British Journal of Anaesthesia **96 (1)**: 127–31 (2006)
doi:10.1093/bja/aei282 Advance Access publication November 18, 2005

Case Report

Tracheo-innominate artery fistula after percutaneous tracheostomy: three case reports and a clinical review

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Tracheo-innominate artery fistula (TIF) is an uncommon yet life threatening complication after a tracheostomy. Rates of 0.1–1% after surgical tracheostomy have been reported, with a peak incidence at 7–14 days post procedure. It is usually fatal unless treatment is instituted immediately. Initial case reports of TIF resulted from surgically performed tracheostomies. We present three fatalities attributable to TIF, confirmed by histopathology, after percutaneous dilatational tracheostomy (PDT). The use of PDT has resulted in tracheostomies being performed by specialists from different backgrounds and the incidence of this complication may be increasing. Pressure necrosis from high cuff pressure, mucosal trauma from malpositioned cannula tip, low tracheal incision, radiotherapy and prolonged intubation are all implicated in TIF formation. Massive haemorrhage occurring 3 days to 6 weeks after tracheostomy is a result of TIF until proven otherwise. We present a simple algorithm for management of this situation. The manoeuvres outlined will control bleeding in more than 80% of patients by a direct tamponade effect. Surgical stasis is obtained by debriding the innominate artery proximally, then transecting and closing the lumen. Neurological sequelae are few. Post-mortem diagnosis of TIF may be difficult, but specific pathology request should be made to assess innominate artery abnormalities.

Br J Anaesth 2006; **96**: 127–31

Keywords: complications, death; complications, fistula; complications, haemorrhage; surgery, tracheostomy; surgery, tracheotomy

Accepted for publication: October 17, 2005

Percutaneous dilatational tracheostomy (PDT) has become a standard technique in critical care medicine. As a result of its widespread use, it continues to attract controversy and debate. The UK Intensive Care Society recently launched the TracMan trial which concluded in its preface that PDT is 'a common procedure but with limited evidence base to support its use.' We present three fatalities resulting from tracheo-innominate artery fistulae (TIF) after PDT.

Although possible to perform under local anaesthesia, the majority of PDTs are performed under general anaesthesia with neuromuscular block on Critical Care Units by non-surgically trained operators. Complications of PDT are traditionally divided into early and late. The majority of publications centre on early complications including bleeding, pneumothorax, technical failure and perioperative hypoxia.[1] The major reported late complications include tracheo-oesophageal fistula, tracheomalacia and tracheal stenosis.[2]

TIF is an uncommon yet life threatening complication that can occur after a tracheostomy. Reported incidence is 0.1–1% after surgical tracheostomy, with peak incidence 7–14 days post procedure.[3 4] It is usually fatal unless treatment is instituted immediately.[5 6] We present a case-series of tracheo-innominate arterial fistulae after PDT with histopathological confirmation.

Case 1

A 43-yr-old female with a community-acquired pneumonia presented to the Critical Care Unit requiring invasive ventilatory support. Subsequent management included an uneventful PDT 11 days from her critical care admission.

Two subsequent uneventful tracheostomy tube exchanges were performed during the admission. Thirty-two days after this procedure, a small haemorrhage developed from the tracheostomy site. This was self-limiting and bronchoscopic investigation did not identify a bleeding site within the respiratory tract. Similarly, no signs of inflammation or infection were present in the tracheostomy wound. However, within 3 h of this initial bleed, massive oral and tracheal haemorrhage occurred. Bleeding did not initially appear pulsatile but the volume of blood prevented effective oxygenation. The trachea was intubated translaryngeally, allowing removal of the tracheostomy tube, inspection of the stoma and digital compression of the suspected bleeding site. Failure to terminate the bleeding and persistent suboptimal ventilation resulted in a fatal cardiac arrest.

Case 2

A 57-year-old male was admitted to the Critical Care Unit following a road traffic accident. His injuries consisted of a fractured pelvis, which required external fixation, and an extensive retroperitoneal haemorrhage. Inability to withdraw mechanical ventilation and an associated pneumonia led to an uneventful PDT on Day 11. Twelve days after the PDT, his respiratory function had improved sufficiently to allow consideration of decannulation. However, prior to this, sudden haemorrhage developed via the tracheostomy tube. Adequate ventilation failed because of the amount of blood in the major airways. Tracheal intubation was achieved via the translaryngeal route. It was not possible to identify a bleeding point and a double lumen endobronchial tube and bilateral intercostal drains were inserted. Unsuccessful attempts, using fibreoptic bronchoscopy, to suction the aspirated blood from the tracheobronchial tree followed. It proved impossible to re-establish effective ventilation, resulting in a fatal hypoxic cardiac arrest.

Case 3

A 69-yr-old female was admitted to the Critical Care Unit after an elective oesophagogastrectomy for oesophageal carcinoma. Her initial postoperative course was uncomplicated until Day 5 when she developed bronchopneumonia. On Day 8, inspiratory stridor and acute respiratory failure led to emergency tracheal intubation and mechanical ventilation. A PDT followed on the same day. Her subsequent management was unremarkable and artificial ventilation was discontinued by Day 10. She was discharged to the ward with a tracheostomy tube *in situ*. On Day 17, she developed massive haemorrhage into the airway resulting in desaturation and cardiac arrest. Despite translaryngeal intubation and prolonged attempts to clear the airway, resuscitation was not possible.

Each procedure was bronchoscopically guided and performed in the presence of an intensive care consultant. Ciaglia Blue Rhino® (Cook, Letchworth, UK) tracheostomy set was used and a size 8.0 mm Crystal Clear® (Rusch, Lurgan, UK) tracheostomy tube was used in each case. Each PDT was considered to have been between tracheal rings I and IV. All cuff pressures were monitored regularly, as is our standard practice.

The occurrence of these three cases within 18 months of each other aroused clinical suspicion of a potential link in underlying pathology. After direction from the clinicians involved, the subsequent post-mortem examinations looked specifically at both the tracheostomy site and the tracheo-innominate trunk.

As a result of their small size, post-mortem diagnosis of TIF can be difficult. For all three patients, sections from the tracheo-innominate artery at the level of the tracheostomy site revealed a small focus of active chronic inflammation extending through the full thickness of the arterial wall into the luminal surface. Histological evidence of necrosis was identified extending through the adjacent wall of the trachea with consequent focal disruption of the tracheal wall. The histopathological confirmation of a TIF was reported on the subsequent death certificates.

Tracheo-innominate fistula

The true incidence of this rare complication is difficult to assess. After surgical tracheostomy, the incidence has been estimated to be 0.1–1%.[4] Pooled data of 5530 tracheostomies recorded the incidence of delayed massive haemorrhage as 0.3%.[7] Initial case reports of TIF resulted from surgically performed tracheostomies. However, the incidence may have declined because of advances in tracheostomy tube technology and the introduction of PDT. The increased popularity of PDT has resulted in tracheostomies being performed by an increasing number of specialists from different backgrounds. It is conceivable that the rate of such a rare complication is perceived by different clinical groups, to be lower than it actually is. Consequently we suggest that TIF, although rare, should be borne in mind by all those involved in tracheostomy management. In our institution more than 1000 PDT have been performed since 1994, giving a crude incidence of 0.3%.

Pathophysiology

Knowledge of the anatomy of the innominate (brachiocephalic) artery (Fig. 1) and its relationship to a tracheostomy tube is essential in understanding the pathophysiology of TIF. The tracheo-innominate artery (or trunk) is the first branch of the aortic arch. It divides into the right common carotid and right subclavian artery, 3–4 cm lateral to the trachea, behind the right sternoclavicular joint. In its inferior proximal portion, its relations include:

- anterior: left tracheo-innominate vein and thymus;
- posterior: trachea (~6–10th ring);
- posterior and left: left common carotid artery;
- right: right tracheo-innominate vein, superior vena cava and pleura.

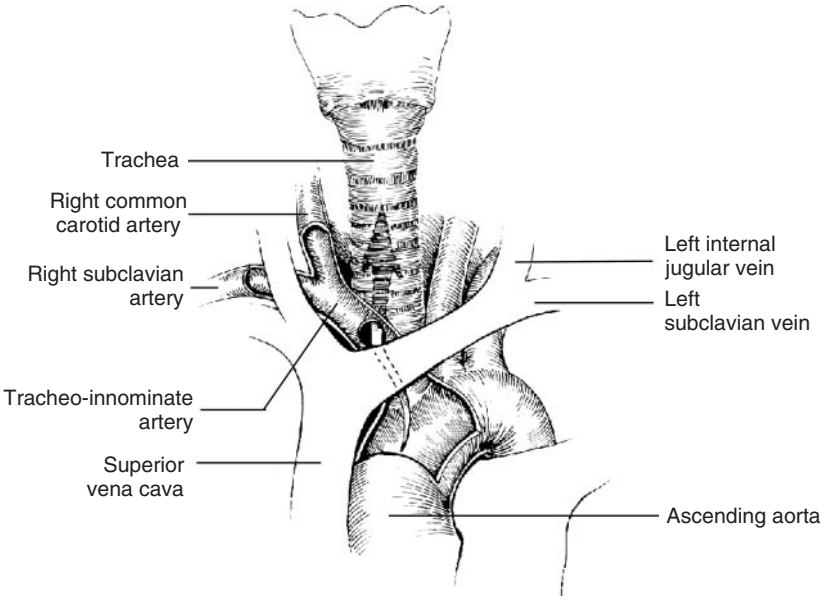

Fig 1 Tracheo-innominate artery fistula. *Source:* Amended from Wolfe: *Complications of Thoracic Surgery: Recognition and Management.* Figure 27-3 © 1992 Mosby, with permission from Elsevier.

The tracheo-innominate artery supplies blood to the right arm and the right side of the head and neck. Its absence on the left is explained by the direct branching of the left common carotid and subclavian arteries from the aortic arch. A high lying innominate artery, particularly in the thin and young, may act as a risk factor in fistula formation.

Aetiology

Pressure necrosis from high cuff pressure, mucosal trauma from malpositioned cannula tip, low tracheal incision, excessive neck movement, radiotherapy or prolonged intubation have all been implicated in TIF formation. Utilization of a high-volume low-pressure cuff may reduce subsequent fistula formation.

Two main mechanisms are capable of producing sufficient pressures to generate the erosive processes that lead to fistula formation:

- A fistula may occur between the anterior tracheal wall and artery. This is secondary to the mechanical force generated by either the tracheostomy tube cuff or tube tip depending on the relative positioning of the tube within the trachea.
- The second mechanism involves pressure generated beneath the angulated neck of a tracheostomy tube. This could produce ischaemia anteriorly on the tracheal mucosa and into the innominate artery.

Several authors suggest that a low lying tracheostomy tube is an obvious cause of fistula formation.[8] However, even when the tracheotomy incision is placed between the second and third tracheal rings, as recommended, these complications can still occur. In a post-mortem study, Oshinsky and colleagues[9] found that 10 standard vertical incisions placed in the second and third rings resulted in all subsequently placed tracheostomy tubes having either cuff or tip anatomically adjacent to the innominate artery, suggesting the potential presence of this complication in all patients with tracheostomies.

Diagnosis

Any peri-stomal bleed or haemoptysis should lead to a full clinical investigation to ascertain the underlying cause. A differential diagnosis for attending clinicians is based on the lag time between the tracheostomy and subsequent haemorrhage.

Haemorrhage within 48 h is typically associated with local factors such as traumatic puncture of anterior jugular or inferior thyroid veins, systemic coagulopathy, erosions secondary to tracheal suction or bronchopneumonia. Usually, the haemodynamic stability of the patient allows easy identification of the problem and corrective action to be taken with minimal morbidity.[10] Vascular erosion from a tracheostomy tube, resulting in a TIF, requires at least 48 h to develop even in the most friable mucosa.

Haemorrhage occurring 3 days to 6 weeks after tracheostomy should be thought of as a result of TIF until proven otherwise.[11] Other causes of catastrophic pulmonary haemorrhage include pulmonary artery flotation catheter induced arterial rupture, thoracic aneurysm rupture and less common vascular fistula (carotid artery, inferior thyroid). It is likely that the majority will occur in the Critical Care Unit, as 70% of all delayed haemorrhages occur during the first 3 weeks.[12] A sentinel bleed is reported in more than 50% of patients who then develop massive delayed haemorrhage.[6 8 13]

Haemorrhage occurring after more than 6 weeks is rarely related to TIF and more likely to be secondary to granulation tissue, tracheobronchitis or malignancy.

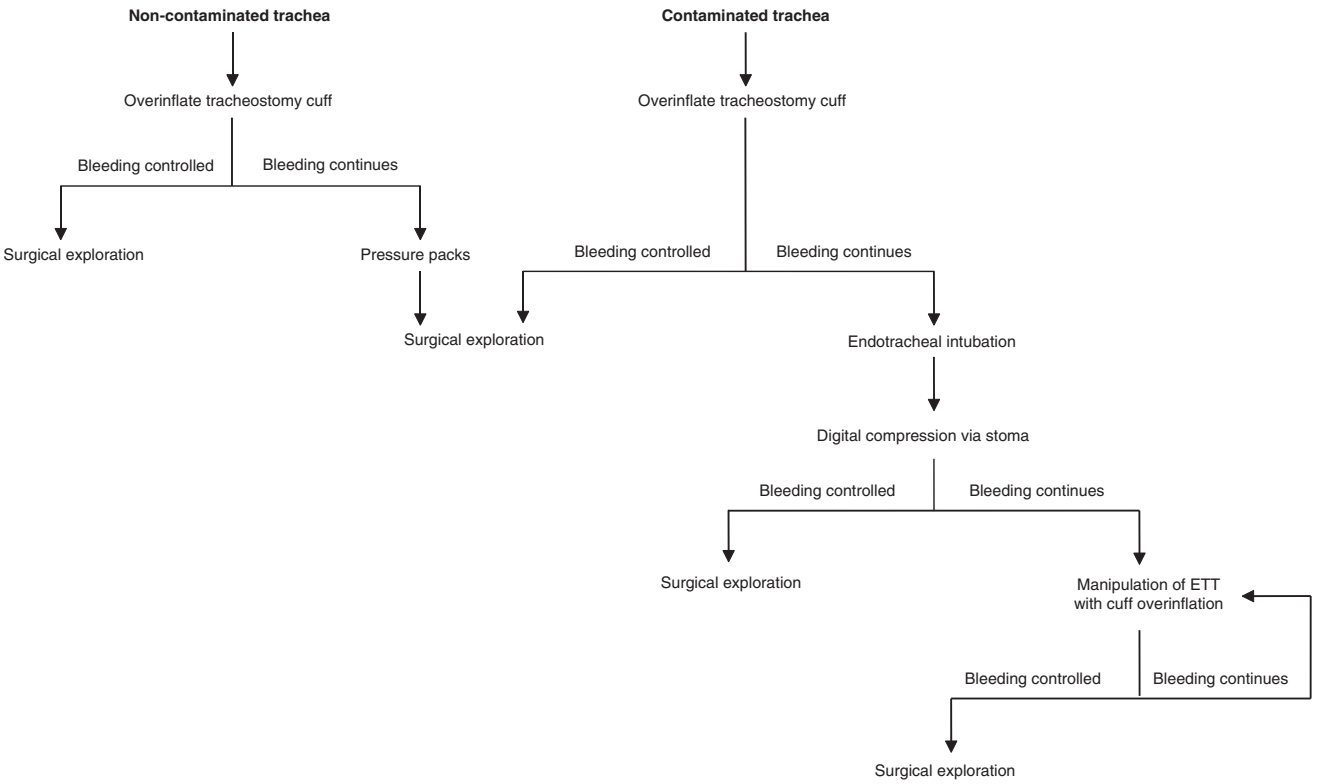

Fig 2 Clinical management of TIF.

Clinical management

Adequate oxygenation is the mainstay of immediate management with simultaneous identification and termination of bleeding. The basic principles of resuscitation, together with the management of early (within 3 days) bleeding, will not be considered here.

Management of a suspected TIF will depend upon whether there is active bleeding into the airway hindering adequate ventilation (Fig. 2).

We advocate the use of immediate bronchoscopy to confirm the extent and source of bleeding. Although bronchoscopy is unlikely to identify the fistula opening *per se*, it may exclude other pathology and allows direct monitoring of attempts to obtain a blood-free airway. Rigid bronchoscopy to clear the tracheobronchial tree of aspirated blood and to terminate blood flow is ideal, but this may not be possible.

Around 50% of TIF present with a self-terminating, sentinel bleed. If bronchoscopy confirms that the main bronchi are blood-free then immediate intervention is not required but further investigation to confirm the cause of bleeding is vital. Other causes of haemorrhage must be excluded before a sentinel bleed associated with TIF can be confirmed. If no other cause is evident then a provisional diagnosis of TIF should be made; urgent surgical advice and consideration of stoma exploration should follow.

If, in the presence of active bleeding, bronchoscopy indicates that the airway is clear of blood but there is ongoing external haemorrhage, the potential for catastrophic airway contamination is real. Overinflating the cuff provides additional airway protection and may control the bleeding temporarily. If, however, bleeding continues then pressure dressings should be applied to the stoma site. These manoeuvres temporarily control bleeding by a direct tamponade effect in more than 80% of patients.[5] As long as the airway remains free of blood; no attempt should be made to manipulate the tracheostomy tube. Immediate surgical exploration should follow.

Where bronchoscopy confirms that there is active bleeding into the airway, from the major threat is respiratory compromise rather than hypovolaemia.[14 15] Airway protection is the primary management aim. Movement of the tracheostomy tube may precipitate disastrous airway occlusion.[4] The aims must be to gain temporary control of the bleeding, get adequate oxygenation and proceed to immediate stomal exploration and definitive treatment.

(i) Overinflating the tracheostomy cuff is first line management. If this measure fails to reduce internal tracheal bleeding then proceed immediately to translaryngeal intubation with digital compression.[7 16] A cuffed oral tracheal tube should be advanced so that the balloon lies distal to the tracheostomy stoma (bronchoscopy should confirm ETT tip just proximal to the carina). The tracheostomy tube should only be withdrawn to facilitate simultaneous translaryngeal tracheal intubation. Digital compression consists of inserting the finger into the pretracheal space to tamponade the innominate

artery against the posterior surface of the manubrium.[11 12] This procedure should terminate bleeding in >90% patients and if maintained, allows transfer to the operating theatres.[6 17]

(ii) If digital compression fails to stem bleed, slow withdrawal of the tracheal tube and cuff over-inflation should follow. Manipulating the ETT tube, and its cuff to produce tamponade is the only other manoeuvre available in this situation and attempts should persist.

If the bleeding stops temporarily, and ventilation is acceptable, then immediate surgical intervention should follow.

Management of TIF is a surgical emergency. Mortality is ~100% without operative intervention. The paucity of evidence to support any imaging leads us to recommend the immediate surgical exploration. The index of clinical suspicion precludes delaying surgical intervention and any imaging, we feel, should only be undertaken within the theatre suite in conjunction with surgical management.

A standard median sternotomy approach is appropriate for access. It has been suggested that in specialist units, two separate incisions involving a right anterior thoracotomy and neck approach may be advantageous in terms of prevention of mediastinitis and sternal dehiscence.[13] Successful surgical interventions have included the use of saphenous vein and innominate vein grafts, sternocleidomastoid patches or pedicled pericardial grafts.[17] However, the mainstay of surgical treatment is to terminate flow within the innominate artery by debriding the innominate artery proximally until healthy tissue is obtained, then transecting and closing the lumen. There is no convincing evidence to suggest that this leads to significant neurological or vascular compromise.[18 19] Arterial reconstruction should no longer be considered, as arterial tie-off presents significantly better mortality and morbidity results.

In a patient presenting with fresh haemorrhage beyond 72 h after tracheostomy, the overriding concern should be of an underlying TIF. The simple algorithm that we have presented may be a useful guide. Our understanding of the long-term sequelae of the PDT is poor. We recommend that any death that results from, or is associated with massive haemorrhage from the respiratory tract in a patient who has received a PDT should have a post-mortem looking specifically at innominate artery pathology.

Acknowledgement

The authors thank Ms E. Walker for her help with preparation of the manuscript.

References

1 Ciaglia P, Graniero KD. Percutaneous dilatational tracheostomy. Results and long term follow up. *Chest* 1992; **101**: 464–672

2 Wood DE, Mathisen DJ. Late complications of tracheotomy. *Clin Chest Med* 1991; **12**: 597–609

3 Schaefer OP, Irwin RS. Tracheoarterial fistula: an unusual complication of tracheostomy. *J Intensive Care Med* 1995; **10**: 64–7

4 Allan JS, Wright CD. Tracheoinnominate fistula: diagnosis and management. *Chest Surg Clin N Am* 2003; **13**: 331–41

5 Bloss RS, Ward RE. Survival after tracheoinnominate artery fistula. *Am J Surg* 1980; **139**: 251–3

6 Courcy PA, Rodriguez A, Garrett HE. Operative technique for repair of tracheoinnominate artery fistula. *J Vasc Surg* 1985; **2**: 332–4

7 Dyer RK, Fisher SR. Tracheal-innominate and tracheal-esophageal fistula. In: Wolfe WG, ed. *Complications in Thoracic Surgery: Recognition & Management.* St Louis: Mosby Year Book, 1992; 288–306

8 Grillo CG. Tracheal Fistula to Brachiocephalic Artery. In: Grillo CG, ed. *Surgery of the Trachea and Bronchi*. Hamilton: BC Decker, 2003; ch. 13, 1–9

9 Oshinsky AE, Rubin JS, Gwozdz CS. The anatomical basis for post-tracheotomy innominate artery rupture. *Laryngoscope* 1988; **98**: 1061–4

10 Muhammad JK, Major E, Wood A, Patton DW. Percutaneous dilatational tracheostomy: haemorrhagic complications and the vascular anatomy of the anterior neck. A review based on 497 cases. *Int J Oral Maxillofac Surg* 2000; **29**: 217–22

11 Nelems JM. Tracheo-innominate artery fistula. *Am J Surg* 1981; **141**: 526–7

12 Bertelsen S, Jensen NM. Innominate artery rupture. A fatal complication of tracheostomy. *Ann Chir Gynaecol* 1987; **76**: 230–3

13 Jones JW, Reynolds M, Hewitt RL, Drapanas T. Tracheo-innominate artery erosion: successful surgical management of a devastating complication. *Ann Surg* 1976; **184**: 194–204

14 Hafez A, Couraud L, Velly JF, Bruneteau A. Late cataclysmic hemorrhage from the innominate artery after tracheostomy. *Thorac Cardiovasc Surg* 1984; **32**: 315–9

15 Gelman JJ, Aro M, Weiss SM. Tracheo-innominate artery fistula. *J Am Coll Surg* 1994; **179**: 626–34

16 Keceligil HT, Erk MK, Kolbakir F, Yildirim A, Yilman M, Unal R. Tracheoinnominate artery fistula following tracheostomy. *Cardiovasc Surg* 1995; **3**: 509–10

17 Utley JR, Singer MM, Roe BB, Fraser DG, Dedo HH. Definitive management of innominate artery hemorrhage complicating tracheostomy. *JAMA* 1972; **24**: 577–9

18 Brewster DC, Moncure AC, Darling RC, Ambrosino JJ, Abbott WM. Innominate artery lesions: problems encountered and lessons learned. *J Vasc Surg* 1985; **2**: 99–112

19 Deslauriers J, Ginsberg RJ, Nelems JM, Pearson FG. Innominate artery rupture. A major complication of tracheal surgery. *Ann Thorac Surg* 1975; **20**: 671–7