Fatal haemothorax following large-bore percutaneous cannulation before liver transplantation

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Percutaneous bypass catheters are routinely used for veno-venous bypass (VVBP) during orthotopic liver transplantation (OLT). The recognized risks include bleeding, injury of vascular and nerve structures and lymphatic leakage. We describe a case where there were difficulties during catheterization and the patient suffered a cardiac arrest on commencing VVBP. Post-mortem examination revealed the bypass catheter tip in the pleural space and a large right haemothorax. Possible mechanisms of vascular perforation and preventative measures are discussed.

Keywords: complications, haemothorax; surgery, liver transplantation; veins, central venous cannulation

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Venovenous bypass (VVBP) was first introduced to the field of orthotopic liver transplantation (OLT) in 1984 in order to maintain venous return when inferior vena cava (IVC) is cross clamped, and therefore minimize haemodynamic alterations and preserve renal function during anhepatic phase.1,2 Percutaneous bypass cannulae have been used since 1991.3 These are usually placed in the femoral, the right internal jugular or the axillary vein. Although the technique is described as rapid, simple, and safe, complications such as massive bleeding, injury of vascular and nerve structures, and lymphatic leakage contribute to morbidity and mortality.4 We describe a fatal complication, which occurred on commencing VVBP.

Case report

A man of 62 yr of age, BMI 32 kg m⁻², with pre-existing asthma and non-insulin dependent diabetes was presented for OLT for cryptogenic cirrhosis. He was considered to be at intermediate risk for liver transplantation (15% mortality). Standard monitoring (ECG, pulse oximetry, non-invasive arterial pressure) was applied and an i.v. cannula inserted. Anaesthesia was induced with propofol and vecuronium. Following tracheal intubation, anaesthesia was maintained with desflurane and analgesia provided with remifentanil. An intra-arterial catheter was placed in his right radial artery.

The local protocol included insertion of a 4-lumen central venous catheter (Quad-Lumen with blue flex tip, Arrow International, Reading, USA), 8.5 Fr Arrow percutaneous Sheath Introducer (Reading, USA) and two 21 Fr (7-mm internal diameter) percutaneous bypass catheters (Femoral Arterial Cannula, Medtronic, MN, USA), one into the right internal jugular vein and the other into the femoral vein (Fig. 1).

Under aseptic conditions, with the patient in the Trendelenburg position, the head rotated 30° from the midline, the right internal jugular or the axillary vein. Although the technique is described as rapid, simple, and safe, complications such as massive bleeding, injury of vascular and nerve structures, and lymphatic leakage contribute to morbidity and mortality.4 We describe a fatal complication, which occurred on commencing VVBP.
Aspiration of blood and easily flushing with saline suggested the correct placement of the intravascular catheter. As a result of the technical difficulties experienced with the removal of the guide-wire, a chest X-ray was performed. While waiting for chest X-ray, another 21 Fr bypass catheter was placed in the left femoral vein uneventfully.

The chest X-ray showed the bypass catheter descending into the right hemithorax close to the right mediastinal margin. No signs of pneumothorax or pleural effusion were present (Fig. 2). The positions of all lines on chest X-ray were considered satisfactory, and a decision was made to carry on with the operative procedure.

The 4-lumen central venous catheter was used for the measurement of central venous pressure, infusion of dopamine, remifentanil, parvolex, and other drugs. The percutaneous Sheath Introducer was used for fluid infusion and the bypass catheter was connected to a rapid infusion device, but not subsequently used for that purpose.

By the time the surgery was started, the following monitoring was established: ECG, invasive arterial pressure, central venous pressure, core temperature, end-tidal carbon dioxide concentration, peripheral oxygen saturation, pressure–volume curves, fractional inspired oxygen, end-tidal anesthetic agent, tidal volume, ventilatory frequency, I:E ratio, and PEEP. A non-invasive LiDCO cardiac output monitor provided cardiac index, mean arterial pressure, heart rate, systemic vascular resistance, and stroke volume (LiDCO plus, LiDCO, Ltd, London). Urine output, arterial blood gases, and coagulation status were also monitored using a Thrombelastograph.

The vital signs remained within the normal ranges during the initial dissection phase. In the minutes running up to the period of bypass, the patient became hypotensive requiring fluid and vasopressor support. No immediate explanation for this was forthcoming. By this time, the patient had already received 6 u of fresh frozen plasma (FFP) and 500 ml of gelofusine.

The patient was then commenced on VVBP. We used Biomedicus Pump Model 540 (Biometric, Medtronic, Minneapolis), which was run by the operating department practitioner trained for VVBP pump. This entails blood being pumped at a flow of 2 litre min \(^{-1}\) from the iliofemoral to internal jugular vein. Immediately the patient became more hypotensive with a systolic pressure of 50 mm Hg and an elevated central venous pressure of 23 mm Hg. This was immediately followed by cardiac arrest, which proved resistant to cardio-pulmonary resuscitation and defibrillation, both performed by the external route. During the cardio-pulmonary resuscitation bypass was running. Attempts at resuscitation continued for 30 min but there was no return of spontaneous cardiac output. The pupils became fixed and dilated. A decision was then made to terminate resuscitation and the patient was declared dead. The bypass device was switched off. The family were counselled and the case referred to the coroner for further investigation.

Post-mortem examination revealed that the large-bore bypass catheter, inserted via the right internal jugular route, had perforated the superior vena cava with approximately 4 cm of distal catheter lying in the right pleural space. The pericardium was intact. There was 1.8 litre of blood in the right pleural cavity (haemothorax) and 0.7 litre of bloodstained effusion in the left pleural cavity (Fig. 3).
There were multiple left-sided rib fractures secondary to cardiac massage during attempted resuscitation.

**Discussion**

There are some 10 000 adult liver transplants performed in USA and Europe each year, with VVBP used in approximately 50% of cases. In the UK there were 680 procedures performed in 2004. There are no data about the incidence of serious complications related to bypass catheter insertion. Previously published reports of percutaneous cannulation detail small series. Two further studies reported a single fatality in a series of 312 and 94 cases related to VVBP catheters. There is also one case report of a central venous catheter-related fatal complication in a paediatric liver transplant patient. If there is up to 1/100–300 complications related to VVBP and approximately 5000 OLT performed on VVBP all over the world per year, the number of potential serious complications and deaths is significant.

The exact mechanism of injury in this case is not entirely clear. Positioning the patient and puncture was similar to that described in recent publications. The catheter used was a standard device utilized for bypass procedures. A number of potential mechanisms are suggested.

The guide-wire was initially passed into the internal jugular vein but its tip, instead of passing straight down into the superior vena cava and right atrium may have turned laterally into the subclavian vein. The larger-bore vein dilator and catheter was then unable to follow the course of the guide-wire into the subclavian vein. Instead on pushing inwards the dilator, bypass catheter, or guide-wire caused a tear in the superior vena cava and entered the pleural space on the right. The fact that the guide-wire was distorted and difficult to remove suggest that dilators were not naturally following its initial course. Similar mechanisms of injury could occur if the guide-wire passed through one of the tributaries of the brachiocephalic vein or superior vena cava (e.g. pericardiophrenic, internal mammary, or azygous veins). The portal to systemic collateral circulation, as present in OLT patients, would dilate such tributaries and increase the risk of malposition.

Should the reversed J-wire with the rigid end have been used? Most commonly, resistance to guide-wire advance may be a result of (partial) occlusion of the vein by thrombus, altered anatomy, incidental puncture of the smaller vein, and puncture of an artery. Sometimes, a soft J-wire tip becomes distorted after initial pass through the needle. In such cases a new guide-wire should be used rather than using the reverse stiff tip.

Some force is required for large-bore catheter insertion to pass the device through superficial structures and the vein wall, but how does the operator gauge what is excessive force in any particular patient? It can be argued that the obturator should only be passed just far enough into the vein to dilate it; thereafter, the blunt tipped softer catheter should be advanced further into the great vessels. Kinked guide-wires suggest malposition. Free passage of guide-wires in and out of the dilators needs to be checked repeatedly. Catheters and dilators are often longer than required. The 17 cm catheter used in this case would lie well within the right atrium in most cases when fully inserted from the right internal jugular vein. The 60 cm long polyurethane dilator is certainly excessively long for jugular placement and presumably is designed for longer bypass cannula, which pass from a femoral puncture up the IVC to the right atrium.

The pathology report was not precise and on later questioning the pathologist could not recall the exact site of perforation. It stated that the vein was perforated posteriorly, with the distal end of the catheter (approximately 4 cm) lying free in the pleural space on top of the pericardium.

**Fig 4** Artistic impression of one potential mechanism of injury. (A) Misplaced guide-wire in the subclavian vein. (B) The stiff dilator cannot curve enough to follow the guide-wire. The wire is kinked and the vessel torn. (C) The tip of the bypass catheter is inserted to lie outside the vessel. RSV, right subclavian vein; SVC, superior vena cava; BC, brachiocephalic vein; RIJV, right internal jugular vein; GW, guide-wire.
This patient took some time to deteriorate clinically despite presumed bleeding into the pleural space from the tear in the brachiocephalic vein and superior vena cava before bypass commenced. This blood loss may have been compensated by the FFP infusion given through another wide-bore catheter line within the superior vena cava. When VVBP was commenced, blood was diverted from the femoral into the internal jugular vein and the pleural cavity under a pressure of 200 mm Hg causing a massive haemothorax and cardiac arrest. During the cardiopulmonary resuscitation bypass was running. Vessel wall compression around the femoral vein cannula, as a sign of the low flow in femoral vein, was not noticed. Similarly, increased pressure within the VVBP tubes as a sign of the back pressure (increased intrapleural pressure could produce back pressure) was not noticed.

It is well documented that flexion and extension of the neck can cause a central venous catheter to migrate several centimetres. In this case movement of the patient with cardiac massage may have pushed the bypass cannula even further in the thorax. If the tip of the bypass line had been initially 4 cm out of blood vessel, blood loss through the 7 mm diameter cannula and 16 side holes should have been rapid, and the patient might have been expected to show clinical signs of bleeding earlier.

Could the use of ultrasound (US) guidance have prevented vessel rupture? Although these devices improve the safety of central venous catheter placement, they may not prevent subsequent malposition or vascular perforation. However, they can be used to visualize a guide-wire passing downwards into the innominate vein rather than turning into the ipsilateral subclavian vein.

Could the misplacement of the catheter have been recognized earlier? Ease of blood withdrawal and flushing of the cannula may not detect that the cannula has perforated a vessel or is lying outside a vessel. In our patient, presumably venous bleeding was occurring through the proximal holes and out through the more distal holes into the pleural space. Aspiration of blood could occur through either the proximal or distal holes giving confusion as to the placement of the catheter.

Similarly, monitoring central venous pressure as a test for intravascular placement of large-bore catheters may not detect partial extravasation. Any measured central venous pressure may reflect pressure from the proximal holes or the distal holes and be difficult to interpret. In this case we measured central venous pressure from the four lumen central venous catheter, which tip was proximal to the tip of the bypass line.

The limitations of a supine plain chest X-ray in terms of catheter positioning should be appreciated. In our case, it was an AP supine film with poor lung volumes. The position of the superior vena cava, which lies immediately adjacent to the medial right pleural border, means that it is impossible to distinguish catheter placement between the two. A pleural collection should raise suspicion of catheter misplacement. Injected X-ray contrast leaking into the pleural space should be diagnostic. However, contrast injected would go down the path of least resistance, which is perhaps likely to be the proximal holes, particularly if the patient was on positive pressure ventilation. Furthermore, continuous fluoroscopy is not always available at short notice in an operating theatre.

Could transoesophageal echocardiography (TOE)-guided placement of internal jugular percutaneous VVBP cannula prevent the described complication? That technique has been described recently and is still not in routine use. It may be useful in detecting complications arising during cannula placement (tricuspid valve damage, bilateral fluid collections, severe hypovolaemia, pericardial tamponade, or mediastinal haematoma). The use of TOE in OLT could be limited because of the presence of oesophageal varices.

Should the procedure be abandoned if percutaneous catheter insertion proves difficult at any time during the procedure or there are doubts about catheter position? This case report suggests that the risk of VVBP lines could be minimized by a detailed knowledge of the complications of catheterization, a high index of suspicion that clinical deterioration may be a result of the catheter and a readiness to take the action. There may be other clinical signs that could indicate displacement of the bypass catheter. In a previously reported fatal complication of central venous cannulation in a paediatric liver transplant patient, it was left hemidiaphragm bulging into the abdomen noticed by the surgeon that indicated haemothorax. Hypotension, decreased respiratory volumes, congested face and neck with normal central venous pressure were also present.

An understanding of the risks of bypass should be appreciated. If a patient deteriorates when they are put on bypass, the bypass pump should be stopped to reassess the situation. The use of bypass may induce pericardial tamponade if the catheter is in the pericardial space, may produce air or thrombus embolization if conditions are right, or produce a haemothorax if there is a leak into the pleura as in this case. In this case the bypass pump was only stopped after the patient was declared dead.

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