

颅内破裂动脉瘤栓塞术后并发吉兰-巴雷综合征 1 例

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【摘要】 吉兰-巴雷综合征 (GBS) 是一种自身免疫性神经疾病, 主要特征是四肢无力和肌张力减低。颅内破裂动脉瘤栓塞术后出现肢体肌力下降一般认为是症状性血管痉挛, 通常发生在术后 4~10 d。本文报道 1 例 55 岁男性左侧颈内动脉 C6 段破裂动脉瘤, 血管内栓塞术后 12 d 突然出现渐进性四肢无力, 四肢肌张力减低、腱反射消失, 完善神经传导检查、脑脊液病毒抗体、血清周围神经抗体检查等检查确诊 GBS。予以免疫球蛋白治疗后康复出院, 7 个月随访恢复正常。这提示颅内破裂动脉瘤栓塞术后脑血管痉挛期出现无明确病因的四肢肌力减退时, 应当考虑到 GBS, 早期诊断对于避免延误病情和及时治疗非常重要。

【关键词】 颅内破裂动脉瘤; 血管内栓塞; 吉兰-巴雷综合征

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A case of Guillain-Barré syndrome after embolization of ruptured intracranial aneurysm

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【Abstract】 Guillain-Barré syndrome (GBS) is an autoimmune neurological disorder characterized by weakness and decreased muscle tone in the limbs. The decrease in limb muscle strength that occurs after endovascular embolization for ruptured intracranial aneurysms is generally considered symptomatic vasospasm, usually occurring 4~10 days after surgery. This article reports a case of a 55-year-old male with a ruptured aneurysm at the C6 segment of the left internal carotid artery who suddenly developed progressive limb weakness, decreased muscle tone in all limbs, and absent tendon reflexes 12 days after endovascular embolization. The diagnosis of GBS was confirmed by comprehensive neurological examinations, cerebrospinal fluid viral antibody testing, and serum peripheral nerve antibody testing. The patient recovered and was discharged after receiving immunoglobulin therapy, and normal recovery was observed during a 7-month follow-up. This suggests that when limb muscle weakness without a clear cause occurs during the cerebral vasospasm period after endovascular embolization of ruptured intracranial aneurysms, GBS should be considered, and early diagnosis is crucial for preventing the delay of the disease's diagnosis and enabling timely treatment.

【Key words】 Ruptured intracranial aneurysms; Endovascular embolization; Guillain-Barré syndrome

1 病例资料

55 岁男性, 因突发头晕、头痛 1 h 余于 2022 年 3 月 21 日入院。入院体格检查: 血压 219/113 mmHg, 神志嗜睡, GCS 评分 14 分, 颈抗两横指, 四肢肌力 4 级。入院后头颅 CT 见鞍上池、大脑镰及部分脑沟表面有多发条片状高密度影 (图 1A), 考虑动脉瘤性蛛网膜下腔出血 (subarachnoid hemorrhage, SAH)。立即行 DSA 检查证实左侧颈内动脉 C6 段动脉瘤 (图 1B、1C)。急诊在全麻下行动脉瘤栓塞术, 术后造影显示动脉瘤完全栓塞 (图 1E、1F)。术后神志和四肢肌力同术前。术后复查头颅 CT 未见新发颅内血肿和脑梗死表现 (图 1D)。术后 12 d, 病人突然出现渐进性四肢无力, 左侧肢体肌力 3 级, 右侧肢体肌力 2 级, 四肢肌张力均减低, 腱反射消失; 术后 13 d, 四肢肌力降至 0 级, 肌张力减退, 腱反射消失。复查头颅 CT 及 MRI 未见新增出血及新发梗死, 故排除迟发性缺血性神经

功能缺损 (delayed-ischaemic neurological deficits, DIND)。立即完善神经传导、脑脊液病毒抗体、血清周围神经抗体等检查, 脑脊液检查出现蛋白-细胞分离、神经传导检查示周围神经损害, 联系神经内科会诊, 确诊吉兰-巴雷综合征 (Guillain-Barré syndrome, GBS), 予以免疫球蛋白治疗。出院时, 双上肢肌力 4 级、双下肢肌力 3 级, 肌张力正常, 腱反射减退, 病理征未引出。2023 年 10 月 21 日随访, 四肢肌力恢复至 5 级, 肌张力正常, 腱反射正常。

2 讨论

SAH 和 GBS 是两种机制不同的疾病, 其中 SAH 是神经外科常见急症, 动脉瘤破裂是最常见的病因, 主要表现为头痛伴恶心、呕吐, 术后常见的并发症包括脑血管痉挛和 DIND^[1]。当 SAH 术后出现肌力改变时, 往往优先考虑是否发生了 DIND, 而非 GBS。SAH 术后 DIND 指在 SAH 术后出现的新发神经功能缺损症状, 包括肢体无力、感觉异常、语言障碍、嗅觉改变等, 一般发生在 SAH 后 4~10 d, 第 8 天是发病高峰期^[2]。

GBS 是一种脱髓鞘性多发性神经病, 可引起感觉、运动和自主神经症状, 病情程度轻重不一, 症状严重程度与神经

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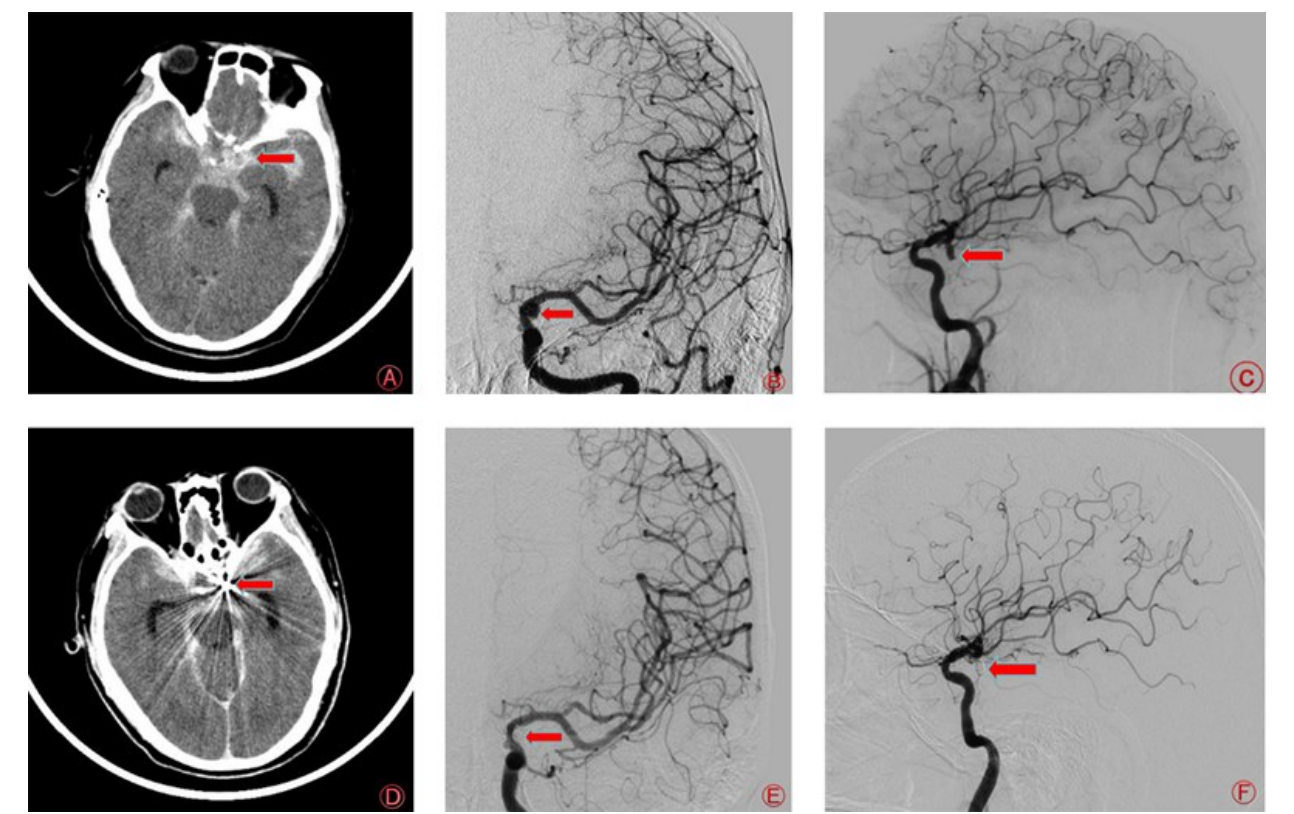


图 1 左侧颈内动脉破裂动脉瘤血管内栓塞术后并发吉兰-巴雷综合征的影像学表现

A. 术前头颅 CT 示自发性蛛网膜下腔出血;B、C. 术前 DSA 证实左侧颈内动脉 C6 段动脉瘤,大小约 4.2 mm×2.4 mm,瘤颈宽约 1.1 mm;D. 术后复查头颅 CT 见鞍区左侧新有结节状金属样密度影及放射状伪影;E、F. 术后 DSA 见左侧颈内动脉 C6 段动脉瘤完全栓塞;红色 ↑ 示左侧颈内动脉动脉瘤

Figure 1 Image findings of a patient with a ruptured intracranial aneurysm of the left internal carotid artery associated with Guillain-Barré syndrome after endovascular embolization

A: Preoperative CT shows spontaneous subarachnoid hemorrhage. B-C: Preoperative DSA confirms a 4.2 mm × 2.4 mm aneurysm in the C6 segment of the left internal carotid artery, with a neck width of approximately 1.1 mm. D: Postoperative CT shows a new nodular metallic density and radiating artifacts in the left cavernous sinus. E-F: Postoperative DSA shows complete embolization of the aneurysm. Red arrow indicates the aneurysm.

损伤程度相关,发病前大多数有感染诱因,临床表现为急性或亚急性起病的对称性四肢软瘫,腱反射减弱或消失,伴或不伴有感觉障碍等神经系统症状,恢复期长短不一^[3]。目前,GBS 的发病机制尚不明确,这种免疫反应以某种错误的表达方式攻击了宿主的神经组织,可能是通过识别分子相似的表位机制,针对这些表位的免疫反应导致急性脱髓鞘神经病变,进而出现一系列的神经功能障碍。术后出现的 GBS,其机制可能是手术引起自身抗原释放,机体免疫系统对自身抗原产生免疫应答,打破免疫系统平衡造成机体免疫功能破坏后引起的一系列反应^[4]。蛋白-细胞分离不是 GBS 的特异性指标,有报道称只有 64% 的病人可出现蛋白-细胞分离现象^[5],所以其并不是诊断 GBS 的必要条件,并且疾病严重程度与脑脊液蛋白含量无关^[6,7]。静脉注射免疫球蛋白被视为一种可行的治疗方法,无论是作为一线或二线治疗^[8,9]。静脉注射免疫球蛋白或血浆置换这两种治疗方法的顺序并没有凸显出任何特别的益处^[10]。随着克服轴突变性和增强神经再生的研究,GBS 的治疗前景是积极乐观的^[11]。手术或创伤是诱发

GBS 的一个可能潜在的危险因素,在临床上易忽视,从而导致治疗延误。因此,早诊断并及时治疗能降低 GBS 的致残率和病死率^[12-15]。

GBS 的诊断具有挑战性,了解其表现的特异性及其缺陷,对于及时和准确地诊断至关重要^[16,17]。本文病例术后连续行腰椎穿刺术释放血性脑脊液及使用尼莫地平预防脑血管痉挛^[18],术后复查头颅 CT 显示出血明显减少,治疗过程中血压平稳,意识清楚,当病人出现四肢肌力减退逐渐加重,未出现意识水平下降,经颅多普勒超声检查颅内血管流速未见明显降低,复查头颅 CT 及 MRI 未见新增出血及新发脑梗死,故排除 DIND。但脑脊液检查出现蛋白-细胞分离、神经传导检查示周围神经损害,神经内科会诊后诊断 GBS,予免疫球蛋白治疗有效,康复出院,进一步验证该病人发生了 GBS。本文病例颅内破裂动脉瘤诊断明确,术前及术后早期无肢体功能障碍,术后 12 d 才出现 GBS 表现,复查头颅 MRI、颈椎及胸椎 MRI 无新发颅内出血和梗死,以及脊髓病变等情况。本文病例表明临床医师在颅内破裂动脉瘤栓塞术后脑血管痉挛

期仍需警惕 GBS 的存在。我们认为该病人颅内破裂动脉瘤栓塞术后并发 GBS 的可能机制:①手术创伤致机体免疫系统发生错误识别,进而对自身正常的周围神经启动免疫攻击;②血脑屏障破坏及脑血管微血栓形成导致神经损伤,损伤可累及神经髓鞘,髓鞘蛋白释放进入脑脊液与血液中,含量迅速增加,引起神经根及周围神经免疫应答,从而导致神经脱髓鞘病变;③手术和麻醉协同作用使机体处于免疫抑制状态,导致神经内分泌轴功能异常,进而介导免疫功能异常。

总之,颅内破裂动脉瘤栓塞术后出现无明确病因的四肢肌力减退时,应当考虑到 GBS,早期诊断对于避免延误病情和及时治疗非常重要。

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