

Acute Paraplegia after General Anesthesia

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Abstract- Acute paraplegia is a rare but catastrophic complication of surgeries performed on aorta and corrective operations of vertebral column. Trauma to spinal cord after spinal anesthesia and ischemia of spinal cord also may lead to acute paraplegia. Acute paraplegia as a complication of general anesthesia in surgeries performed on sites other than aorta and vertebral column is very rare. Here we present a 56 year old woman with acute paraplegia due to spinal cord infarction after laparoscopic cholecystectomy under general anesthesia probably caused by atherosclerosis of feeding spinal arteries and ischemia of spinal cord after reduction of blood flow possibly due to hypotension during general anesthesia.

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Introduction

Paraplegia means severe weakness of both lower limbs and is one of the most common presentations of spinal cord disorder at thoracic level (1-3). Bilateral or asymmetric Babinski sign, sensory level below the neck and sphincteric dysfunction are other manifestations of spinal cord disease in this level (2,3). There are many etiologies for this disorder and the most common causes are demyelinating disorders, infectious disorders, vascular, hematologic and rheumatologic disorders (2-4). Also some cases are reported as a complication of surgery of aorta (5-7), kyphoscoliosis, thoracic surgery or surgery near or in relation to aorta (8,9). In most reports paraplegia was related to spinal or epidural anesthesia and cases with general anesthesia are quite rare in literature if present at all (10-12).

Case Report

A 56 years old female was referred for neurologic consultation by surgery department in March 2008. The patient's complaint was weakness of both lower limbs since previous night. After recovery from general anesthesia for laparoscopic cholecystectomy, the patient was paraplegic with urinary retention. Reflexes were absent in both lower limbs and Babinski sign was upward bilaterally. There was no definite sensory level

but a suspicious T10-T12 sensory level was detected after neurologic examinations. Examination of cranial nerves and upper limbs forces and reflexes were normal. The patient had a positive history of mild hypertension. She reported similar symptoms and signs of transient paraplegia 2 months earlier which was managed by an orthopedic surgeon. MRI was recommended at that time but the patient declined. She did not give any history of diabetes mellitus but had hyperglycemia at admission and diabetes mellitus was diagnosed. Mild hypertension was present and there was no other positive history for other diseases or drugs.

An emergent MRI was performed and neurosurgical consultation was done with three neurosurgeons. There was no obvious pathologic lesion in MRI of the spinal cord at thoracic and lumbosacral level (with and without contrast media) done in the first day (Figures 1 and 2). The patient refused anymore treatment or tests and said that her condition is temporary and she had the same condition a few months ago and would be normal very soon. She left hospital for Norooz ceremony and Holiday.

Five days later she was admitted to neurology department for management and rehabilitation of paraplegia. Patient's symptoms and signs were the same as before her voluntary discharge, with paraplegia (0/5) and urinary retention. She said that she would accept any treatment or test.



Figure 3. MRI with contrast from ischemic lesion (6 months later)



Figure 4. MRI with contrast (6 months later)

Work up to evaluate the possible causes of transverse myelitis was done on CSF and blood samples (Table 1). The patient received methylprednisolone 1000mg/day for 5 days and then 20 gram IVIG per day for 5 consecutive days.

The patient was hospitalized for rehabilitation for three months but the signs and symptoms changed a little and patient's force was 1/5 in distal of lower limbs. MRI of the spinal cord at thoracic and lumbosacral level was repeated after 6 months and there was obvious necrotic lesion in the spinal cord with some enhancement that mostly suggested ischemia of spinal cord at T11 to L1 (Figures 3-7). After 18 months of

rehabilitation she is able to walk with walker for at least 200 meters.

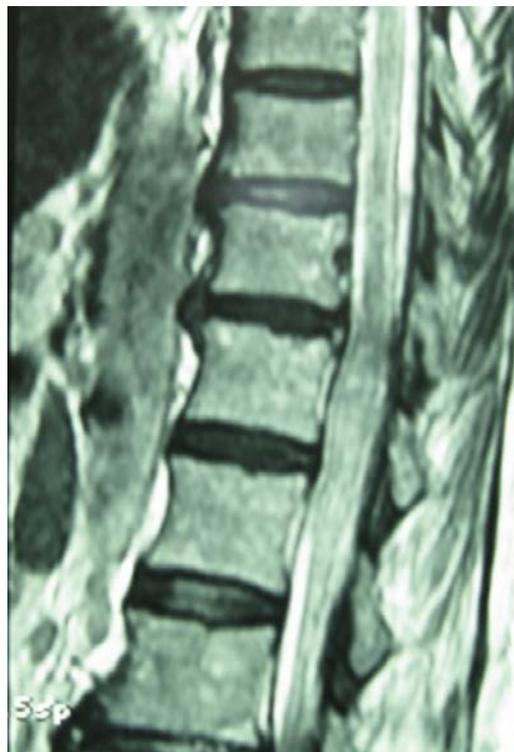


Figure 5. T2 MRI without contrast at thoracic level (above the lesion, 6 months later)

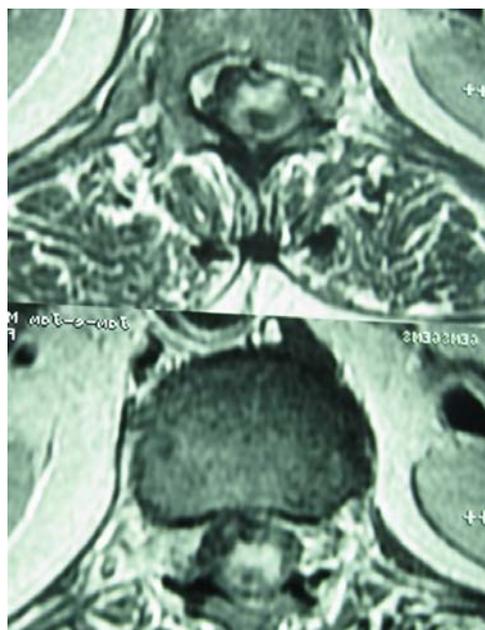


Figure 6. MRI with contrast at thoracic level (6 months later)



Figure 7. T2 MRI without contrast at cervical level(6 months later)

Discussion

Paraplegia due to spinal cord disease has many etiologies (4,13). Paraplegia after surgery is rare and mostly occurs after surgery of aortic aneurysm, surgery near or in relation with aorta or spinal cord, correction of kyphoscoliosis and direct or indirect trauma to the spinal cord due to spinal or epidural anesthesia. But there is no report after laparoscopic cholecystectomy with general anesthesia. In these cases the etiology of paraplegia is direct trauma to the cord or ischemia due to direct trauma to the spinal vasculature or indirect ischemia due to blood flow reduction (1,4). In our case the spinal cord infarction is obvious in MRI images. Its etiology can be related to hypotension and atherosclerosis of the spinal arteries due to diabetes mellitus and hypertension. Another possibility that must be considered is an anomaly of the cord structure such as an arteriovenous malformation (AVM) in the cord or dura.

In either etiology previous history of transient paraplegia is a risk factor for the patient and can be judged as a type of transient ischemic attack of the cord (TIA). So we can suggest that in the patients with previous history of transient paraplegia especially if

there is any risk of atherosclerosis, spinal cord ischemia must be considered a possibility and constant monitoring of blood pressure to prevent episodes of hypotension must be done during surgeries done under general anesthesia.

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