Management of a patient with Opalski’s syndrome in intensive care unit and mini review of the literature

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Abstract:
Stroke syndromes include a variety of syndromes with often overlapping clinical presentations. When ipsilateral hemiplegia is associated with symptoms of a lateral medullary syndrome, it corresponds to the submedullary syndrome of Opalski. A 72-year-old woman presented with sudden onset of headache, gait disturbance, and recurrent vomiting. Her clinical status gradually deteriorated and she was admitted to the intensive care unit where a variety of problems, related to her diagnosis (Opalski syndrome), were managed. Hippokratia 2012, 16, 4: 373-374

Key words: Opalski syndrome, intensive care unit

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Introduction
Lateral medullary infarction (Wallenberg’s syndrome) is a relatively common vertebrobasilar vascular syndrome. However, ipsilateral hemiparesis as part of lateral medullary infarction is rare, and is known as Opalski’s syndrome. We present the management of a 72 year old female patient with Opalski syndrome in our intensive care unit.

Case report
A 72-year-old female patient (body weight 76 kg, height 160 cm, BMI 30.08 kg/m²) was admitted to our intensive care unit after being hospitalized for 3 days in another general hospital. She presented to the Emergency Department there, with sudden onset of headache, gait disturbance, vertigo, and recurrent vomiting. Her past medical history included diabetes mellitus under insulin therapy with Lantus® 30 i.u. q.d., Novorapid® 8-12-8 i.u q.d., arterial hypertension under multi-antihypertensive regimen with perindopril 2.5 mg q.d., amlodipine 5 mg q.d., carvedilol 6.25 mg b.i.d and furosemide 20 mg q.d. Surgical history was unremarkable. Family history was non-contributory. She had no known drug allergies.

The next day her clinical status aggravated with tachycardia and an episode of atrial fibrillation, dysarthria and dysphagia, drooping of the right corner of mouth, nystagmus and reduced corneal reflex of the right eye. Computed Tomography (CT) with intravenous contrast media, revealed an isodense area without clear limits in the right cerebellopontine angle with nonhomogeneous perfusion, regional oedema and displacement of the 4th ventricle and brainstem to the left. A few hours later, the patient’s neurological status suddenly deteriorated (Glasgow Coma Scale –E2/V1/M6); she was intubated and transported to our hospital where a second CT was performed. The latter revealed a tension hydrocephalus and an infarction lesion in the right cerebellopontine angle. The 4th ventricle was not visualized. The patient was transported to the operating room where a valveless drain tube was placed in the anterior horn of the right lateral ventricle under general anesthesia. Following surgery, she was admitted to intensive care unit (ICU).

In the ICU, the patient was placed under sedation with continuous intravenous infusion (c.i.v.) of propofol 25-35μg/kg/min and mechanical ventilation (FiO₂ 40%, PEEP 6 cmH₂O, 10 breaths/min with a tidal volume of 6 ml/kg). Antiedemic regimen (dexamethasone 16 mg i.v. q.d., mannitol 20% 100ml i.v. q.i.d.), antimicrobial and antiulcer prophylaxis were administered; blood glucose was controlled via insulin protocol with bolus administrations. Hemodynamically, the patient was stable with atrial fibrillation and normal ventricular rate. Upon admission, a transcranial Doppler study and a transorbital optical nerve sheath ultrasonography were performed with normal findings.

On day 3, enteral nutrition was initiated and, in order to meet the increasing daily needs of insulin to maintain blood glucose between 100-150 mg/dl, insulin c.i.v. was started according to a sliding scale. Atrial fibrillation was managed with propranolol 40 mg p.o. t.i.d.. Mechanical ventilation parameters were also modified (PEEP 8 cm H₂O) due to atelectasis of the left lower pulmonary lobe. On day 5, a gradual dose reduction of antiedemic regimen was initiated. On day 7 transdermal tracheostomy was conducted and the next day a second computed tomography scan was performed revealing a significant reduction of hydrocephalus.

Based on the new radiological findings, sedation was...
stopped; an early neurologic evaluation was made (GCS E3/V1/M1 with normal pupils’ reaction to light). The following days de-escalation of mechanical support was achieved (change of mode to ASV followed by PSV), insulin administration was changed from c.i.v. to bolus i.v. as blood glucose control became more effective and nifedipine 30mg p.o.s (Levin) t.i.d was added as antihypertensive therapy. On day 20, the patient became septic with fever (38.8°C), aggravating respiratory parameters, and hypotensive with sudden onset of paroxysmal atrial fibrillation (ventricular rate of 145-160 beats per min).

An amiodarone infusion was started, blood cultures were performed and antibiotic regimen was changed. Progressively, the neurological state of the patient ameliorated and the hydrocephalus drain tube was removed. Laboratory cardiac indices of ischaemia and transthoracic echocardiography revealed no aberrations; yet, the patient remained dependent to amiodarone infusion. A cardiologist consultation was made and finally the rhythm was controlled with amiodarone 200mg p.o.s. b.i.d and atenolol 20 mg p.o.s. b.i.d. The patient was freed from mechanical ventilation (breathing through tracheostomy) and a full neurological evaluation was performed. The latter revealed GCS of E4/V1/M6, with ipsilateral to the lesion hemiparesis, dysphagia, drooping of the right corner of mouth and reduced corneal reflex of the right eye. On day 38, the patient was transferred to the ward with the diagnosis of Opalski’s syndrome. The patient was stable during her stay in the ward and a transport to rehabilitation clinic was planned.

Discussion

Ipsilateral hemiparesis with symptoms and signs of lateral medullary infarction were first described by Opalski in 1946. Some pathologic and neuroradiologic reports have shown that the lesion is located lower than in Wallenberg syndrome caused by occlusion of the posterior inferior cerebellar artery. The ipsilateral hemiparesis seen in this syndrome is attributed to the involvement of corticospinal fibers caudal to the pyramidal decussation. Others have explained these combined findings as a result of vertebral artery (VA) occlusion/stenosis or VAD, compromising the medullary penetrating arteries that arise from distal VAs or anterior spinal arteries. Our patient had almost all neurological findings of the syndrome. The latter include -apart the ipsilateral hemiparesis, the Dejerine syndrome, where the lesion is in midoblongata, the Ménière disease, the Marie-Foix syndrome, the Opalski syndrome and others.

Considering the possible involvement of multiple nerve structures, management of patients affected by the syndrome poses a considerable challenge. This case has given us an insight of this rare condition.

References