CASE REPORTS

Airway problems after carotid endarterectomy

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Summary

Respiratory obstruction has been reported as a complication of carotid endarterectomy; the causes include traumatic mucosal oedema, direct tracheal compression by haematoma and oedema secondary to lymphatic and venous congestion. We report four cases of acute respiratory obstruction complicating carotid endarterectomy. Two of these cases suffered respiratory arrest in the postoperative ward and required emergency tracheal intubation in difficult circumstances. All of these patients had developed wound haematomas and all required surgical intervention. (*Br. J. Anaesth.* 1996; **76**: 156–159)

Key words

Surgery, endarterectomy. Airway, obstruction. Complications, airway obstruction.

Carotid endarterectomy is of proven efficacy in moderate to severe carotid artery stenosis [1]. Surgery involves a vascular graft and manipulation of the carotid body. Recognized complications include postoperative hyper- or hypotension, wound haemorrhage, local oedema and neurological deficits [2]. Airway obstruction is well recognized; this may be caused by haematoma formation, oedema or a combination of both [3]. During a 1-yr period in our unit when 100 procedures were performed, there were four cases of haematoma leading to a compromised airway which required acute airway management and surgical intervention.

Case reports

patient no. 1

A 71-yr-old male was admitted for left carotid endarterectomy having suffered a cerebrovascular accident 10 months previously. His past medical history included hypertension and ischaemic heart disease treated with aspirin, nifedipine and bendrofluazide. His preoperative arterial pressure was 180/90 mm Hg. The operation was uneventful. Throughout the procedure the patient had a stable cardiovascular system and an arterial cannula was used to monitor arterial pressure. According to our standard routine management, heparin 3000 u. was administered i.v. and reversed later with protamine 30 mg i.v. After operation, in the recovery unit, invasive arterial pressure monitoring was continued. Recovery was complicated by systolic hypertension which was treated with increments of hydralazine i.v., however, a systolic arterial pressure of 260 mm Hg was reached before control was achieved.

A neck haematoma was first noticed 1 h from completion of the procedure. At this time the patient's coagulation profile was normal. The haematoma continued to expand and 5 h after operation the patient was noted to be hoarse. On examination there was no respiratory distress and the trachea was central. The patient was reviewed by the vascular surgeon and the area of haematoma was marked. One hour later, with no further evidence of enlargement of the neck swelling, the patient was transferred to the postoperative surgical ward where invasive arterial pressure monitoring was discontinued. Nine hours after operation the patient became acutely dyspnoeic and developed stridor. This progressed rapidly to complete airway obstruction and respiratory arrest. At laryngoscopy no normal structures were seen, the soft tissues were grossly oedematous and friable, with fresh bleeding on pharyngeal suction. Intubation was achieved blindly with a 6.0-mm tracheal tube via the right side of the pharynx. The patient was sedated, paralysed and transferred to the operating theatre where a large haematoma was evacuated. The left facial vein was identified as the source of bleeding and was ligated. Small bleeding points were also sutured on the arterial vein patch. Before tracheal extubation was attempted a gum elastic bougie was inserted. The tracheal tube was then removed and laryngoscopy showed the larvnx to be central with both vocal cords moving normally. After removal of the bougie there was no stridor.

Haemoglobin concentration decreased from 13.3 g dl^{-1} before operation to 9.9 g dl^{-1} . The patient made a full recovery and was discharged on day 5 after operation with no neurological deficit.

PATIENT NO. 2

A 72-year-old male was admitted for left carotid endarterectomy having suffered multiple transient ischaemic attacks in the preceding months. He had a

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history of hypertension. His treatment consisted of aspirin, bendrofluazide, atenolol and diltiazem. Preoperative arterial pressure was 170/68 mm Hg. The operation was uneventful. Intraoperatively heparin 3000 u. i.v. was administered and reversed later with protamine 30 mg, i.v.

After operation, in the recovery unit, he developed hypertension, peaking at 220/130 mm Hg, despite early treatment with bolus doses of i.v. hydralazine. Neck swelling was first noted 30 min after operation; however, after regular surgical review in the recovery unit it was decided to pursue conservative management. With arterial pressure stable and no apparent further increase in haematoma size, the patient was transferred to the surgical ward. Here, after 8 h, the patient developed acute respiratory embarrassment leading to respiratory arrest. At laryngoscopy no anatomical structures could be identified and the trachea was intubated blind with a 7.5-mm tracheal tube through swollen mucosal folds. The patient was then transferred to the intensive care unit. On return to theatre the following morning a large haematoma was evacuated and a bleeding point on a branch of the internal jugular vein ligated. Tracheal extubation 4 h later was uneventful. His haemoglobin concentration decreased from 13 to 9.6 g dl⁻¹ and required transfusion of 2 u. of packed red cells. Postoperative recovery was complicated by transient right-sided weakness and dysphasia which resolved before discharge on day 7 after operation.

patient no. 3

A 79-yr-old male was admitted for right carotid endarterectomy. He had suffered several transient ischaemic attacks in the preceding months and had a history of treated hypothyroidism. His regular medication consisted of aspirin, propranolol and thyroxine. Preoperative arterial pressure was 160/70 mm Hg. The operation was complicated by low stump occlusion pressure, difficulty in inserting the Javed shunt and oozing from the vein graft, requiring extra sutures. Heparin 3000 u. i.v. was administered and reversed later with protamine 30 mg i.v. In the recovery unit a systolic pressure of 220 mm Hg was recorded 6 h after operation which was treated promptly with i.v. hydralazine. At this time the patient was agitated, hoarse and had developed a wound haematoma. He was reviewed by the surgeon at this time and it was decided to manage the patient conservatively on the postoperative surgical ward. At 10 h after operation he had developed stridor and was therefore returned urgently to theatre.

A rapid sequence induction was performed with etomidate 8 mg, midazolam 2.5 mg and suxamethonium 100 mg. Initially at laryngoscopy no normal structures could be identified because of the grossly distended pharyngeal veins and oedematous soft tissues. On further manipulation, the larynx was seen deviated to the far left of the pharynx. Intubation was then accomplished with an 8.5-mm tracheal tube. At operation a large haematoma was evacuated and haemostasis was produced with sutures to the vein graft. After operation he was transferred to ITU and the trachea extubated uneventfully within a few hours. His preoperative haemoglobin concentration of 11.2 g dl^{-1} decreased to 7.6 g dl⁻¹ after operation. His recovery was complicated by a transient hemiparesis and cerebellar signs which resolved before discharge.

PATIENT NO. 4

A 56-yr-old male was admitted for left carotid endarterectomy. He had suffered several transient ischaemic attacks in the previous 6 months and was receiving regular aspirin treatment. He had no other significant medical history. Preoperative arterial pressure was 130/80 mm Hg. At operation a Javed shunt was inserted and standard anticoagulation and reversal administered. In the recovery unit he developed systolic hypertension of 210 mm Hg which was treated successfully with sublingual nifedipine. At this time he was noted to be hoarse and had developed a neck haematoma. He remained in the recovery unit and was reviewed by the surgeon who considered surgical intervention unnecessary. Before transfer to the surgical ward, the haematoma was unchanged in appearance for longer than 1 h, with minimal drainage.

At 6 h after operation in the surgical ward, the neck swelling had progressed and it was decided to explore the wound. On assessment of the airway there was limited mouth opening as a result of gross neck swelling. An inhalation induction was performed with oxygen, nitrous oxide and enflurane. As the airway was easy to maintain, etomidate 12 mg and suxamethonium 100 mg were administered. Intubation was straightforward with a 9.0-mm tracheal tube. At operation a large haematoma was removed, a small artery was cauterized and a defect in the left facial vein sutured. Tracheal extubation immediately after operation was uneventful. His preoperative haemoglobin concentration decreased from 13.9 to 12 g dl⁻¹. He was discharged home on day 4 after operation with no neurological deficit.

Discussion

Previous reports have postulated that the aetiology of airway obstruction after carotid endarterectomy surgery may be direct tracheal compression by haematoma, oedema secondary to either direct mucosal trauma or lymphatic and venous congestion resulting from haematoma formation [4, 5]. Reports suggesting oedema as the primary causal factor have described evacuation of haematoma which did not immediately relieve the airway obstruction; indeed 10 h of elective ventilation after operation has been recommended [6]. Dexamethasone has also been recommended prophylactically after prolonged neck surgery for this reason [7]. However, evacuation of haematoma has also been reported to relieve airway obstruction immediately [8].

Among the four cases described here, there were striking similarities in the perioperative events, which may have contributed to the development of similar postoperative complications. All patients were receiveing aspirin as antiplatelet therapy which theoretically may have resulted in impaired clot formation, aggravating any bleeding. Also, they all received i.v. anticoagulation during operation, although this was reversed adequately with i.v. protamine.

Despite continuous arterial pressure monitoring and the presence of a recovery anaesthetist, postoperative hypertension was common to all cases in the first 6 h. This was controlled successfully with either hydralazine or nifedipine and may have contributed to arterial bleeding but would not account for haematoma formation caused by venous bleeding. Hypertension is well recognized after carotid endarterectomy; it may be caused by carotid body stimulation, pain or neurological insult. Urgent treatment of such severe hypertension is imperative in order to avoid cerebral haemorrhage. When using i.v. vasodilator therapy the dose must be titrated to effect in order to avoid myocardial or neurological damage caused by inadvertent hypotension.

In all cases, the neck haematomas were large and associated with a decrease in haemoglobin $(1.9-3.6 \text{ g dl}^{-1})$. All patients were reviewed by vascular surgeons who, in the absence of airway difficulties and no further neck swelling, considered surgical intervention inappropriate.

Of these patients, three subsequently developed hoarseness but only in one case was evacuation of the haematoma carried out when minimal airway distress was present. In the other two cases conservative management continued until surgery was prompted by respiratory arrest when gross anatomical distortion was found at laryngoscopy. At exploration definite venous bleeding points were identified in three cases; two of these were also bleeding from the arterial vein patch.

It is of interest that in the four cases presented, in two the trachea was extubated at the end of the procedure and in one at 4 h after operation without complications. The fourth was transferred to the regional intensive care unit where evacuation of haematoma was scheduled for the next day and the trachea was extubated 4 h after operation.

Of the three mechanisms of airway obstruction described above, we suggest that haematoma causing lymphatic and venous congestion leading finally to oedema was the most likely sequence of events. This is supported by the delayed onset of airway obstruction and the rapid resolution of oedema after evacuation of the haematoma facilitating early tracheal extubation after operation.

In the emergency situation the airway may be difficult to assess the degree of expected difficulty is often not fully appreciated. The final lifethreatening deterioration is frequently rapid with little warning. Stridor appears to present late as a clinical sign. However, subtle changes in voice quality progressing to hoarseness in the presence of such acute neck swelling should be treated as an indication of impending airway obstruction. In the above cases developing haematoma was initially managed conservatively (6-10 h) before intervention. It is of concern that two of these patients suffered respiratory arrest and in both the trachea was intubated blindly on the ward under difficult conditions.

In order to avoid the catastrophic consequences of respiratory arrest from a completely obstructed airway we suggest that progressive neck swelling should be managed aggressively with early surgical intervention. Successful evacuation of such haematomas under local anaesthesia has been reported. Also, after extensive neck surgery patients should be managed in a high dependency unit with appropriate monitoring, levels of nursing staff and the immediate availability of anaesthetic expertise.

In the event of airway obstruction, management strategies that have been documented include inhalation induction, with or without helium, to decrease turbulent airflow. Fibreoptic intubation has also been advocated, although this technique required a skilled operator and vision may be impaired by anatomical distortion, venous congestion and secretions. Also, the smallest tracheal tube accommodated by the standard intubating bronchoscope is 6.5 mm which may be difficult to pass. Both the retrograde intubation technique and the creation of a surgical airway may be difficult and extremely dangerous in the swollen neck.

It is of concern that anaesthesia was induced in one patient with a rapid sequence induction resulting in unexpected difficulty in seeing the larynx. Fortunately, there were no long-term sequelae as a result of this delay in securing the airway. However, in a previous report of six similar cases, four of which were initially paralysed resulting in inability to ventilate the lungs, all suffered severe hypoxia and cardiac arrhythmias [4]. In three of these the trachea was intubated with difficulty using small tracheal tubes and one underwent emergency tracheostomy while asystolic.

The appropriate moment for extubation may be difficult to assess. From our experience it would seem that elective postoperative ventilation is not always necessary. Various methods may assist in assessing the ability to extubate the trachea successfully at the end of the procedure, namely an audible leak around the deflated cuff at 15 cm H_2O of airway pressure or leaving a gum elastic bougie or a Cooke airway exchanger *in situ* after removal of the tracheal tube.

All patients recovering from carotid endarterectomy require high dependency care to ensure continuous assessment of respiratory and cardiovascular systems. In our unit there is a planned upgrading of the postoperative surgical ward to high dependency status.

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