CLINICAL INFORMATION

Progressive hematoma in anterior neck after endovascular treatment of middle cerebral artery aneurysm

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KEYWORDS
Superior thyroid artery; Cervical hemorrhage; Airway obstruction; Aneurysm endovascular treatment

Abstract
Background: Cervical hematomas can lead to airway compromise, a life threatening condition, regardless of the cause. The following case is the first presentation of cervical hematoma as a complication of endovascular treatment of middle cerebral artery aneurysm.
Case report: A 49 year-old woman was scheduled for stent placement under general anesthesia for middle cerebral artery aneurysm. Few days before intervention, acetyl salicylic acid and clopidogrel treatment was started. Following standard monitoring and anesthesia induction, the patient’s trachea was intubated with a 7.5 mm endotracheal tube and the procedure was completed without any complications. Three hours later, dyspnea developed and physical examination revealed progressive swelling and stiffness in the neck. Endotracheal intubation was performed with a 6 mm diameter unuffed tube with the aid of sedation. The vocal cords were completely closed due to compression. There was no leak around the endotracheal tube. The rapidly performed computerized tomography scans showed an enormous hematoma around the neck and extravasation of contrast medium through superior thyroid artery. After coil embolization of superior thyroid artery, she was taken to the intensive care unit as intubated and sedated. Surgical exploration of the hematoma was not recommended by the surgeons, because she was on clopidogrel. After two days, the patient’s trachea was extubated safely ensuring that the swelling was sufficiently ceased and leak detected around the endotracheal tube.

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Conclusions: Securing the airway rapidly by endotracheal intubation is the most crucial point in the management of cervical hematomas. Diagnostic and therapeutic procedures should be performed only afterwards.

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Background and objectives

Cervical hematoma due to superior thyroid artery (STA) dissection can be a life-threatening condition because of airway compromise. It is encountered mostly after thyroid surgeries, traumas and tumors and rarely spontaneously.1 Main symptoms are dysphonia, dysphagia, neck swelling, hoarseness and eventually respiratory distress and suffocation.1,2

To the best of our knowledge, the following case is the first presentation of cervical hematoma as a complication of endovascular treatment of middle cerebral artery aneurysm.

Case report

A 49 year-old woman was scheduled for stent placement under general anesthesia for middle cerebral artery aneurysm. She had hypertension and a history of stroke. In addition, balloon angioplasty was performed for treatment of stenotic right internal carotid artery. Few days before intervention, acetylsalicylic acid and 300 mg clopidogrel treatment was started. On arrival to the operating room; electrocardiogram, non-invasive blood pressure and peripheral oxygen saturation was monitored. Propofol 2 mg.kg⁻¹, fentanyl 1 mcg.kg⁻¹ and rocuronium 0.5 mg.kg⁻¹ were administered for induction of general anesthesia and maintenance was achieved with 2% sevoflurane and remifentanil infusion. The patient’s trachea was intubated with 7.5 mm endotracheal tube without any difficulty. The procedure was completed without any complications. Three hours later, in the postanesthesia care unit, dyspnea developed and physical examination revealed progressive swelling and stiffness in the neck. Endotracheal intubation was performed with a 6 mm diameter uncuffed tube with the aid of sedation with 100 mg propofol and 3 mg midazolam. The vocal cords were completely closed due to compression. Cormack-Lehane laryngoscopic view was Grade II. There was no leak around the endotracheal tube. The rapidly performed computerized tomography scans showed an enormous hematoma around
the neck and extravasation of contrast medium through STA (Fig. 1). After coil embolization of STA, she was taken to the intensive care unit as intubated and sedated. Surgical exploration of the hematoma was not recommended by the surgeons, because she was on clopidogrel. After two days, the patient’s trachea was extubated safely ensuring that the swelling was sufficiently ceased and leak detected around the endotracheal tube.

Discussion and conclusions

We reported a case with airway compromise due to cervical hematoma as a complication of endovascular treatment of middle cerebral artery aneurysm. The rate of morbidity and mortality during endovascular treatment of intracranial aneurysms range from 1.06% to 15.7%. Rupture, thromboembolism, neurological complications like disability and impaired cognitive status, post-procedure rebleeding and death are among the well-known complications. But we could not find any reports mentioning a cervical hematoma as a complication of endovascular treatment of an intracranial aneurysm. The procedure had been completed without any difficulty or complications so we do not suppose any wire rupture to cause STA hemorrhage during the exchange procedure. The tests before the procedure showed that the patient was not hyperresponsive to clopidogrel. We suggest that the bleeding in our patient might be due to a spontaneous STA rupture under dual antiagregant treatment.

Cervical hematomas can lead to upper airway obstruction and death regardless of the cause. Tew et al. reported a patient who developed respiratory arrest due to an unrecognized cervical hematoma. Even in the absence of respiratory distress, elective tracheal intubation should be considered in an enlarging cervical hematoma. Otherwise, airway protection may be difficult requiring tracheotomy or even impossible. We also performed an early intubation with the aid of sedation in order to secure the airway promptly. Even so, her vocal cords were already completely closed due to the compression and a 6 mm diameter uncuffed tube fit into the trachea with difficulty and provided ventilation without any leak. It may be better to keep in mind to use an armed endotracheal tube to ensure the patency of the airway because conventional endotracheal tubes can be compressed by an enlarging hematoma. We did not have an armed tube in reach so we preferred not to wait for one and intubated the patient’s trachea as soon as possible.

Although airway control is the mainstay of the management of cervical hematomas, the risk of hemodynamic deterioration due to a possible baroreflex mediated mechanism should not be forgotten. Sethi et al. reported a case of cervical hematoma following anterior cervical spine surgery who experienced severe hypotension and bradycardia after intubation and adequate ventilation. The authors suggested that this situation was based on the mass effect of the hematoma on the carotid sinus resulting in baroreceptor reflex because the hemodynamic instability had reverted by evacuation of the hematoma. Our patient was hemodynamically stable during all the follow-up.

Surgical exploration and evacuation of the hematoma is usually suggested to facilitate the extubation and shorten the duration of hospital stay. Also, Yu et al. used partial hematoma decompression local anesthesia to facilitate intubation in their case with delayed bleeding due to STA dissection 16 days after anterior cervical discectomy. After decompression they intubated the patient easily and safely. Since our patient was hemodynamically stable and the surgery was thought unsafe, we decided to wait for spontaneous cessation of the hematoma unlike the other cases in the literature.

In conclusion; securing the airway rapidly by endotracheal intubation is the most crucial point in the management of cervical hematomas. Diagnostic and therapeutic procedures should be performed only afterwards.

Conflicts of interest

The authors declare no conflicts of interest.

References