处于脑出血吸收包膜形成高峰时期，误诊为包膜性血肿，故术前未行颅脑MRI检查。

该患者包膜性肿块占位效应显著，精神状态恶化，不管是包膜性血肿或脓肿包膜期，均具有手术指征，考虑占位体积大、位置深，因此实施微创穿刺引流[6,7]。与开颅脓肿切除相比，微创穿刺引流尽管创伤小、感染率低、失血少，但脓液引流不彻底，脓肿壁清除困难，易反复发作[8]。该患者微创术后脓肿壁破坏，3个月后，脓肿逐渐被吸收。

综上所述，对颅内包膜占位性病变，无论定性是否与其临床表现一致，除颅脑CT检查外，均需完善颅脑MRI明确病变性质，避免经验主义的惯性思维。除此之外，对包裹性脓肿可实施微创穿刺引流，微创穿刺引流可破坏脓肿壁，促进脑脓肿逐步被吸收。

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（本文编辑：唐颖馨）

（上接第781页）

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（本文编辑：唐颖馨）