Acute Spontaneous Spinal Epidural Hematoma Complicating Oral Anticoagulant Therapy

Oral Antikoagulan Tedaviyle İlişkili Akut Spontan Spinal Epidural Hematom Olgusu

Süleyman Türedi, Abdülkadir Gündüz, Süha Türkmen, Tevfik Patan, Asım Kalkan Karadeniz Teknik Üniversitesi Tıp Fakültesi, Acil Tıp Anabilim Dalı, Trabzon, Türkiye

Abstract

Bleeding is a common complication in patients receiving anticoagulant therapy, and is mostly seen in the gastrointestinal tract. Intraspinal hematoma is a unique and dangerous complication of anticoagulant therapy.

In our case, a 70 year - old male patient who had been using oral anticoagulants was admitted to the Emergency Department with nausea, neck pain and motor weakness in the right upper and lower extremities. His clinical findings resembled cerebral disease. Spontaneous spinal epidural hematoma (SEDH) was diagnosed as a result of cervical and thoracic magnetic resonance imaging. The patient received conservative treatment for a specific period of time and was discharged without any sequel. In this case, we describe the diagnostic and therapeutic approach towards acute intraspinal hematoma occurring as a complication of anticoagulant therapy.

Key words: Anticoagulant therapy, spinal epidural hematoma, intraspinal hematoma

Received: 12.01.2009 **Accepted:** 17.03.2009

Özet

Kanama antikoagulan tedavinin yaygın bir komplikasyonudur ve sıklıkla gastroeintestinal kanalda meydana gelir. Intraspinal hematom antikoagulan tedavinin nadir ve tehlikeli bir komplikasyonudur.

Vakamızda; oral antikoagulan tedavi kullanan 70 yaşında erkek hasta acil servise bulantı, boyun ağrısı, sağ alt ve üst ekstremitelerde kuvvet kaybı şikayeti ile başvurdu. Servikal ve torakal manyetik rezonans inceleme sonucunda spontan spinal epidural hematom (SEDH) tanısı koyuldu. Hasta konservatif tedavi ile izlendi ve sekelsiz olarak taburcu edildi.

Bu vaka sunumunda, antikoagulan tedavinin bir komplikasyonu olarak görülen akut intraspinal hematom vakasına tanı ve tedavi yaklaşımını sunduk.

Anahtar kelimeler: Antikoagulan tedavi, Spinal epidural hematom, intraspinal hematom

Alındığı Tarih: 12.01.2009 Kabul Tarihi: 17.03.2009

Introduction

Spontaneous spinal epidural hematoma (SEDH) is a rare condition requiring urgent diagnosis (1). Many cases have been reported in the literature, and there are diverse theories about its etiology. SEDH may be primary or secondary. The majority of cases are secondary to other underlying causes, such as trauma, anticoagulant therapy, vascular anomalies, blood dyscrasia, and epidural anesthesia. The mainstay of treatment is urgent surgical decompression and evacuation of the hematoma via laminectomy.

There are only a few reports of successful conservative treatment in this condition in which symptoms and signs have resolved without surgery. We report a patient who had spontaneous SEDH complicated with oral anticoagulant therapy and who was treated conservatively with a good neurological outcome. In the Emergency Department, spontaneous SEDH should be considered as one of the

differential diagnoses of cord compression once more common causes have been excluded (2).

Case report

A 70-year-old man with a history of coronary bypass-graft surgery who had been taking 10 mg of warfarin sodium (Coumadin) daily for nine months suddenly developed neck and back pain, and three days later he was admitted with right lower extremity weakness, paraesthesia and nausea. There was no history of recent head or neck trauma, and he was taking no other medications. There was no family history of any significant illness. His previous prothrombin times (PTs) were within the therapeutic range.

The patient's vital signs and mental functions were normal, and his cranial nerves were intact. There was motor weakness (4/5 power) of the upper right extremity and also hyporeflexia and motor weak-

ness (1/5 power) of the lower right extremity. Vibration and position sensations were normal. There were no signs of meningeal irritation. Findings from the rest of the examination were normal.

Laboratory test results were as follows; white blood cell count, 14.6 X 10° /L; hemoglobin level, 11 g /dL; platelet count, 247 X 10° /L; total bilirubin level, 1.4 mg/dL(control, 0-1.2); albumin level, 3.4 g/dL (control, 3,5-5,5); alkaline phosphates level, 75 U/L (reference range, 30-115 U/L); alanine aminotransferase level, 34 U/L (reference range, 1-40 U/L). The remaining biochemistry values were normal. PT level was 46 seconds (control, 11-15.5 seconds), international normalized ratio (INR) was 5.47 (therapeutic range, 2.0-3.0), and partial thromboplastin time was 54.4 seconds (control, 26-35 seconds). Radiographs of the cervical spine showed no abnormalities.

The patient was monitored with a suspicion of central nervous system bleeding in the light of the clinical and laboratory values. Following a cerebral CT scan a subcortical hypodense lesion (infarct?) was determined in the bilateral occipital lobes. However, considering the incompatibility between the CT finding and patient's clinical findings, emergency spinal magnetic resonance imaging (MRI) was performed due to a suspicion of spinal hematoma. The MRI revealed a posteriolaterally based epidural hematoma through the cervical and thoracicl vertebral colon in the spinal channel (Figures 1, 2).

Anticoagulation was reversed promptly with the administration of fresh frozen plasma (20 ml/kg) for 10 days and Vitamin K (1x10 mg, i.v.) for 10 days. In the light of the results of the MRI, neurosurgical opinions were sought. The brain surgeon did not consider operating, due to the time of commencement and clinical nature of the patient's symptoms. The patient was managed with conservative treatment. Meanwhile, recovery took place and neurological findings were improving. On the 15th day, the patient was discharged from hospital with motor weakness (4/5 power) of the lower right extremity.

Discussion

Spontaneous SEDH is a rare condition that has been reported to occur in different age-groups, from pediatric patients to the elderl (2). The dorsal epidural space of the thoracic spine is the most common site of intraspinal hemorrhage (3). This is also true for intraspinal hemorrhage associated with anticoagulant drug use.

In addition to the use of anticoagulant agents, these hematomas are also associated with bleeding diathesis, antiplatelet therapy, trauma, straining; vascular malformations, lumbar puncture, disc herniation, Paget's disease of bone, Valsalva maneuver, and possibly hypertension (4-11). In 40%-50% of cases of spinal epidural haematoma, no etiological causes can be identified (11). In this case, anticoagulant therapy was a predisposing factor.

Warfarin acts by inhibiting the synthesis of vitamin K dependent clotting factors. The degree of depression depends on the dosage administered (12).

MR imaging is considered to be the technique of choice for diagnosis (8). Signal characteristics of subacute and chronic SEDH have been described (4), but a few series have focused on the MR imaging features of acute SEDH within the first 48 hours of presentation, when management decisions are critical (13). In this case, MRI was the most important and useful method.

The most common presentation is an acute onset of severe local and radicular spinal pain, rapidly followed by motor and sensory deficits below the level of the hematoma and bowel and bladder

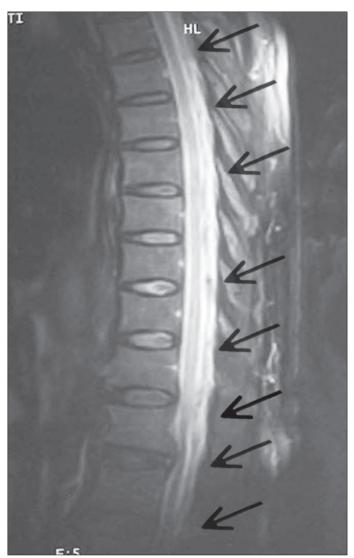


Figure 1. Sagittal T2-weighted image showing spinal epidural hematoma



Figure 2. Axial T2-weighted image showing the hyper intense lesion compressing the cord

dysfunction. However, there are also anectodal reports of painless epidural hematomas (14).

Outcome after surgery is somewhat variable. Several authors recommend early decompressive surgery and suggest that if the operation is performed before interference with the blood supply to the cord occurs, the prognosis is favorable (15). Neurological recovery after surgery varies with the severity of the preoperative impairment and the interval between presentation and surgery (15).

Foo and Rossier reviewed the outcome of 158 cases of SEDH treated surgically. Ninety-five percent opatients who had incomplete sensorimotor deficit, in contrast to 45.3% of patients with complete deficit, returned to baseline neurological function after surgery. Surgical outcome is more favorable if the SEDH is located in the lumbosacral region, compared with the thoracic region, and is localized to one bone level (16). Because there is a tendency towards neurologic progression, most patients are treated by prompt decompressive surgery within 36 hours. Therefore, reliable diagnostic features within the acute (≤48 hours) phase are necessary for successful surgery, when indicated (15).

There are an increasing number of reports, however, describing the resolution of SEDH using nonoperative management (4). In our patient, severe cord compression was revealed by the MRI, but no sensory deficits were seen. The SEDH in this case was extended over cervicothoracic vertebral levels.

For selected patients with incomplete and non-progressing deficits, conservative management may be possible. Surgery is nevertheless indicated for patients with progressive neurology and complete cord compression (2).

There have been considerable delays in intervention for many patients because of delays in reversing anticoagulation or, as in our patient, failure to recognize this complication immediately at the onset of symptoms. These delays should be avoided at all costs because recovery seems to be determined by the neurological status at the time of surgery. Primary care physicians and internists need to be aware of this complication because they are most likely to encounter these patients before specialists, especially with the increasing use of anticoagulation for common medical problems such as venous thrombosis and chronic atrial fibrillation (14).

Intraspinal bleeding must be immediately suspected in any patient taking anticoagulant drugs who develops the aforementioned symptoms. Anticoagulation should be reversed immediately with fresh frozen plasma and intravenous vitamin K administration, and corticosteroid therapy may be initiated. The MRI appearance of intraspinal hematomas varies depending on many factors, the age of the hematoma being the most important. If MRI is unavailable immediately or is contraindicated, a computed tomographic scan can be performed. It is difficult to diagnose in the Emergency Department

due to other intracranial incidents being encountered. However, in the event of lateralized findings, especially in patients receiving anticoagulant therapy, it is vital to consider intraspinal hematoma and decide on a treatment plan (surgical or conservative). History, clinical findings and MRI play a significant role in diagnosis.

Conflict of Interest

No conflict of interest is declared by the authors.

References

- Melanie BF, Amar SS, Robert LW. Acute Spontaneous Spinal Epidural Hematomas. AJNR. August . 1999; 20: 1365-72.
- Kong JKF,Mak KH. Spontaneous spinal epidural haematoma-an unusual cause of spinal cord compression. Hong Kong Medical Journal 2003; 9: 55-7
- Patel H, Boaz JC, Phillips JP, Garq BP. Spontaneous spinal epidural hematoma in children. Pediatr Neurol 1998; 19: 302-7.
- Holtas S, Heiling M, Lönntoft M. Spontaneous spinal epidural hematoma. Findings at MR imaging and clinical correlation. Radiology 1996; 199: 409-33.
- Groen R, Ponssen H. The spontaneous spinal epidural hematoma. A study of the etiology. J Neurol Sci 1990; 98: 121-38.
- Lee K, McWhorter J, Angelo J. Spinal epidural hematoma associated with Paget's disease. Surg Neurol 1988; 30: 131-4.
- Brawn L, Bergval U, Davies-Jones G. Spontaneous spinal epidural haematoma with spontaneous resolution. Postgrad Med J 1981; 62: 885-7.
- Gundry C, Heithoff K. Epidural hematoma of the lumbar spine.18 surgically confirmed cases. Radiology 1993; 187: 427-31.
- Caldemeyer K, Mocharla R, Moran C, Smith R. Gadolinium enhancement in the center of a spinal epidural hematoma in a hemophiliac. J Comput Assist Tomogr 1993; 17: 321-3.
- Muhonen M, Piper J, Moore S, Menezes A. Cervical epidural hematoma secondary to an extradural vascular malformation in an infant. Case report. Neurosurgery 1995; 36: 585-8.
- Duffill J, Sparrow OC, Millar J, Barker CS. Can spontaneous spinal epidural haematoma be managed safely without operation? A report of four cases. J Neurol Neurosurg Psychiatry 2000; 69: 816-9.
- Allison EJ, McKinney TJ, Langenberg JN. Spinal epidural haematoma as a result of warfarin/fluconazole drug interaction. Eur J of Emerg Med 2002; 9: 175-7.
- 13. Shen CC, Wang YC, Yang DY, Wang FH, Shen BH. Brown-sequard syndrome associated with Horner's syndrome in cervical epidural hematoma. Spine 1995; 20: 244-7.
- Vinod AP, Kalapura T, Pincus M, Baskharoun R. Intraspinal hemorrhage complicating oral anticoagulant therapy. Arch Intern Med 2000; 160: 237-40.
- Foo D, Rossier A. Preoperative neurological status in predicting surgical outcome of spinal epidural hematomas. Surg Neurol 1981; 15: 389-401.
- 16. Priest W. Epidural hemorrhage due to hemophilia. Lancet 1935; 2: 1289-91.