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INTRACRANIAL COMPLICATIONS OF LUMBAR SPINAL SURGERY

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ABSTRACT

In this study, we present the largest series composed of 10 patients, with different kinds of remote intracranial complications after lumbar spinal surgery. Remote intracranial events happened after lumbar spine surgery that were performed from 2002 to 2017 in senior author's practice were reviewed and ten patients were retrieved from the database with relevant clinical and radiological information. We have 10 patients (8 women and 2 men; mean age: 56±18.5 years; age range: 12–81 years) with remote intracranial events happened after lumbar spine surgery. Although the presenting symptoms may be mostly nonspecific, remote intracranial event should be suspected in any patient with intractable headach e, focal neurological deficits or unexplained deterioration of consciousness following spine surgery.

Key words: Spinal surgery, comlication, intracranial complications.

Level of Evidence: Retrospective clinical study, Level III.

INTRODUCTION

Lumbar spine surgery is related with various complications. Remote intracranial events after lumbar spine surgeries are relatively rare. These events usually happen due to intracranial pressure alterations caused by cerebrospinal fluid (CSF) loss following dural tear [1,18] or by peroperative systemic blood pressure changes [29,33]. These remote events may be classified into 6 groups: intracerebral hematoma (ICH), subdural/epidural hematoma (SDH/EDH), intraventricular/subarachnoid hemorrhage (IVH/SAH), cerebellar hemorrhage (CBH), cerebral venous sinus thrombosis (CVT) and others.

Etiology of these various remote complications remains unclear, yet evidence suggests that they are caused by excessive intra-opereative CSF loss and excessive post-operative suction by drains, which results in cerebral dehydration. Excessive cerebral dehydration may cause tearing of the bridging and pial veins [17,30,32]. Furthermore, CVT and hemorrhagic venous infarction may also occur in patients with pro-thrombotic states as CSF leakage indirectly contributes to volume loss in the cerebral sinuses [18,24]. Beside these, Liu et al. described pituitary apoplexy in a patient operated for lumbar fusion with anterior laparoscopic approach [23] and Choi et al. reported seizure subsequent to percutaneous endoscopic lumbar discectomy under local anesthesia [6].

In this study, we present one of the largest series composed of 10 patients, with different kinds of remote intracranial complications happened after lumbar spinal surgery. We also discuss presumed pathophysiology, differential diagnosis, and avoidance of remote intracranial complications after lumbar spinal surgery.

PATIENTS AND METHODS

Chart-review was conducted using protocols approved by the Committee for the Ethical Issues of Bahçeşehir

University, and affiliated hospitals. Remote intracranial events happened after lumbar spine surgery that were performed from 2002 to 2018 in senior author's practice were reviewed and ten of more than six thousand patients were retrived from the database with relevant clinical and radiological information.

Data regarding the following variables were looked for: demographics, clinical presentation, index spinal procedure, postoperative complications, presence/absence of drain placement, location of intracranial events, radiologic findings, treatment strategies, and outcome (Table-1).

RESULTS

There were 10 patients (8 women and 2 men; mean age: 56 ± 18.5 years; age range: 12–81 years) with remote intracranial events happened after lumbar spine surgery. All patients were symptomatic postoperatively. Onset of symptoms ranged from immediately after surgery to postoperative Day 5. Symptoms included headache (n=8), aphasia (n=2), seizure (n=3), visual problem (n=2) and altered mental status (n=3). Perioperative hemodynamic instability had not been documented for any patient. All patients underwent head CT, which revealed ICH (n=3):, SDH (n=2), IVH (n=1), SAH (n=2), CBH (n=3), CVT (n=1), intracranial tumor (n=1) and pituitary apoplexy (n=1). Five patients had more than one pathology. Seven patients were treated conservatively, while 3 patients required cranial neurosurgical intervention. Craniotomy was performed for the patient presenting with seizure who had been revealed to have concomitant intracranial tumor (n=1), microsurgical decompressive surgery was done via transphenoidal route for the patient with apoplexy (n=1) and ventriculostomy was placed temporarily to one of the patients with ICH to relieve hydrocephalus (n=1). Ultimately, 8 patients achieved a full recovery with minimal or no residual neurological deficit. Two patients with ICH died in the following weeks postoperatively one of whom had been declared as beeing brain dead on the 20th postoperative day, and the other patient had been discharged to a rehabilitation facility with some cognitive impairment and died due to pulmonary complications. Patients with ICH had the worst prognosis.

The first case who presented with low-back and bilateral lower extremity pain was a 51-year-old woman. She had neurological claudication. There was hypoesthesia on bilateral L3-L5 dermatomes. Lumbar MRI scans demonstrated spinal stenosis at multiple levels and minimal degenerative scoliosis. She underwent posterior laminectomies from L2 to L5 with L5-S1 discectomy, L5-S1 TLIF cage placement, posterior spinal fusion from T10 to S1 with instrumentation. A dural tear was identified at L4 level during the procedure. Primary closure was performed with 5-O silk, and dura sealant (Adherus®, HyperBranch Medical Technology, Inc, North Carolina). A hemovac drain was placed into the epidural space. Post-operative period was uneventful. On the fifth day, the patient readmitted to our clinic with severe back and leg pain. On physical examination, subcutaneous and paravertebral fluid collection were noted. CSF collection at the surgical site was observed on MRI, therefore re-operation was planned. At midnight, she complained of severe headache all around the head, nausea and and vomiting, which were not alleviated in the recumbent position. CT head scan depicted cerebral infarction with accompanying minimal ICH and IVH. In a few hours, she developed altered consciousness, increased vomiting and left hemiparesia despite conservative approach. On repeat head CT scan, increase in edema areas around infraction zones were observed. On MR angiography there was sinus thrombosis. Low molecular weighted heparin anticoagulation was initiated, however heparin related thrombocytopenia occured and heparin was replaced with rivaroxaban substitute. Her status got better day-by-day. After 38 days of second admission, the MRI depicted clot resolution. She had minimal residual neurologic deficits. Six-month of oral rivaroxaban regimen was scheduled.

The second patient, A-58 year-old man, admitted to our clinic due to long lasting low-back and bilateral leg pain. There were hypoesthesia on L5 and S1 dermatomal zones and 1/5 motor weakness of right foot plantar flexion. Lumbar MRI scans revealed lumbar spinal stenosis at L4 and L5 levelswith protruding additional intervertebral disc herniation. He underwent posterior laminectomy for L4, laminotomy for L5, L5-S1 microdiscectomy, and bilateral L3 to L5 transpedicular fixation with arthrodesis. The surgery was uneventful. The patient awoke from surgery neurologically intact. On the post-operative 6th hour, he became lethargic with sudden onset headache and bilateral blurred vision. An emergency head CT displayed an intrasellar lesion with varying densities, extending upward to the optic chiasm. The lesion had mass effect over optic chiasm and cavernous segments of bilateral internal carotid arteries. Corticosteroid replacement therapy was started. Other possible etiologies were searched thoroughly, nothing except the pituitary apoplexy was found. There was no electrolyte imbalance and the hormone profile was normal. A microsurgical decompressive surgery via transphenoidal route was performed on post-operative day 1. The lesion was hemorrhagic and necrotic. Histopathological analysis was compatible with PA within a chromophobe adenoma. Post-operative MRI showed the decompression of the optic chiasm. Post-operative hormone profile and electrolyte levels were in normal range. He was discharged with ongoing steroid replacement regimen. He was in awell condition 4 months after the surgery.

Tablo-1. Data regarding the following variables were looked for: demographics, clinical presentation, index spinal procedure, postoperative complications, presence/absence of drain placement, location of intracranial events, radiologic findings, treatment strategies, and outcome

Remote complication	Age (years)/ Sex	Presentation	Diagnosis	Index Spinal Procedure	Durotomy	Drain	Complaint, Symptom, Onset	Radiologic Findings	Treatment Strategies	Outcome
ICH	67 / F	back pain and severely impaired functional mobility	arachnoid cyst, spinal stenosis.	T9-11 laminectomies, arachnoid cyst fenestration, T8-12 posterior bilateral transpedicular screw fixation	deliberate	+	HA, somnolent, within 24 hours postoperatively	right frontal intracerebral hemorrhage accompanying with subarachnoid hemorrhage and bilateral cerebellar hemorrhages	Conservative	no further neurological deficits, died 3 months later after discharge
	75 / F	back pain and neurogenic claudication	lumbar spinal stenosis	L1-L5 laminectomies, , T10-S1 posterior bilateral transpedicular screw fixation	incidental		HA, somnolent, left hemiparesia, after 4 days postoperatively	right basal ganglia hemorrhage accompanying with subarachnoid hemorrhage/ intraventricular hemorrhage	Ventriculostomy	brain dead on the 20th postop day
	44 / F	back pain and postural instability	Severe Thoraco-lumbar kypho-scoliosis	L1-L4 laminectomies, , T10-L5 posterior bilateral transpedicular screw fixation	incidental		HA, after 3 days postoperatively	right frontal intracerebral hemorrhage	Conservative	Complete recovery
CBH	53 / F	Low- back and bilateral legpain	L4-5 lumbar spinal stenosis + intervertebral disc + grade II spondylolist	L4 laminectomy, L4–L5 microdiscectomy, L3-5 bilateral transpedicular screw fixation + L4-5TLIF	incidental	+	HA, dysarthria, N/V, 2 days postop	small site of hemorrhage in right cerebellar hemisphere	Conservative	Complete recovery
	48 / F	Low- back and bilateral legpain	Lumbar spinal stenosis	L4 laminectomies, L4–L5 microdiscectomy, L4-5 bilateral transpedicular screw fixation+ L4-5TLIF	incidental	+	HA, dysarthria, N/V, progressive ataxia, After 24 hours postoperatively	small sites of hemorrhage in both cerebellar hemispheres	Conservative	Complete recovery

Remote complication	Age (years)/ Sex	Presentation	Diagnosis	Index Spinal Procedure	Durotomy	Drain	Complaint, Symptom, Onset	Radiologic Findings	Treatment Strategies	Outcome
SDH	81 / M	Low-back pain, neurogenic claudication	Lumbar spinal stenosis	L4 laminectomy	incidental	+	HA, severe back / bilateral leg pain, 3 weeks postoperatively	bilateral frontal, temporal, and parietal subacute subdural hematomas	Surgical evacuation and lumbar CSF fistula repair	Complete recovery
		12 / F	Spastic tetra-paraparesia	Dorsal selective rhizotomy	deliberate		HA	Right fronto-temporal subacute subdural hematoma	Conservative	Complete recovery
CVT	51 / F	low-back and bilateral lower extremity pain	spinal stenosis, degenerative scoliosis	L2-5 posterior laminectomies, L5-S1 microdiscectomy+ TLIF cage placement, T10-S1 posterior spinal fusion + instrumentation	incidental	+	HA, N/V 5 days	venous infarction in bilateral thalamus + basal ganglia, hemorrhagia in right caudate nucleus +lateral ventricules, thrombosis in right sigmoid sinus + transverse sinus + sinus rectus	Conservative	Minimal residual deficits
Pit. Apo.	58 / M	low-back and bilateral leg pain	L4-5 lumbar spinal stenosis + intervertebral disc	L4 posterior laminectomy, L5 laminotomy, L5-S1 microdiscectomy, bilateral L3-5 transpedicularscrew fixation + arthrodesis	none	-	HA, lethargy, bilateral temporal hemianopsia 6 hours	intrasellar lesion extending upward to the optic chiasm + cavernous segments of bilateral internal carotid arteries	Decompression via transphenoidal route	No visual field defect
Seizure	66 / F	bilateral lower extremity pain and neurogenic claudication	L4-S1 spinal stenosis, grade II spondylo listhesis	L4-S1 posterior laminectomies, bilateral L4- S1 transpedicular screw fixation	none	-	Anisocoria, right sided hemiparesia, seizure 20 minutes	Intracranial right frontal, contrast-enhanced lesion with peripheral edema and midline shift	Craniotomy and tumor excision	Complete recovery

The patient who had immediate postoperative seizure was a-66-year-old woman. She presented with bilateral lower-extremity pain and neurological claudication at 100 meters. Her neurological examination and the pre-operative laboratory values were normal. Lumbar MRI scans revealed L4 to S1 spinal stenosis and grade II spondylolisthesis. She underwent posterior laminectomies for L4 to S1 with bilateral L4 to S1 transpedicular screw fixation. The procedure ended with no intraoperative complications. However, 20 minutes after turning off the anesthetics, her spontaneous respiration was still irregular, and in neurological examination there was no verbal response, anisocoria and right sided hemiparesia was observed with concomitant seizure. The patient re-intubated and an emergent head CT was performed that revealed a right frontal, intracranial contrast-enhanced lesion with peripheral edema and midline shift. Twenty-four hours after the initial surgery the patient underwent craniotomy and tumor excision, with complete neurological recovery.

One of the three patients who had remote cerebellar hemorrhage was a 48-year-old woman. She presented with pain in her lower back and both legs. The neurologic examination showed bilateral hypoesthesia on L3-L5 dermatomes. MRI scans revealed lumbar spinal stenosis, a herniated disc at L4–L5 level, and grade II spondylolisthesis at L4–L5 level. She underwent posterior laminectomies from L3 to L5, L4–L5 microdiscectomy with L3-5 bilateral transpedicular fixation. A dural tear was identified intraoperatively and primary watertight closure was performed with 4-O silk, however approximately 100 ml of CSF escaped before the closure. When this accident occurred, an immediate drop in blood pressure was observed which was attributed to the CSF leakage. After the routine treatment for hypotension (10 mg intravenous ephedrine sulfate), the blood pressure returned to normal range within approximately 10 min. Prior to closure, a hemovac drain was placed into the epidural space. The patient awoke from surgery neurologically intact. Her blood pressure was normal in the postoperative period. At 12 hours after surgery, the patient complained of severe headache and over the next 12 hours developed some dysarthria, followed by vomitting. Over the course of the next 24 hours the patient was conscious. In the first 24 hours after surgery, 500 ml of fluid were removed via the hemovac drain. Emergent head CT demonstrated small sites of hemorrhage in right cerebellar hemisphere. The results of laboratory investigations, including platelet count and a clotting screen, were all in the normal range. The patient was managed conservatively with anti-edema treatment (4 mg intravenous dexamethasone every 6 h), analgesics, and immobilisation. Her neurological status did not deteriorate any further after the detection of hemorrhage. Control head CT at 48 hours showed no enlargement of the hemorrhage sites and there was no hydrocephalus. Nine days after the operation, the patient was discharged in good condition with no neurologic deficits and she was full mobile.

One of the three patients who had ICH was a-67-year-old woman with a history of diabetes mellitus and Parkinson's disease. She presented with intractable back pain and severely impaired functional mobility. Paraparesia of 3/5 motor weakness and hypoesthesia on bilateral T8 to S1 dermatomes were significant findings. Lumbar MRI scans demonstrated arachnoid cyst and thoracolumbar spinal stenosis at multiple levels with kyphoscoliosis. She underwent posterior laminectomies from T9 to T12 and T8 to T11posterior spinal fusion with bilateral transpedicular instrumentation. Preoperative blood pressure was 110/75 mmHg, after general anesthesia administration blood pressure dropped to 102/74 mmHg and remained within this value throughout the surgery. Total per-operative blood loss was 200 cc. The dura was opened for arachnoid cyst fenestration. Primary watertight closure was performed with 4-O silk, and dura sealant (Adherus®, HyperBranch Medical Technology, Inc, and North Carolina) was used to cover the repair site. A hemovac drain was placed into the epidural space. Post-operative laboratory values were within normal limits. Motor function was similar with preoperative state upon awakening. However, within 24 hours, the patient had severe onset headache, became somnolent and left hemiparesia became more prominent. Hence, an emergent head CT was held and revealed a right frontal intracerebral hemorrhage accompanying with subarachnoid hemorrhage, bilateral symmetrical cerebellar hemorrhages and intraventricular hemorrhage. MR-Venography of the patient was in normal limits, which excluded hemorrhagic venous infarction. In the first 24 hours postoperatively, 600 ml of serosanguineous fluid were removed via the hemovac drain and then it was removed. The patient was admitted to ICU and managed conservatively with both anti-edema treatment (4 mg intravenous dexamethasone every 6 h) and anti-epileptic treatment (500 mg intravenous Levetiracetam every 12 h). Her neurological examination status improved to baseline.

The patient was discharged to a rehabilitation facility 6 weeks after surgery with no further complications or neurological deficits. However, cognitive defects, residual dysphagia and neurogenic bladder was still present. She died 3 months later of aspiration pneumonia.

One of the two patients who had SDH was a-81-year-old man with a history of hypertension, diabetes mellitus. He presented with lower back pain, minimal lumbar scoliosis and severe neurogenic claudication at 10 meters. He was intact on neurological examination. Lumbar MRI scans demonstrated spinal stenosis at multiple levels. All laboratory data including coagulation status were within

normal limits. He underwent posterior laminectomies for L4 to L5 with L4-L5. A dural tear was identified intraoperatively which was closed with a running 4-0 silk suture and was tested under direct visualization with an applied Valsalva breath at 40 mm Hg. No residual leak was observed at that time. A hemovac drain was placed into the epidural space. Post-operative neurological examination and laboratory values were normal. However the patient noticed a slight diffuse headache while standing up and over the next 3 weeks, the headache increased dramatically and analgesics were ineffective. The pain consistently relieved in the recumbent position, therefore he remained confined to bed most of the time. Additional symptoms included severe back and bilateral leg pain. Upon admission to hospital, on physical examination, subcutaneous and paravertebral fluid collection were noted. His Glasgow Coma Scale score was 15/15 with no focal motor deficit. CSF collection at the surgical site was observed on lumbar MRI, therefore re-operation was planned. After MRI scans of the brain, which revealed bilateral subacute subdural hematomas extending along the frontal, temporal, and parietal regions, more prominent on the right side, the surgical plan was changed and it was decided to drain the hematoma from bilateral burr-holes together with repairing the CSF fistula. After draining the hematoma, the lumbar spinal exposure was held. We observed a dural tear, sutured primarily and applied dura sealant (Adherus®, HyperBranch Medical Technology, Inc, North Carolina) over the repaired dural defect. No per-operative complication occurred. The patient's complaints improved dramatically after the surgery. She was discharged from the hospital the third day after the operation with resolution of her symptoms.

DISCUSSION

Despite the large patient series have been reported about lumbar spinal surgery, literature on the prevalence of postoperative remote intracranial complications is sparse. Most of the reported cases are about ICH and CBH with or without IVH/SAH after lumbar spinal surgery [11,18,20,22,32]. SDH (less than 10 cases) [1,7,16,18,28], seizure (5 cases) [6,12], EDH (1 case) [18], pituitary apoplexy (1 case) [23] and CVT (1 case) [24] are other various remote complications.

The highest incidence of remote intracranial complication after lumbar spinal surgery is attributed to postoperative ICH and CBH [4,17]. Although more common etiologies for spontaneous cerebral hemorrhage are hypertension, coagulopathy and anti-coagulant treatment, it is rarely present in previously published reports for remote ICH or CBH after spine surgery. Instead, the most consistent contributing factor is a dural tear [18], one of the most common complications of spine surgery with a prevalence of 1 to 17 % [8,10,15,17,19]. The causes of peroperative dural tear include either eroded or thin dura, dura adhesion, and redundant dura in patients diagnosed with a tight spinal stenosis in primary spine surgery or patients who have epidural fibrosis and scar tissue adherent to the dura during revision of spine surgery [25,34]. The pathophysiological mechanism of ICH/CBH after CSF loss is unclear, yet the evidence suggests that the hemorrhages has been postulated to happen after excessive intra-operative CSF loss and excessive post-operative suction by drains, which results in cerebral dehydration, resulting in stretching and therefore tearing of the cerebral and cerebellar veins [4,17,20,22].

In our clinical practice, presentation of the patients ranges from transient cerebellar findings to large lobar hematoma causing hydrocephalus, which can be accurately diagnosed by computed tomography (CT) and magnetic resonance imaging (MRI). Kaloostian et al reported that 33% of patients with ICH were symptomatic within the first 10 hours postoperatively (17). Similarly, in our series, symptoms of 3 patients with ICH begun after 12 hours. ICHs/CBHs are usually located subcortically [17,21-22]. CBH usually has a streaking pattern of subarachnoid blood along the superior aspect of the cerebellar folia, so-called "zebra sign", which is related to a prior or ongoing loss of CSF [4,13,34].

Treating the complication of ICH varies depending on location and extent of hemorrhage and clinical examination status of the patient. Patients who have a small hemorrhage without significant mass effect and whose neurological status is appropriate may be managed conservatively with immediate removal of subfascial drain and complete bed rest, but in some cases surgical removal of hematoma, re-exploration of the surgical site or external ventricular drainage for hydrocephalus may be needed [5,11,17-18,26-27]. Mortality rates reported for remote ICH and CBH after spinal surgery reveals a mortality rate of 14% and 10 to25%, respectively [21,32]. Kaloostian et al reported that in their series of 8 patients with remote ICH, 2 were declared brain dead during their postoperative hospital stay, and 1 expired of aspiration pneumonia [17]. In our experience, 2 of 3 patients with ICH, died due to complicating systemic problems. However, all of the patients with CBH were managed conservatively, recovered well and discharged neurologically intact.

Up to date, there have been reported only seven cases of intracranial SDH after spinal surgery [1,9,13,18,30]. Similar mechanisms for remote CBH/ICH are thought to be responsible for subdural hematoma formation after CSF leakage with ensuing intracranial hypotension [2,4,17,18,20,22,25]. However, the exact pathophysiological mechanism is not known. In the present study, none of the 2 patients had a history of head injury or received anticoagulants and the postoperative coagulation status

was within normal limits. The patient with SDH after selective dorsal rhizotomy was treated conservatively with bed rest, hydration and analgesic, but the treatment of second case with huge lumbar CSF fistula necessitated bilateral hematoma evacuation with spinal dural defect repair.

Seizure and delayed emergence from anesthesia after spine surgery has been reported [2,6,12,31]. Here, we describe seizure after elective lumbar spine surgery as a first clinical manifestation in a patient with undiagnosed brain tumor. Seizure after accompanied by neurological findings may be the first sign of intracranial tumor. Several factors likely contributed to the exacerbation of mass effect and neurological sequelae after the laminectomy, including prone position, intraoperative fluid administration, surgical stress, and residual anesthesia.

A meticulous review of the literature showed that other sparse remote intracranial complications after lumbar spine surgery have been described [3,6,9,12,23-24]. In our study, we included the patients with cerebral venous thrombosis (CVT), pituitary apoplexy and seizure; respectively (described in case series). CVT is a extremely rare complication after spine surgeries, which may occur in patients with pro-thrombotic states as CSF leakage indirectly contributes to volume loss in the cerebral sinuses [18,24,27]. To the best of our knowledge, this is the second report of intracranial CVT after lumbar spine surgery complicated by dural tearing which has high mortality and morbidity rates if no intervention is done in time. High clinical suspicion in patients with severe headaches worsening in erect positions, focal neurological deficits, seizure and depressed consciousness with necessitate radiological tools and genetic analysis for thrombophilia.

Here, we also describe seizure after elective lumbar spine surgery as a first clinical manifestation in a patient with undiagnosed brain tumor. Seizure accompanied by neurological findings may be the first sign of intracranial tumor. Several factors likely contributed to the exacerbation of mass effect and neurological sequelae after lumbar spine surgery; including prone position, intraoperative fluid administration, surgical stress, and residual anesthesia.

Beside these, Liu et al described pituitary apoplexy in a patient operated for lumbar fusion, with anterior laparoscopic approach [23]. Pituitary apoplexy should be kept in mind for differential diagnosis in cases of headache, nausea, vomiting, ophthalmoplegia, visual loss and electrolyte imbalance occurring after spinal surgeries, which we encountered as the second case in the literature.

Although the presenting symptoms may be mostly nonspecific, remote intracranial event should be suspected in any patient with intractable headache, focal neurological deficits or unexplained deterioration of consciousness following spine surgery; especially complicated by known dural tear and CSF leak. These symptoms will necessitate radiological studies for diagnosis; and appropriate treatment method will be initiated spesific for each patient will save their life with good clinical outcomes.

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