Archives of Nephrology

ISSN: 2639-3573

Volume 2, Issue 2, 2019, PP: 11-15

Spontaneous Spinal Epidural Hematoma Mimicking Ischemic Stroke: An Experience in a Patient Undergoing Hemodialysis

Tetsu Akimoto[1,2]*, Daisuke Nagata[2]

[1]Department of Chronic Kidney Disease Pathophysiology, Jichi Medical University, Shimotsuke, Tochigi, Japan.
[2]Division of Nephrology, Department of Internal Medicine, Jichi Medical University, Shimotsuke, Tochigi, Japan.
tetsu-a@jichi.ac.jp

***Corresponding Author:** Tetsu Akimoto, Division of Nephrology, Department of Internal Medicine, Jichi Medical University, Yakushiji, Shimotsuke-Shi, Japan.*

Abstract

Hemiparesis develops in response to various neurological disorders, including stroke, neoplasms and several inflammatory processes. Occasionally, it may otherwise occur due to a lesion in the high cervical spinal cord. In this report, we describe a case of spontaneous cervical spinal epidural hematoma in a chronic hemodialysis patient who initially presented with left hemiparesis and dysarthria, which encouraged us to incorrectly attribute the illness to a cerebrovascular event. Several diagnostic and management concerns relevant to this disease condition are discussed.

Keywords: hemiparesis, spontaneous cervical spinal epidural hematoma, stroke mimic, hemodialysis, end-stage kidney disease.

Introduction

Hemiparesis develops in response to various neurological disorders. Although the presence of several concomitant cranial nerve signs or facial weakness generally prompts a search for cerebral etiologies, such as stroke, neoplasms and several inflammatory processes, it may otherwise occur due to a lesion in the high cervical spinal cord [1,2]. In this report, we describe a case of spontaneous cervical spinal epidural hematoma in a patient receiving chronic hemodialysis (HD) treatment who initially presented with left hemiparesis and dysarthria. Various diagnostic and management concerns relevant to this disease condition are discussed.

Case Report

The patient was a man in his seventies who experienced a sudden onset of neck pain and left upper limb weakness when he was in a hospital for the surgical revision of an autogenous radial-cephalic arteriovenous fistula in his forearm. Despite no apparent past history of neurological disease, he gradually developed weakness in his left lower limb in association with the deterioration of the upper limb symptoms over the subsequent eight-hour period. He had end-stage renal disease due to hypertensive nephrosclerosis and had received HD treatment for nine months. His medical history included ischemic heart disease, which had been diagnosed five years previously, and he had undergone several vascular access interventions, including percutaneous transluminal angioplasty, after the initiation of the periodic HD program. He denied any antecedent trauma, exertional activity, sneezing or vomiting.

On a physical examination, he was alert but exhibited mild dysarthria characterized by slurred speech without any facial drooping or tongue deviation and left hemiparesis with moderate weakness (grade 3/5 on the Medical Research Council [MRC] scale) in the left arm both proximally and distally, and severe weakness (MRC scale grade 2/5) in the left leg [3]. The sensory examination revealed normal perception of pinprick, light touch, vibration and proprioception. The deep tendon reflexes in the lower limbs were

brisk, while the left side reflex was more prominent than that of the right side, and the ipsilateral extensor plantar response (Babinski sign) was also present. The patient's blood pressure was 178/97 mmHg and his respiratory rate was 12 breaths/min. A laboratory evaluation revealed: blood urea nitrogen, 45.1 mg/dL; serum creatinine, 5.97 mg/dL; hemoglobin, 12.1 g/dl; hematocrit, 36.9% and a platelet count of 14.7 x 10^4/μL, with a normal prothrombin time of 10.4 seconds and an activated partial thromboplastin time of 30.1 seconds. Electrocardiography demonstrated normal sinus rhythm. The diagnostic brain magnetic resonance imaging (MRI), including T2 star-weighted imaging, failed to demonstrate any findings compatible with intracranial hemorrhage. Despite the absence of changes in diffusion-weighted imaging of the brain, the presumptive diagnosis was ischemic stroke, based on the fact that the patient was suffering from left hemiparesis associated with dysarthria. Thus, anti-platelet therapy was administered with intravenous ozagrel sodium, an inhibitor of thromboxiane A2 synthesis, at a dose of 80 mg/day. Twenty-four hours later, his slurred speech persisted, while his neck pain had worsened and right shoulder pain had developed. Besides the progression of the left side muscle weakness to paralysis (left arm, grade 0/5; left leg, grade 0/5), right side motor disturbance also gradually developed over the next 48 hours (right arm, grade 1/5; right leg, grade 1/5). MRI of the cervical spine revealed a dorsal epidural mass extending from C2 to C5 and causing spinal cord compression (**Figure 1**). Based on the clinical symptoms and imaging findings, an epidural hematoma of the cervical spine was highly suspected. The administration of ozagrel sodium was discontinued and C3 through C5 bilateral laminectomy was emergently performed for decompression and evacuation of the epidural hematoma, which was confirmed by gross and microscopic inspection. No other pathology was identified after complete evacuation of the hematoma. The postoperative course was uneventful. Despite the application of rehabilitation for muscle weakness beginning three days after the operation, the recovery from quadriplegia was marginal (right arm, grade 3/5; right leg, grade 1/5; left arm, grade 0/5; left leg, grade 1/5).

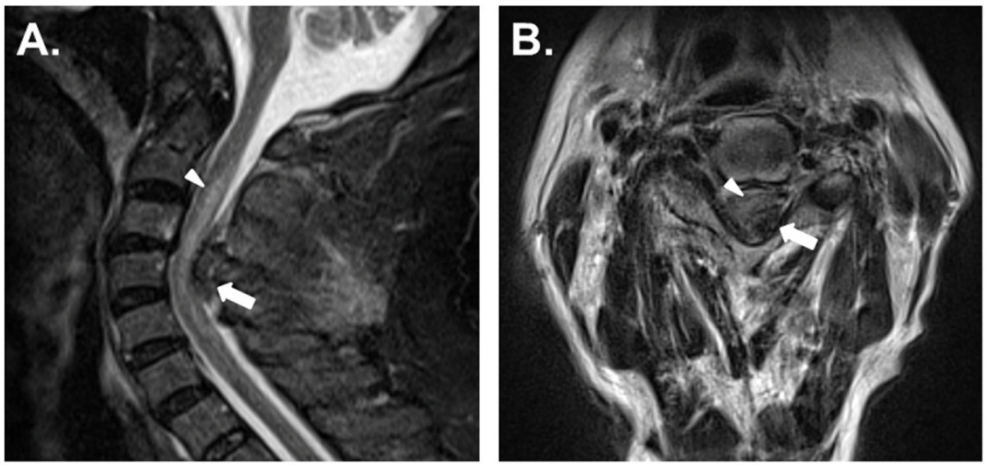

Figure 1.

Fig 1. *MRI of the cervical spinal cord. (A) A sagittal T2-weighted short inversion time inversion recovery image showed a longitudinal heterogeneous hypointense mass extending from the C2 to C5 (arrow) on the posterior side of the spinal cord and a compressed cord with intramedural hyperintensity (arrowhead), indicating recent cervical spinal epidural hematoma, centered on C4, with spinal cord edema. (B) The axial view of a T2-weighted image showed an ovoid mass (arrow) in the left and posterolateral aspect, and spinal cord compression (arrowhead).*

Discussion

Spontaneous cervical spinal epidural hematoma is a rare disabling spinal emergency, representing less than 1% of spinal epidural space-occupying lesions [2,4]. Hemorrhagic disorders due to anticoagulants, thrombolytics and anti-platelet agents, platelet dysfunction, pregnancy, vascular malformation, neoplasms and systemic diseases, such

as hypertension and rheumatoid arthritis, have been regarded as predisposing factors, although numerous cases without any known underlying cause have also been demonstrated [2,4,5]. Given the abnormal platelet function resulting from the uremic milieu and/or the disturbance of hemostasis induced by HD anticoagulation [6], the clinical scenario of the current case may not be surprising. Indeed, there are several anecdotal reports describing the disease in patients on chronic HD [7-12]. However, all of these patients exhibited paraplegia or quadriplegia in the acute phase, which led to a prompt diagnosis after admission. The significance of our experience should therefore be evaluated carefully. This is the first report to describe the disease in one such patient who manifested hemiparesis at the initial presentation, emphasizing the diagnostic pitfalls in evaluating patients with acute hemiparesis.

Patients with spontaneous cervical spinal epidural hematoma may occasionally experience neck or back pain radiating to the corresponding dermatome, which is not dealt with seriously until the subsequent development of cord compression and neurological deficits [2,5]. Most patients manifest paraplegia or tetraplegia [2,4,5]; hemiparesis has been regarded as a rare feature of the disease [13-15], although information on hemiparetic casesis still being accumulated anecdotally [2,16-21]. In most cases, the presence of the spinal lesion was accurately and promptly disclosed during the diagnostic workup [2]. However, it should be noted that there were several patients whose manifestations were incorrectly attributed to ischemic cerebrovascular accidents, and thus, the patients were subjected to treatment with an anticoagulant [16,17], a thrombolytic agent [18,19] or an anti-platelet agent [20,21] before the link between the neurological deficits and cervical spinal epidural hematoma was confirmed.

In the present patient, the neck pain was inadvertently dismissed during the initial physical assessment, while the concurrent dysarthria, which has received attention as a potential sign for discriminating conditions mimicking stroke from actual stroke [22,23], encouraged us to attribute the illness of our patient to a cerebrovascular event [1], thereby leading to the improper anti-platelet therapy. The bleeding risk associated with antiplatelet agents may not necessarily be comparable to those of thrombolytics and anticoagulants; however, a previous presentation showing the expansion of an already developing cervical spinal epidural hematoma following the addition of heparin, based on a presumptive diagnosis of a cardiac ischemic event [24], implies that the clinical picture of the current patient might have been unfavorably modulated by the anti-platelet treatment. At present, we have no idea about the precise cause of dysarthria in our patient. However, it is a rather subjective symptom of patients with stroke, and it may be reasonable to speculate that the slurring of the patient's speech might have resulted from removing his dentures during the acute phase of the disease, as was described by Son et al. [18].

In ordinary clinical practice, patients with acute stroke may not initially see a neurologist, since academic medical centers with systemic neurological assessment programs are not necessarily available in most communities [2,25]. This may also be the case in the majority of chronic HD patients with neurological emergency, while such complications have been shown to contribute to the morbidity and mortality of patients with various degrees of chronic kidney disease [26]. In this context, nephrologists may also need to be familiar with the neurological problems that almost invariably develop in the patients with renal failure in order to assure their optimal management. We feel that an early and accurate diagnosis as well as awareness of spontaneous spinal epidural hematoma remains a challenge for relevant physicians, despite the steady increase in the cumulative number of publications on this topic [2,5,12,13,19]. Thus, we strongly recommend the accumulation of more cases similar to our own to delineate the nature of this pathology more precisely. Alternatively, or in addition, we believe that this condition, especially disease associated with hemiparesis with or without slurred speech, should be included in the list of stroke mimics [2,16-21,27], thereby leading to a high index of suspicion, prompt recognition and immediate intervention, which are essential to reduce or prevent major morbidity from the disease [24].

References

[1] Aminoff MJ. Weakness and Paralysis. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J. (eds.) Harrison's Principles of Internal Medicine. 18th ed. New York: Mc Graw Hill; 2012. p181-186.

[2] Akimoto T, Yamada T, Shinoda S, Asano Y, Nagata D. Spontaneous spinal epidural hematoma as a potentially important stroke mimic. J Cent Nerv Syst Dis. 2014; 6:15-20.

[3] James MA. Use of the Medical Research Council muscle strength grading systemin the upper extremity. J Hand Surg Am. 2007;32(2):154-156.

[4] Alexiadou-Rudolf C, Ernestus RI, Nanassis K, Lanfermann H, Klug N. Acute nontraumatic spinal epidural hematomas. An important differential diagnosis inspinal emergencies. Spine. 1998; 23 (16): 1810-1813.

[5] Liu Z, Jiao Q, Xu J, Wang X, Li S, You C. Spontaneous spinal epidural hematoma: analysis of 23 cases. Surg Neurol. 2008; 69 (3): 253-260

[6] Galbusera M, Remuzzi G, Boccardo P. Treatment of bleeding in dialysis patients. Semin Dial. 2009; 22 (3): 279-286.

[7] Takahashi K, Koiwa F, Tayama H, Satomi A, Akizawa T, Ideura T. A case of acute spontaneous epidural haematoma in a chronic renal failure patient undergoing haemodialysis: successful outcome with surgical management. Nephrol Dial Transplant. 1999; 14 (10): 2499-2501.

[8] Sung JM, Hsieh CC, Yu CY, Huang JJ. Acute spontaneous spinal epidural hematoma in a hemodialysis patient with a bleeding tendency. Nephron. 2002; 91 (2): 358-360.

[9] Ziyal IM, Aydin S, Inci S, Sahin A, Ozgen T. Multilevel acute spinal epidural hematoma in a patient with chronic renal failure--case report. Neurol Med Chir (Tokyo). 2003; 43 (8): 409-412.

[10] Deger SM, Emmez H, Bahadirli K, Kale A, Ebinc FA, Turkoglu M, Arinsoy T, Sindel S. A spontaneous spinal epidural hematoma in a hemodialysis patient: a rare entity. Intern Med. 2009; 48 (24): 2115-2118.

[11] Chang MO, Koh ES, Kim MJ, Chang YS, Chung S. Spontaneous spinal epidural hematoma. QJM. 2012; 105 (7): 705-706.

[12] Hibi A, Kasugai T, Kamiya K, Kamiya K, Kominato S, Ito C, Miura T, Koyama K. Successful Recovery from Spontaneous Spinal Epidural Hematoma in a Patient Undergoing Hemodialysis. Am J Case Rep. 2017; 18: 1357-1364.

[13] Liao CC, Lee ST, Hsu WC, Chen LR, Lui TN, Lee SC. Experience in the surgical management of spontaneous spinal epidural hematoma. J Neurosurg. 2004; 100 (1Suppl Spine): 38-45.

[14] Marinella MA, Barsan WG. Spontaneously resolving cervical epidural hematoma presenting with hemiparesis. Ann Emerg Med. 1996; 27 (4): 514-517.

[15] Anderson TJ, Donaldson IM. Spontaneous resolution of cervical spinal epidural haematoma. Postgrad Med J. 1989; 65: 488-490.

[16] Hsieh CF, Lin HJ, Chen KT, Foo NP, Te AL. Acute spontaneous cervical spinal epidural hematoma with hemiparesis as the initial presentation. Eur J Emerg Med. 2006; 13 (1): 36-38.

[17] Wang CC, Chang CH, Lin HJ, Lin KC, Kuo JR. Misdiagnosis of spontaneous cervical epidural haemorrhage. Eur Spine J. 2009; 18 (Suppl 2): 210-212.

[18] Son S, Kang DH, Choi DS, Kim SK, Lim BH, Choi NC. A case of spontaneous spinal epidural hematoma mimicking a stroke. Neurologist. 2012; 18 (1): 41-43.

[19] Patel R, Kumar A, Nishizawa K, Kumar N. Hemiparesis in spontaneous spinal epidural haematoma: a potential stroke imitator. BMJ Case Rep. 2018; 2018. pii: bcr-2017-222686.

[20] Sakamoto N, Yanaka K, Matsumaru Y, Nose T. Cervical epidural hematoma causing hemiparesis. Arch Neurol. 2003; 60 (5): 783.

[21] Li C, He R, Li X, Zhong Y, Ling L, Li F. Spontaneous spinal epidural hematoma mimicking transient ischemic attack: A case report. Medicine (Baltimore). 2017; 96 (49): e9007.

[22] Förster A, Griebe M, Wolf ME, Szabo K, Hennerici MG, Kern R. How to identify stroke mimics in patients eligible for intravenous thrombolysis? J Neurol. 2012; 259 (7): 1347-1353.

[23] Sarikaya H, Yilmaz M, Luft AR, Gantenbein AR. Different pattern of clinical deficits in stroke mimics treated with intravenous thrombolysis. Eur Neurol. 2012; 68 (6): 344-349.

[24] Phookan G, Lehman RA, Kuhlengel KR. Cervical spinal epidural haematoma: the double jeopardy. Ann Med. 1996; 28 (5): 407-411.

[25] Morgenstern LB, Lisabeth LD, Mecozzi AC, Smith MA, Longwell PJ, McFarling DA, Risser JM. A population-based study of acute stroke and TIA diagnosis. Neurology. 2004; 62 (6): 895-900.

[26] Brouns R, De Deyn PP. Neurological complications in renal failure: a review. Clin Neurol Neurosurg. 2004; 107 (1): 1-16.

[27] Schmidley JW, Mallenbaum S, Broyles K. Spinal epidural hematoma: an important stroke mimic. Acute Med. 2013; 12 (1): 36-39.

Citation: **Tetsu Akimoto, Daisuke Nagata.** *Spontaneous Spinal Epidural Hematoma Mimicking Ischemic Stroke: An Experience in a Patient Undergoing Hemodialysis. Archives of Nephrology. 2019; 2 (2): 11-15.*