

左房粘液瘤致大面积脑梗死误诊误治 1 例

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心脏粘液瘤往往临床表现不典型,很容易导致临床漏诊。本文就 1 例心房粘液瘤并发脑卒中的诊治进行回顾及探讨,报道如下。

病例资料

患者女性,32 岁,因“与人争吵后出现不语,四肢无力半小时”入院,患者与家属争吵后出现不言语、四肢乏力,无意识障碍,无头痛,无恶心呕吐,被送入院就诊。否认高血压、糖尿病、心脏病等基础病史。查体:心率 76 次/min,呼吸 18 次/min,血压 130/70 mmHg,神志清楚,不与人交谈,双瞳孔等大等圆,直径 2.5 mm,光反应灵敏,鼻唇沟无变浅,伸舌不配合,心律齐,各瓣膜区未及病理性杂音,四肢肌力 5 级,病理征未引出。查心电图、头颅 CT 未见明显异常。请神经内科会诊后考虑癔症,过度通气,暂予以留观处理。于次日 11 am 请精神科会诊后考虑精神因素收住精神科,当天下午 3 pm 出现意识障碍加重,呕吐数次,查体欠合作,神志嗜睡,右侧肢体扬鞭征阳性,右侧肢体肌张力低,右侧病理征阳性。血常规:白细胞 $4.5 \times 10^9/L$,血红蛋白 112 g/L,血小板计数 $145 \times 10^9/L$,C-反应蛋白 15 mg/L,丙氨酸转氨酶 23 U/L,天门冬氨酸转氨酶 35 U/L,尿素氮 7.6 mmol/L,肌酐 $81 \mu\text{mol/L}$,凝血功能:活化部分凝血酶原时间 43.5 s,凝血酶原时间 13.4 s,凝血酶时间 17.7 s,纤维蛋白原 6.58 g/L,D-二聚体 $1.98 \mu\text{g/mL}^{[1]}$,纤维蛋白(原)降解产物 6.40 $\mu\text{g/mL}$ 。头颅核磁共

振(MRI):左侧基底节区、左侧额颞顶叶大面积脑梗死。液体衰减反转恢复序列(fluid attenuated inversion recovery, FLAIR)及弥散加权成像(diffusion weighted imaging, DWI)呈明显高信号,非对比剂增强动脉自旋标记灌注成像(arterial spin labeling, ALS)呈明显低灌注,脑血流(cerebral blood flow, CBF)减低,见图 1。再次请神经内科会诊,于 5 pm 转入神经内科,追问病史患者家属诉入院前即有右侧肢体乏力,考虑到患者病程已超过溶栓时间窗,不再考虑溶栓,予以脱水降颅压等治疗。后因脑水肿,颅内压增高,脑疝形成,于次日 3 am 行左侧开颅探查、去骨瓣减压、颅内压监测装置(脑室型)置入术,术中发现左额叶脑内出血,遂同时行血肿清除术,术后转入 ICU 加强监护治疗。期间完善脑 CT 血管造影(CTA):左侧大脑中动脉 M1 段可见局限性充盈缺损,其远侧分支纤细稀疏,见图 2。超声心动图示左房内见异常回声,大小约 45 mm × 26 mm,呈椭圆形,回声强度中等,分布均质,活动度较大,舒张期瘤体脱入二尖瓣口,收缩期进入左房内,考虑心脏粘液瘤可能性大,见图 3。在 ICU 住院 1 周,病情平稳后要求回老家行手术治疗,术后病理证实为粘液瘤。目前患者仍处于睁眼昏迷状态,瘫痪在床,在进行康复治疗。

讨论

心脏粘液瘤的形成机制目前尚不清楚。Kirschner

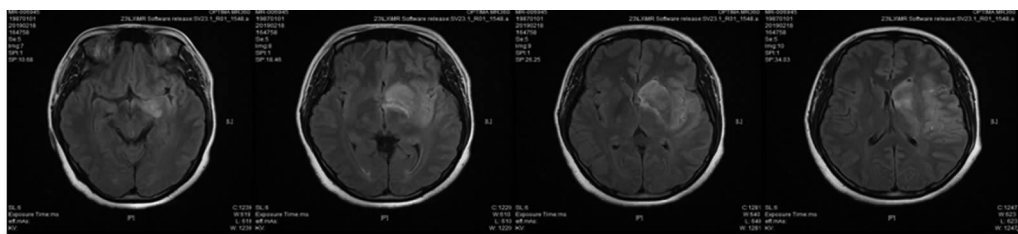


图 1 头颅核磁共振

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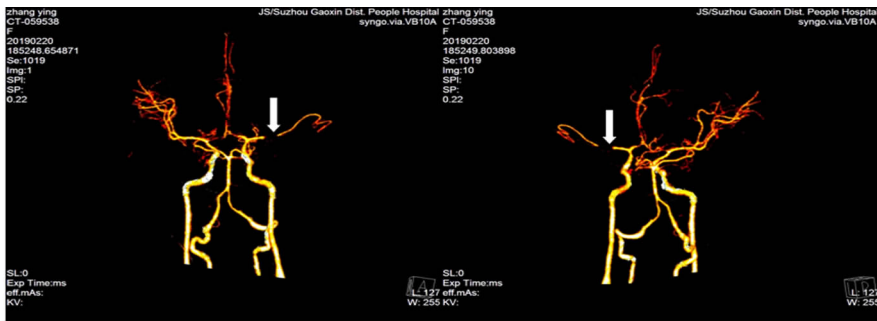


图2 脑CT血管造影



图3 超声心动图

等^[2]于1845年首次报道粘液瘤也可能是一种常染色体显性疾病,它是由染色体长臂上的一个基因突变引起的,大约是17号染色体(17q22-24)。从心脏粘液瘤组织的来源来看,粘液瘤肿瘤细胞来源于原始的多能间充质干细胞。心脏粘液瘤多见于女性,发病高峰在30~60岁左右。^[3]心脏粘液瘤最常见的部位是左心房, Lee等^[4]从梅奥诊所收集了74例心脏粘液瘤患者,其中81%为心房粘液瘤。粘液瘤发现可能是偶然的,2%~10%的患者可能无症状,心房粘液瘤可引起2%的青年人发生脑卒中,脑卒中的发生往往早于心脏不适。该患者在初诊及住院后心脏听诊确实无心脏杂音,从而在急诊没有引起医师足够的重视,还包括对本病的认识不足,所以没有及时做心脏超声发现粘液瘤^[5]。另该患者由于年龄轻、有吵架情绪诱因,表现不讲话,误导医师更多的考虑功能性疾病,忽视了其他重要的有意义的体征信息,从而延误了脑卒中的诊治。

脑卒中与粘液瘤有关,通常是由肿瘤或血栓形成引起的,主要发生在左侧大脑中动脉区,大概是因为它的优势血流。这也导致中动脉栓塞会引起大面积的脑梗死,而大面积脑梗死也容易并发脑出血,其原因是由于梗死区内的小滋养动脉通透性增高,当侧支循环开放、血液再灌注时,可导致小滋养动脉压力增高而破裂出血。该患者术中发现并发出血,随后进行了血肿清除,降低颅内压^[6]。

最近一项研究报道,在9%~22%的心房粘液瘤中观察到栓塞性卒中。栓塞的发生率与肿瘤的大小无关,而与肿瘤的移动性、易碎性有关^[7,8]。对于实性、圆形的心脏粘液瘤,表浅血栓是栓子的来源,

而对于软、不规则的心脏粘液瘤,栓子最有可能与粘液瘤组织的栓塞表面和蜕膜碎片有关^[9]。形态上,具有长绒毛的表面的肿瘤容易被栓塞。息肉型肿瘤致全身性栓塞的发生率高于圆形型^[10]。

欧洲卒中组织在2008年“缺血性脑卒中治疗指南”中指出,怀疑多发性脑或全身动脉区梗死时推荐栓塞时进行心脏超声心动图检查。超声心动图是青年脑卒中和无症状脑卒中患者的一种不可忽视的检查方法^[11]。若没有脑血管病的危险因素,也没有房颤,就应怀疑粘液瘤可能是脑栓塞的原因之一,尽管它很少见。对于有脑卒中的年轻患者来说,考虑心源性的来源是很重要的。心脏粘液瘤由于栓塞性危险因素,一旦确诊,就需要立即手术切除,术后需进行脑CT、MRI或数字减影血管造影随访^[12]。

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