

## Posterior Shoulder Dislocation As a Post-Operative Complication of Carotid Endarterectomy: Case Report and Review of Literature

Idit Melnik, Ehud Atoun, Ronen Debi, Yafim Harach, Ofer Levy, Ornit Cohen, Boris Yoffe

Idit Melnik, Yafim Harach, Boris Yoffe, Department of General and Vascular Surgery, Barzilai Medical Center, Ashkelon, Israel; Affiliated with the Faculty of Health Sciences, Ben-Gurion University of the Negev.

Ehud Atoun, Ronen Debi, Ornit Cohen, Department of Orthopaedics, Barzilai Medical Center, Ashkelon, Israel; Affiliated with the Faculty of Health Sciences, Ben-Gurion University of the Negev.

Ofer Levy, Reading Shoulder Unit, Royal Berkshire Hospital, Reading, the United Kingdom

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**Correspondence to:** Ehud Atoun MD, Department of Orthopaedics, Barzilai Medical Center, Ashkelon, Israel.

Email: [dratoun@gmail.com](mailto:dratoun@gmail.com)

Telephone: +972577678722

Fax: +97246380044

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### ABSTRACT

Changes in blood pressure after carotid endarterectomy (CEA) may lead to the cerebral hyperperfusion syndrome (CHS), which might be accompanied by postoperative seizures. We report a case of a 71-year old man that underwent left CEA and developed a tonic-clonic seizure in the recovery room after hearing a familiar voice. Physical

and radiological imaging revealed a massive rotator cuff tear and locked posterior shoulder dislocation of the right humerus with a large impaction fracture. He was successfully treated with a reverse shoulder arthroplasty and gained good functional results.

**Key words:** Shoulder, Dislocation, Carotid endarterectomy, Seizure

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### INTRODUCTION

Carotid endarterectomy (CEA) is most commonly performed for a high-grade internal carotid artery stenosis. Manipulation of the carotid bulb during carotid endarterectomy may result in hemodynamic instability intraoperatively and in the early postoperative period. Thus, it is standard care for CEA patients to be placed in a monitored setting of blood pressure. Those changes in blood pressure may lead to the cerebral hyperperfusion syndrome (CHS), a rare syndrome which causes most of postoperative intracerebral hemorrhages and seizures in the first two weeks after surgery.

Posterior shoulder dislocation has repeatedly been reported as a complication of convulsive disorders. The dislocation is frequently accompanied by a massive rotator cuff tear and an anterior humeral head impaction fracture (reverse Hill Sachs lesion).

We report a case of a posterior fracture dislocation of the shoulder with a massive rotator cuff tear following CEA. This is a complication that has not been previously reported in the literature.

### CASE REPORT

A 71-year-old man with a history of diabetes mellitus, coronary artery disease, and peripheral arterial disease underwent a left CEA with Dacron patch angioplasty for asymptomatic high-grade stenosis under

general anesthesia in April of 2012 at our institution. The procedure was technically unremarkable. The patient was anticoagulated with intravenous Heparin. A Dacron patch was used for closure of the common and internal carotid arteries. After extubation, during recovery from the anesthesia the patient was hypertensive with blood pressure up to 204/83. He was treated with beta-blockers and intravenous nitroglycerine with partial response. Blood test revealed mild hyponatremia, hypomagnesemia and hypocalcemia. When the patient's wife entered the recovery room, immediately after hearing her voice he developed a tonic-clonic seizure and lost conscious. He was re-intubated and admitted for further treatment in the ICU. Post seizure brain CT failed to demonstrate cerebrovascular attack. He gradually underwent extubation with no recurrence of a hypertensive episode.

While gaining conscious he started to complain of pain in his right shoulder. Physical examination revealed locked internal rotation of the upper limb with a void in the anterior aspect of the shoulder. CT scan of the shoulder revealed a posterior dislocation of the humerus with a large impaction fracture of the humeral head (Figure 1) and severe fatty degeneration of the supraspinatus and infraspinatus. US scan of the shoulder revealed a massive rotator cuff tear. The patient refused to further treatment at this stage, therefore, no attempt for close reduction of the dislocation was performed. In the early post-operative period the patient had marked limitation of range of motion with severe pain therefore he decided to go through with surgery six weeks after the dislocation. Due to the time from injury, size of the bony and tendon injury and his age the patient was a candidate for reverse shoulder arthroplasty that allows shoulder function in the absence of rotator cuff. The surgery underwent without complication (Figure 2) with good functional results at three years follow up.

## DISCUSSION

Hemodynamics alternations after CEA (as the hypertensive crises presented in our case) can lead to a cerebral hyperperfusion syndrome (CHS) or hypertensive encephalopathy (HE).

The term cerebral hyperperfusion syndrome was first reported by Sundt *et al*<sup>[5]</sup> in 1981. He described the triad of ipsilateral migrainous headache, focal neurological deficit and seizure in the absence of cerebral ischemia following CEA. It was suggested that there might be a synergistic link between CHS and HE. It does seem increasingly likely that the two are interlinked and may represent synergistic triggers for a subsequent "common pathway". Considering the two syndromes together might explain the early post CEA seizure as was observed in our case.

The incidence of CHS after CEA is 0-3%, although some report an incidence as high as 18.9%<sup>[1]</sup>. The pathophysiology of CHS is due to impaired baseline cerebral autoregulation mechanisms as arterioles are already maximally dilated to account for the altered cerebral perfusion secondary to carotid stenosis<sup>[2]</sup>. In the presence of an impaired cerebral autoregulation mechanism, changes in systemic arterial pressure have a direct effect on CBF. This concurs with the 'impaired perfusion pressure breakthrough theory', which suggests that the structurally weaker capillaries in chronically ischemic brain are more likely to rupture and bleed due to the distension during reperfusion<sup>[4]</sup>.

As mentioned above, one of the symptoms of CHS and HE is focal motor seizures. Those, Post endarterectomy seizures are a rare complication that raises a major concern. Naylor *et al* reported a series of eight patients who developed post endarterectomy seizures, seven of which developed post-ictal neurological deficit (stroke-5,



Figure 1 Axial CT image of posterior dislocation of humerus.



Figure 2 AP post-operative X-ray of rt. shoulder.

TIA-2), all had had significantly elevated blood pressure at onset of seizures<sup>[1]</sup>.

Our case correlates with those prior reports of an early hypertensive blood pressure after CEA that leads to CHS/HE with a manifestation of a focal motor seizure. However, in this case the sound of a familiar person was the actual trigger for the seizure. This, emphasizes the need for a tranquil environment after the surgery.

Posterior shoulder dislocation is rare, accounting for less than 5% of all shoulder dislocation. They are frequently associated with seizures, trauma or electrocution. The mechanism is an unbalanced muscle contraction, which occurs during the seizure when the internal rotator muscles of the shoulder contracts with greater force than the external rotator causing the humeral head to move superiorly and posteriorly<sup>[3]</sup>. In our case, the dislocation that caused a significant bony injury to the humeral head and a massive rotator cuff tear was successfully treated with reverse shoulder arthroplasty and the patient gained full range of motion after treatment. To our knowledge, this is the first case of a posterior shoulder dislocation as a result of a post endarterectomy seizure.

## CONCLUSION

We report the first case of a posterior shoulder dislocation caused by post endarthrectomy seizure. The patient had CHS and with the trigger of hearing a familiar voice he developed a tonic clonic seizure that resulted in a posterior shoulder dislocation. Physicians treating those patients should be aware of the “post CEA seizure syndrome” and tend to avoid any unnecessary stimulation in the post-operative period and treat post CEA hypertension more aggressively.

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