

# Spinal cord infarction caused by sacral canal epidural steroid injection

## A case report

Gang Wang, MD<sup>a</sup>, Jing Liang, MD<sup>b</sup>, Zishan Jia, MD<sup>a,\*</sup>, Lei Wan, BS<sup>a</sup>, Mingxia Yang, MD<sup>c</sup>

### Abstract

**Rationale:** Spinal cord infarction is one of the complications of epidural steroid injections (ESIs), but has only been reported in cervical vertebra by transforaminal injection and lumbar vertebra by transforaminal injection; and up to now, there is no reporting about spinal cord infarction caused by caudal injection. Here, we report a case.

**Patient concerns:** A 52-year-old man was admitted to our hospital. He was diagnosed as lumbar disc herniation in other hospital, and the patient suffered bilateral lower limb motor and sensory disorders after administration of caudal ESI.

**Diagnosis:** Spinal cord infarction, tethered cord syndrome (TCS), and acute myelitis.

**Interventions:** High doses of hormones, gamma globulin impact therapy, and rehabilitation were performed.

**Outcomes:** The patient's condition was stable when he discharged from the hospital after 20 days of treatment. Discharge status: grade 0 of bilateral lower limbs muscle strength, inability to urinate and defecate by himself, slightly decreased touch, and needling sensation below the umbilical plane.

**Lessons:** When patients are diagnosed with lumbar disc herniation and need to receive invasive treatments, magnetic resonance imaging (MRI) should be performed before the invasive procedures.

**Abbreviations:** CSF = cerebro-spinal fluid, ESI = epidural steroid injection, MRI = magnetic resonance imaging, TCS = tethered cord syndrome.

**Keywords:** epidural steroid injection, sacral canal, spinal cord infarction, tethered cord syndrome

## 1. Introduction

Injecting certain amount of steroid to the spinal cord via epidural space to treat lumbar pain or lower extremity pain is referred to as epidural steroid injection (ESI). This operation is suitable for the treatment of lumbar disc herniation, lumbar spinal stenosis, stealth spina bifida, caudal plexus neuralgia, and lumbosacral radiculitis. Interlaminar injection, transforaminal injection, and caudal injection are common ways for epidural drug injections. Spinal cord infarction is one of the complications of ESIs, but it had only been reported in patients by transforaminal injection via cervical vertebra<sup>[1,2]</sup> and by transforaminal injection via lumbar

vertebra.<sup>[3,4]</sup> Up to now, no case had been reported to have spinal cord infarction caused by caudal injection. Herein, we reported a case in which the patient suffered from bilateral lower limb movement and sensory dysfunction following ESI via sacral canal and was diagnosed with spinal cord infarction.

## 2. Case report

The patient was a 52-year-old married male, who was diagnosed with “lumbar disc herniation, lumbocrural pain” because of intermittent waist and bilateral lower limbs pain for 4 years, but little was known about the diagnosis and the patient was not performed lumbar magnetic resonance imaging (MRI) examination. On September 6, 2016, the patient was performed epidural injection via sacral canal by a pain management doctor without image guidance. According to the patient's statement, the injection drug was 30mL normal saline with 35mg 0.5% lidocaine hydrochloride and 100mg prednisolone. After injection, the patient immediately suffered from severe abdominal pain and distending pain of bilateral lower limbs, the pain lasted for about 2 minutes and then relieved. About 10 minutes later, the patient lost pain sensation below the navel, felt numbness of bilateral lower limbs, and could not move. Then he was unable to relieve himself. After receiving relevant treatment, including providing nutrients for the nerves and replacing cerebro-spinal fluid (CSF) for 3 days in the local hospital, there was no significant improvement. The patient was, therefore, admitted to People's Liberation Army General Hospital on September 9, 2016. There was no specific information available pertaining to any other medical history.

Editor: Dennis Enix.

GW and JL contributed equally to this work.

The authors have no funding and conflicts of interest to disclose.

<sup>a</sup> Department of Rehabilitation Medicine, Chinese PLA General Hospital, Beijing,

<sup>b</sup> Department of Rehabilitation Medicine, Special Caring Hospital of Hebei

Province, Shijiazhuang, <sup>c</sup> Rehabilitation Department of Chinese PLA 163 Hospital, Changsha, Hunan, China.

\* Correspondence: Zishan Jia, Department of Rehabilitation Medicine, Chinese PLA General Hospital, Beijing 100853, China (e-mail: jzs301@sina.com).

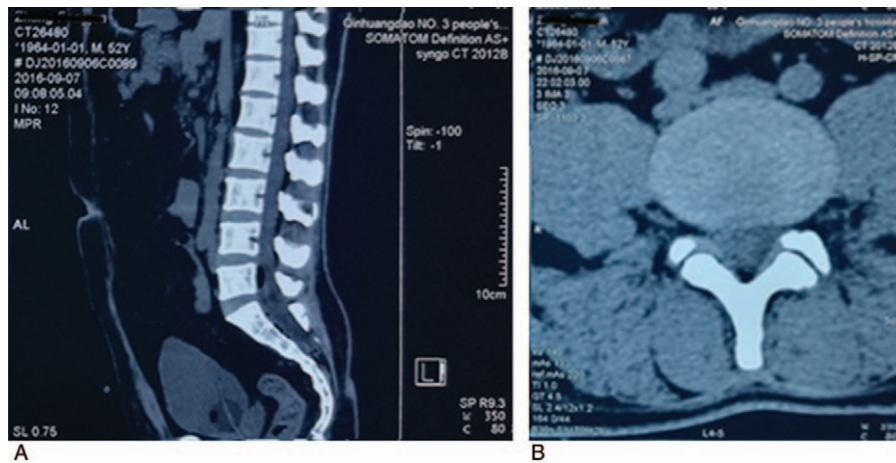
Copyright © 2018 the Author(s). Published by Wolters Kluwer Health, Inc.

This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Medicine (2018) 97:11(e0111)

Received: 4 June 2017 / Received in final form: 16 December 2017 / Accepted: 20 February 2018

<http://dx.doi.org/10.1097/MD.0000000000010111>



**Figure 1.** (A) Sagittal and (B) axial CT showing L4-5 disc herniation and intraspinal gas (1 day after injection). CT=computed tomography.

After admission to our hospital, the patient was in a normal state of mind with normal physical strength and normal sleep, but he complained about abdominal distension and poor appetite. No obvious change of body weight was observed. The patient had no defecation for 4 days, and as he could not urinate by himself and a catheter was kept. Physical examination showed that he was in clear mind; no abnormality in lungs, heart, and abdomen was observed. The spine physiological curvature was normal and there was no limbs edema. The muscular strength and tone of bilateral upper limbs was normal, but the muscular strength of bilateral lower limbs was grade 0, and the muscular tone of bilateral lower limbs decreased. His pain and temperature sensation below the navel decreased, but the proprioception and vibration sensation, the bilateral biceps and triceps reflex, and the knee and Achilles tendon reflex were all normal, and the bilateral Babinski sign was also negative.

Images from the lumbar computed tomography and MRI examinations before admission have been presented in Figs. 1–3. Images from the lumbar MRI re-examination and thoracic vertebrae MRI after admission were shown in Fig. 4.

Results of lumbar puncture revealed an initial pressure of 110 mm H<sub>2</sub>O and a terminal pressure of 90 mm H<sub>2</sub>O. General examination of the CSF showed a yellow fluid with a total cell number of 10,800 \* 10<sup>6</sup>/L, white blood cell count of 12 \* 10<sup>6</sup>/L, positive albumin qualitative test, glucose level of 2.1 mmol/L, chloride concentration of 122.3 mmol/L, and protein concentration of 1450.0 mg/L.

According to the medical history and imaging findings, we make the diagnosis: spinal cord infarction, tethered cord syndrome (TCS), and acute myelitis.

The results of CSF examination also accord with the diagnosis of spinal cord infarction and acute myelitis.

After consulting with neurologists, we decided to offer the patient a symptomatic treatment, which included improving circulation, providing nutrients for the nerves and high doses of hormones, and gamma globulin impact therapy. On September 18, 2016, a lumbar vertebra re-examination revealed TCS, abnormal signals within the spinal cord, spinal cord demyelination pathology with an increased scope as compared to before, and L4-5 disc herniation (Fig. 5). The bilateral lower limbs sensation gradually improved, but still accompanied with bilateral lower limb swelling, numbness, and formication. The patient continued to receive steroid therapy and the dosage was

gradually reduced. In addition, the patient was given extremity motor function rehabilitation treatment.

After 20 days of hospitalization, the patient's condition was found to be stable and was discharged from the hospital. Discharge status: grade 0 of bilateral lower limbs muscle strength, normal muscle tension, inability to urinate and defecate by himself, slightly decreased touch and needling sensation below the umbilical plane, normal proprioception, and vibration sensation.

### 3. Discussion

ESI is often used for the treatment of acute lumbar disc herniation with a positive effect. Additionally, they are administered to treat



**Figure 2.** Coronal view CT showing bilateral L4 nerve root set out and downlink from the L3 level (1 day after injection). CT=computed tomography.



**Figure 3.** Sagittal T2 weighted MRI showing spinal cord tail conus medullaris located at L4 level and thickened spinal tail (1 day after injection). MRI = magnetic resonance imaging.

neurogenic claudication and mechanical spine-related pain. Literature has shown that the use of ESI has significantly increased since the year 2000<sup>[5]</sup>; and in the United States, more than 9 million patients are known to receive ESI therapy annually.<sup>[6]</sup> The most common ways to accomplish epidural injections are interlaminar injection, transforaminal injection, and caudal injection. Currently, the caudal ESI is widely used owing to its simple operation, lower risk of dura puncture, and safer procedure compared to other methods of injections.<sup>[7]</sup>

In 2014, Food and Drug Administration issued a warning that ESI could cause rare but serious complications, including blindness, stroke, paralysis, and death. These warnings were concluded mainly based on certain reports in which the patients received transforaminal epidural injection with steroids containing particles.<sup>[8]</sup>

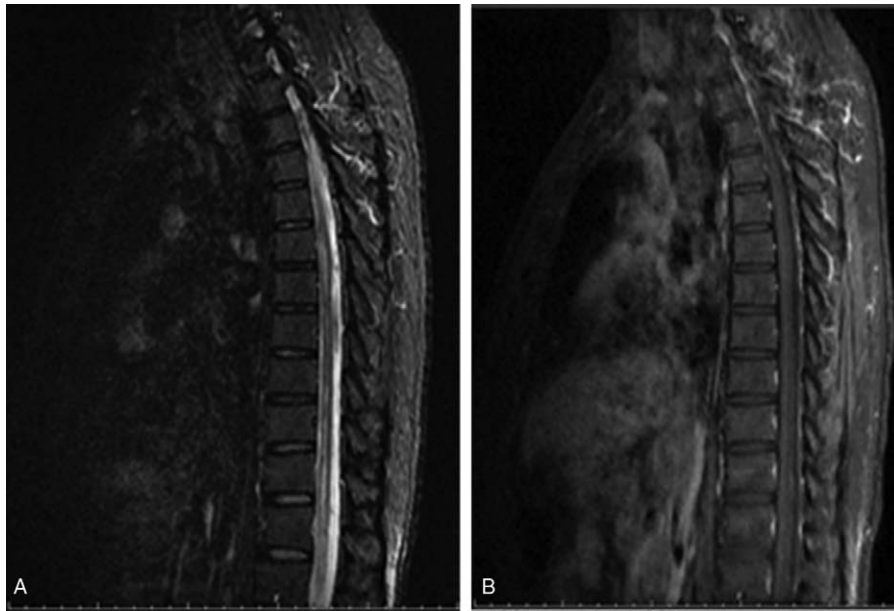
Studies in the past have suggested that betamethasone, methylprednisolone, and triamcinolone, containing certain particles or the substances within them converging into particles, might be bigger in size than the red blood cells. If this kind of steroid is injected into radicular artery, these particles may formulate emboli in arteries, arterioles, or arterial capillary.<sup>[9]</sup> Animal studies have shown that when the vertebral arteries of

anesthetized pigs are injected with steroids, the consciousness of the pigs injected with steroids containing particles fail to recover and their clinical symptoms are consistent with changes in MRI and pathology. In contrast, the pigs injected with steroids containing no particles showed no obvious symptoms of nerve damage, no radiographic abnormalities, and no abnormalities after autopsy.<sup>[10]</sup> However, there were reports on patients who manifested symptoms of spinal cord infarction after receiving epidural injection with dexamethasone phosphate sodium containing nonparticulate steroids.<sup>[11]</sup>

Clinical symptoms of spinal cord infarction mainly depend on the location of the infarction; many patients suffer back pain that radiates to the sacrococcygeal region, rectum, and bladder. Two-thirds of blood supply of anterior spinal cord is supplied by anterior spinal arteries, and the remaining one-third is supplied by posterior spinal arteries. Spinal cord infarction often occurs in anterior spinal arteries, as they lack communicating branches. The initial clinical symptoms of anterior spinal arteries infarction usually begun with flaccid paralysis, reduced or disappeared flaccid paralysis, reduced pain and temperature sensation distal to the level of injury, followed by increased muscular tension, activated tendon reflex, and possible positive Babinski sign.<sup>[12]</sup> Proprioception and vibration sensation are usually not affected as they are transmitted by the spinal posterior funiculus. However, the spinal spinothalamic tract transmits the pain and temperature sensation, which is usually located at a more front, outer, and approachable corticospinal tract, and dominates the voluntary movements.<sup>[13]</sup> The patient in the present report showed clinical symptoms of anterior spinal arteries infarction. MRI proved to be highly efficient in diagnosing spinal cord infarction owing to its better sensitivity. Therefore, the changes in spinal cord signals and spinal cord edema could be observed during the initial hours of the disease,<sup>[14]</sup> and long T1 and T2 signals were gradually monitored.<sup>[15]</sup>

Previous reports have shown that spinal cord infarctions only occur in patients with transforaminal ESI; however, there are no published reports on spinal cord infarction caused by caudal injections. Interestingly, the spinal cord infarction in the present case may be associated with TCS.

TCS is caused by a variety of etiology: the spinal cord and nerves are fixed by inelastic structure; during the growth and development process, spinal conus medullaris is fixed to a lower position, leading to spinal cord and nerves damage, thereby resulting in a series of neurological dysfunction syndromes.<sup>[16]</sup> TCS was first described in 1976 by Hoffman et al.<sup>[17]</sup> If the spinal conus medullaris is located below L2 level, then a diagnosis of low position spinal conus medullaris could be drawn. But TCS is usually accompanied with some complaints and clinical signs. In other words, the low position spinal conus medullaris is a radiologic diagnosis, while TCS is a clinical diagnosis. It is estimated that the incidence of TCS is about 0.017% to 0.639% worldwide.<sup>[18]</sup> Although TCS is generally considered as a childhood disease, it could also occur in adults.<sup>[19–21]</sup> The symptoms of TCS vary, ranging from no symptoms to paraplegia.<sup>[22,23]</sup> Adult TCS is generally diagnosed when patients suffer back pain and/or lower limbs pain. Most adult TCS patients live a normal life and there are no particular complaints as they have ignored their own innate disease since childhood. Compared to patients diagnosed at childhood, they exhibit more tolerance to certain symptoms.<sup>[24]</sup> When the spinal conus medullaris and nerve roots are affected, the patients suffer back pain, lower limbs weakness, foot deformities, spinal scoliosis, missing sensation, or bladder or rectum dysfunction.<sup>[22,23,25–27]</sup>



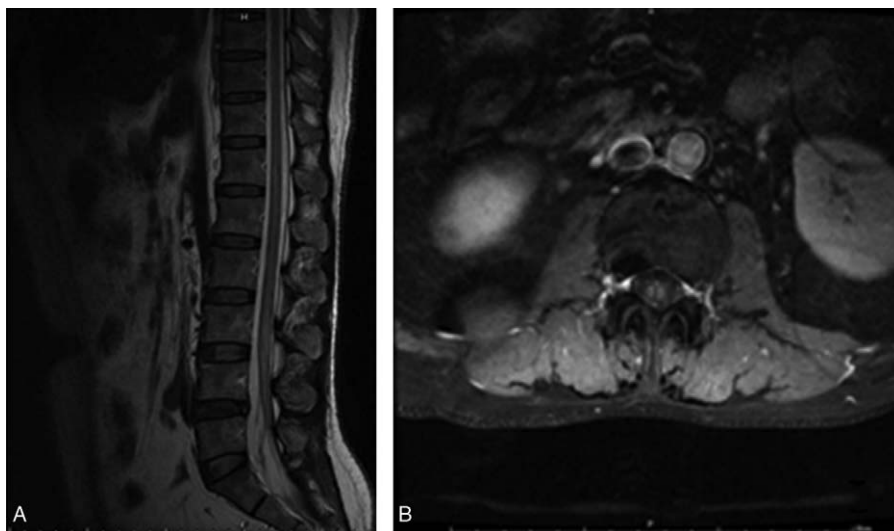
**Figure 4.** (A) Sagittal T2- and (B) T1-weighted MRI showing small flake long T1 and T2 signals at T5 to T12. No enhancement was observed when enhanced. Thoracic spinal cord demyelination was observed (2 days after injection). MRI = magnetic resonance imaging.

The neurologic dysfunction occurring in adulthood is usually irreversible.

When foreign substances are injected into the spinal cord arteries, both thromboembolism or direct oppression can cause spinal cord infarction.<sup>[28]</sup> There may be 2 reasons attributing to spinal cord infarction in any patient: tethered spinal cord, wherein the bottom of thecal sac is too low and is pierced by the deeply entered needle during caudal injection, thus causing the entrance of drugs into thecal sac and leading to spinal cord infarction; the space of the bottom of the vertebral tube is too small due to the low position spinal cord, and after injecting 32 mL liquid, the local pressure increases, thus resulting in spinal cord infarction.

The patient in the current report was diagnosed with lumbar disc herniation syndrome for back pain and lower limb pain, but the patient received caudal ESI in the absence of further confirmation by MRI, thus leading to a rare spinal cord infarction, which eventually resulted in bilateral lower limb paralysis. Acute myelitis is divided into 3 types, one of which is demyelinating myelitis, which can cause demyelination in the spinal cord. This patient suffered secondary inflammatory spinal cord demyelination and was influenced up to the T5 level.

Given the wide use of ESI and the potential serious complications associated with them, the following recommendations may serve as important considerations:



**Figure 5.** (A) Sagittal T2- and (B) axial T1-weighted MRI spinal cord tail conus medullaris located at L4 level; thickened spinal tail; multiple patchy long T2 signal; short T2 signal around the central canal; and L4-5 disc herniation. Mild-moderate enhancement was observed with enhanced scan (12 days after injection). MRI = magnetic resonance imaging.

- (1) When patients are diagnosed with lumbar disc herniation and need to receive invasive treatments, MRI should be performed before the invasive procedures.
- (2) Steroids that do not contain large particles such as dexamethasone sodium phosphate must be used for injection.
- (3) Operations should be carried out under the guidance of imaging (computed tomography, ultrasound, or X-rays).
- (4) The total liquid volume for caudal epidural drug injection should not be more than 20 mL.
- (5) It should be examined whether there is blood or CSF by pulling back before the injection.
- (6) Local anesthetic test should be performed if steroids containing larger particles are being used for injection.
- (7) There have been no published reports on spinal cord infarction caused by caudal injections. Therefore, it is believable that canal ESI has lower risk of spinal cord infarction as compared to transforaminal ESI, and the spinal cord infarction in the present case had much to do with TCS.

## Reference

- [1] Moon J, Kwon HM. Spinal cord infarction after cervical transforaminal epidural steroid injection: case report and literature review. *Case Rep Neurol* 2017;9:1–5.
- [2] Muro K, O'Shaughnessy B, Ganju A. Infarction of the cervical spinal cord following multilevel transforaminal epidural steroid injection: case report and review of the literature. *J Spinal Cord Med* 2007;30:385–8.
- [3] Quintero N, Laffont I, Bouhmid L, et al. [Transforaminal epidural steroid injection and paraplegia: case report and bibliographic review]. *Ann Readapt Med Phys* 2006;49:242–7.
- [4] Kennedy DJ, Dreyfuss P, Aprill CN, et al. Paraplegia following image-guided transforaminal lumbar spine epidural steroid injection: two case reports. *Pain Med* 2009;10:1389–94.
- [5] Manchikanti L, Pampati V, Falco FJ, et al. Growth of spinal interventional pain management techniques: analysis of utilization trends and Medicare expenditures 2000 to 2008. *Spine* 2013;38:157–68.
- [6] Bicket MC, Chakravarthy K, Chang D, et al. Epidural steroid injections: an updated review on recent trends in safety and complications. *Pain Manag* 2015;5:129–46.
- [7] Manchikanti L, Malla Y, Wargo BW, et al. A prospective evaluation of complications of 10,000 fluoroscopically directed epidural injections. *Pain Physician* 2012;15:131–40.
- [8] Feeley IH, Healy EF, Noel J, et al. Particulate and non-particulate steroids in spinal epidurals: a systematic review and meta-analysis. *Eur Spine J* 2017;26:336–44.
- [9] Derby R, Lee SH, Date ES, et al. Size and aggregation of corticosteroids used for epidural injections. *Pain Med* 2008;9:227–34.
- [10] Okubadejo GO, Talcott MR, Schmidt RE, et al. Perils of intravascular methylprednisolone injection into the vertebral artery. An animal study. *J Bone Joint Surg Am* 2008;90:1932–8.
- [11] Gharibo CG, Fakhry M, Diwan S, et al. Conus medullaris infarction after a right L4 transforaminal epidural steroid injection using dexamethasone. *Pain Physician* 2016;19:E1211–4.
- [12] Caplan LR. *Caplan LR. Spinal-cord strokes. Stroke: A Clinical Approach* 2nd ed Butterworth-Heinemann, Boston, MA:1993;487–96.
- [13] Davidoff RA. The dorsal columns. *Neurology* 1989;39:1377–85.
- [14] Elksnis SM, Hogg JP, Cunningham ME. MR imaging of spontaneous spinal cord infarction. *J Comput Assist Tomogr* 1991;15:228–32.
- [15] Yamamoto N, Miyagi R, Sakai T, et al. Magnetic resonance imaging and diffusion-weighted imaging findings in posterior spinal cord infarction: case report. *J Orthop Sci* 2017;22:1151–4.
- [16] Yamada S, Won DJ, Pezeshkpour G, et al. Pathophysiology of tethered cord syndrome and similar complex disorders. *Neurosurg Focus* 2007;23:E6.
- [17] Hoffman HJ, Hendrick EB, Humphreys RP. The tethered spinal cord: its protean manifestations, diagnosis and surgical correction. *Childs Brain* 1976;2:145–55.
- [18] Bowman RM, McLone DG, Grant JA, et al. Spina bifida outcome: a 25-year prospective. *Pediatr Neurosurg* 2001;34:114–20.
- [19] Gokay H, Barlas O, Heggul KT, et al. Tethered cord in the adult mimicking the lumbar disc syndrome: report of two cases. *Surg Neurol* 1993;39:440–2.
- [20] Hesselink JW, Tans JT, Hoogland PH. Diastematomyelia presenting in two male adults with low back pain. *Clinl Neurol Neurosurg* 1986;88:223–6.
- [21] Iskandar BJ, Fulmer BB, Hadley MN, et al. Congenital tethered spinal cord syndrome in adults. *J Neurosurg* 1998;88:958–61.
- [22] Kokubun S, Ozawa H, Aizawa T, et al. Spine-shortening osteotomy for patients with tethered cord syndrome caused by lipomyelomeningocele. *J Neurosurg Spine* 2011;15:21–7.
- [23] Phuong LK, Schoeberl KA, Raffel C. Natural history of tethered cord in patients with meningomyelocele. *Neurosurgery* 2002;50:989–93. discussion 993–5.
- [24] Abdallah A, Emel E, Abdallah BG, et al. Factors affecting the surgical outcomes of tethered cord syndrome in adults: a retrospective study. *Neurosurg Rev* 2018;41:229–39.
- [25] Fehlings MG, Arvin B. Recurrent tethered cord syndrome: a novel approach for a difficult surgical condition? *J Neurosurg Spine* 2009;10:275–6. author reply 276–7.
- [26] Lapsiwala SB, Iskandar BJ. The tethered cord syndrome in adults with spina bifida occulta. *Neurol Res* 2004;26:735–40.
- [27] Sofuoglu OE, Abdallah A, Emel E, et al. Management of tethered cord syndrome in adults: experience of 23 cases. *Turk Neurosurg* 2017;27:226–36.
- [28] Ludwig MA, Burns SP. Spinal cord infarction following cervical transforaminal epidural injection: a case report. *Spine* 2005;30:E266–8.