CASE REPORT

Acute facial oedema post live-donor renal transplantation

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SUMMARY

Facial oedema leading to airway compromise immediately after surgery is a rare event. We report a case of acute facial swelling sufficient to cause a significant obstruction of the patient's airway in recovery. We believe it was caused by partial obstruction of the venous drainage from the head associated with a sudden and large fluid load. A 35-year-old man underwent a live ABO-incompatible renal transplantation during which a central line was inserted into the right subclavian vein and a large volume of fluid was given intraoperatively. He also had a longstanding permacatheter on the left side used for haemodialysis. In the recovery room he developed acute facial swelling which did not resolve with steroids or antihistamines. He was managed by intubation of his airway and ventilation in the intensive care unit overnight before he made a complete recovery with no further intervention. We hypothesise that this event was related to an impaired venous return from his head secondary to the central venous line and the permacatheter partially obstructing the venous drainage from his head and neck combined with an acute large venous fluid load.

BACKGROUND

- To understand the basic physiology related to startling forces
- Rare but important cause for acute facial oedema in a perioperative patient
- Applied physiology and anatomy play a major role in perioperative care in patients with renal failure

CASE PRESENTATION

A 35-year-old Caucasian man underwent a live ABO-incompatible renal transplantation (A donor to O recipient). He had an 18-year history of haematuria secondary to C3GN mesangiocapillary glomerulonephritis associated with CFHR5 mutation. This progressed into chronic renal disease which was managed by haemodialysis through a permacatheter in the left subclavian vein. His wife volunteered to donate a kidney, providing him with an ABO incompatible, human leucocyte antigen mismatch of 2–2–1 kidney.

He followed the local ABO-incompatible kidney transplant protocol, which included preconditioning with rituximab 375 mg/m² daily from 30 days preoperatively, tacrolimus 0.075 mg/kg twice daily and mycophenolate 250 mg twice daily from 14 days before surgery. From 9 days before surgery and the day before surgery he underwent six plasma exchange transfusions (1 plasma volume

replaced with Hartmann's solution and either albumin 4.5% or fresh frozen plasma on each occasion).

On the day of the operation, he was given basiliximab 20 mg and methylprednisolone 500 mg at induction of anaesthesia. A general anaesthetic technique was used with thiopentone 300 mg, rocuronium 70 mg, alfentanil 1 mg and midazolam 3 mg, his trachea was intubated and was ventilated. A capnograph (GE Healthcare type E-CAiOV) was used to maintain the normocapnia at 4.5–5 kPa and anaesthesia was maintained with 1.2 minimum alveolar concentration of desflurane. A triple lumen (1×14G and 2×16G) central line was inserted into his right internal jugular vein immediately preoperatively under ultrasound guidance and no abnormal appearance of the vein was noted. He was started on monosol 900 mL/h at the start of the operation to maintain renal perfusion according to the local policy for renal transplantation. Monosol K with 4 mEq potassium/L is a solution for haemodialysis, haemodiafiltration and haemofiltration and contains sodium 140 mmol/L, potassium 4 mmol/L, magnesium 0.75 mmol/L, calcium 1.75 mmol/L, chloride 119 mmol/L, lactate 30 mmol/L, glucose 5.55 mmol/L and osmolarity 301 mOsm/L.

He remained anuric during the operation. Following an uneventful procedure with minimal blood loss lasting four and three quarter hours the anaesthesia was allowed to wear off, the muscle relaxant reversed with glycopyrrolate 0.5 mg and neostigmine 2.5 mg. The patient was extubated when breathing and then transferred to the recovery area.

In accordance with hospital deep venous thrombosis prophylaxis protocol the patient had thromboembolic stockings with flowtrons throughout the procedure and he was given 5000 IU of low-molecular weight heparin (Fragmin) subcutaneously. Patients undergoing routine transplant are not put on any other anticoagulation unless indicated. In the recovery area his blood pressure (180/100 mm Hg), heart rate (70/min) and oxygen saturation (97%) remained stable and he was alert, but occasionally drowsy. The monosol was continued in recovery at 900 mL/h according to the local renal transplant protocol. Within 20 min he started reporting pain over the operation site. About 10 min later he started to develop swelling of the face and neck. At this time, he was able to protrude his tongue and was talking full sentences and he had an oxygen saturation of 96% on 4 L/min of oxygen delivered via a Hudson mask.

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A chest X-ray performed at this time demonstrated correctly positioned central venous lines, no pneumothorax, no surgical emphysema with a normal mediastinum and heart. Immediately, securing the airway was considered to be the most important issue at this time and took priority over further investigations. As there was no suggestion of cardiac pathology, an echocardiogram was considered not to be indicated.

INVESTIGATIONS

- ▶ Chest X-ray correctly positioned central venous line. No pneumothorax or surgical emphysema and *normal heart*.
- ▶ At 6 h C1 esterase levels (1) were C3 47 mg/dL (normal range 70–165 mg/dL); C4 6 mg/dL (16–54 mg/dL); C1 esterase 18 mg/dL (15–35 mg/dL) and 17 h postevent blood levels for trypsin were 6.5 mcg/L (normal range 2–14 mcg/L).

DIFFERENTIAL DIAGNOSIS

- ▶ Anaphylactoid reaction
- ▶ Partial venous obstruction from the head and neck

TREATMENT

A consultant anaesthetist not involved with the transplant reviewed the patient and an initial working diagnosis of anaphylactoid reaction was made. Hydrocortisone 200 mg and chlorpheniramine 10 mg were administered intravenously. However, over the next 5 min facial swelling continued to increase and the patient was able to speak only in short sentences.

The initial diagnosis of anaphylactoid reaction was then considered less likely because of the immunosuppressants given prior to surgery. Despite this, his blood gas saturation remained at 96% on 4 L/min oxygen. Over the next 5 min the patient deteriorated further and a decision to secure the patient's airway by reintubating his trachea was made. He was transferred to an anaesthetic room and standard monitoring attached.

Following a gaseous induction using 8% sevoflurane in oxygen the patient's lungs were ventilated by hand with ease and paralysis was achieved with rocuronium. Nasal fibre optic intubation was attempted, but the vocal cords were not easily visualised, so oral laryngoscopy using a video laryngoscope (Stortz) was performed. The larynx was oedematous and the vocal cords were difficult to visualise, but a size 6.0 cuffed oral endotracheal tube was inserted into the trachea over a gum elastic bougie. The patient was then transferred to the intensive care unit where he was sedated and ventilated overnight.

OUTCOME AND FOLLOW-UP

The facial swelling settled spontaneously and he was extubated the following day. He was neither haemodialysed nor filtered between the second intubation and second extubation.

The patient developed delayed graft function (DGF) for 5 days post-transplantation hence needed two sessions of haemodialysis.

As his symptoms had settled no further investigations were considered appropriate at this time. We then found that MR venograms performed 1 and 2 years earlier showed high-grade stenosis of right brachiocephalic vein and focal narrowing seen at the junction of the brachiocephalic vein and superior vena cava (SVC). Subsequently, another venogram performed 6 months after the transplant showed the right brachiocephalic vein and the SVC junction was occluded.

The venous stenosis was treated with Atlas 8 mm angioplasty. As we now had a working diagnosis and we felt SVC thrombus was not the cause. The underlying cause which gave rise to the clinical picture was aggravated by the pre-existing venous

stenosis and the use of bilateral neck lines—permacath on the left and central line on the right side.

DISCUSSION

Causes of facial swelling include a reaction to a specific antigen, which may be environmental (eg, pollen or insect bites) or drugs; infections (sinusitis, blepharitis and dental abscess); trauma (eg, sunburn or fractured facial bones) or surgical emphysema.

In 1981, Conacher reported a case of acute onset facial oedema at extubation in a woman with chronic liver disease and who had undergone injection of oesophageal varices. She was treated conservatively and not reintubated. The cause was believed to be the changes in the capillary hydrostatic pressure associated with a Valsalva manoeuvre.¹ Di Cocco *et al* have reported a case of sudden onset of face, arm and upper body swelling associated with thrombosis of the right internal jugular vein with extension into the brachiocephalic vein and partial obstruction of the SVC. This has occurred in a woman with a complex thrombotic condition 7 days following renal transplantation.² She was unsuccessfully treated with anticoagulation and so underwent open reconstruction of the SVC using a superficial vein bypass graft.

Griffiths and O'Sullivan postulated that intubation may be associated with life-threatening airway oedema in a patient with C1-esterase inhibitor deficiency. They described managing a patient for caesarean section with a regional anaesthetic technique, avoiding any airway intubation.³ Munshi *et al*⁴ described postoperative unilateral facial oedema which, they suggested, was due to acute flexion of the neck.

We think an anaphylactoid reaction is unlikely as an explanation of the problems experienced by our patient because he was immunosuppressed for the surgery; there were no changes in biochemical markers for anaphylaxis (although this may have been affected by the immunosuppressant drugs); and we could not

Learning points

- ▶ If rapid infusion of a large volume of fluid is required, such as during and after renal transplantation, it should be carried out with caution as the patient is likely to have difficulties excreting a large intravenous fluid load.
- ▶ Patients with restricted venous drainage from the head are at risk of oedema of the head if we use jugular veins for additional central access during renal transplantation. This may result in airway compromise requiring urgent intervention.
- ▶ In the acute situation a measurement of central venous pressure from the tip of the line in the superior vena cava could have been useful, but the result may have been misleading if it measured the pressure on the cardiac side of an obstruction. If so the result would not reflect the pressure in the upstream jugular venous system.
- ▶ Other sites can be considered for central venous access in patients with restricted venous drainage from the head.
- ▶ Preoperative venous mapping could be considered for patients with renal failure who need central venous monitoring intraoperatively if they have received repeated central venous access shortly before surgery, for example if they received multiple permacath insertions over a short period of time.

identify any exposure to new allergens or likely drug triggers associated with the event. We do not think that the patient had C1-esterase inhibitor deficiency as his levels were not low in the postoperative period. We found no evidence of trauma or surgical emphysema and there was no history of thrombophilia.

We hypothesised that the cause of his facial swelling was an acute response to the large volume of monosol infused in theatres and recovery in a patient with restricted but not critical venous drainage from the head. The mechanism could be an acute obstruction of the venous blood flow from the head related to his permacath with a thrombus on the left side and the new central venous line on the right side, resulting in sudden uncompensated increase in local venous pressure. This in turn could lead to an increased hydrostatic pressure at the venous end of the capillaries, which would have disturbed the balance of Starling forces in his head, and resulted in less fluid resorption from the interstitial fluid compartment. A positive feedback of more swelling, causing more venous obstruction could explain the progressive nature of the swelling, which was further enhanced by the combination of fluid overload plus the DGF.

The swelling reduced within a few hours with conservative measures, which could be consistent with the venous pressure

falling and the restoration of normal capillary hydrostatic pressure. This could be related to a lower intravenous fluid input along with a change in position of the head, resulting in better venous drainage over the central line.

Contributors KSB and YC were involved with the conception, design and drafting of the manuscript. BL contributed with revising it critically for important intellectual content. AE gave the final approval of the version to be published.

Competing interests None.

Patient consent Obtained.

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