CLINICAL INFORMATION

Cardiac tamponade: a rare complication of central venous catheter – a clinical case report

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Abstract The extensive use of central venous catheters (CVC) in a hospital environment leads to increased iatrogenic complications, as more catheters are used enclosed and its maintenance is prolonged. Several complications are known to be related to central venous catheter, of which the uncommon cardiac tamponade (CT), hardly recognized and associated with high mortality.

We present a clinical case, with favorable outcome, of a patient who developed a CT 17 days after CVC placement, and try to reflect on the measures that can be taken to reduce its incidence, as well as the therapeutic approaches to practice in the presence of a suspected CT.

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Introduction

Central venous catheter (CVC) is widely used in hospitals for central monitoring; placement of temporary pacemakers; administration of fluids, blood products, parenteral nutrition or drugs (antibiotics, vasopressors, chemotherapy, and others). This extensive use of CVC prompts an increase in iatrogenic conditions associated with this technique, as more catheter are being placed and kept longer.\(^1\)\(^2\)

Complications of central venous catheterization are known and cardiac tamponade (CT) is a rare and hardly recognized complication associated with high mortality.\(^3\)\(^4\)

Based primarily on case reports, its incidence ranges from 0.001% to 1.4% and its associated mortality from 65% to 100% in adults; in children, the incidence is higher (1–3%) and mortality is lower (30–50%).\(^5\)\(^6\)

The signs and symptoms of CT, in addition to being unspecific, may arise within few minutes after CVC insertion or up to five months after placement,\(^3\)\(^5\)\(^7\) which explains the missed or late diagnosis and the fate of many of the described cases.

We present a case with a favorable outcome in a patient who developed a CT 17 days after CVC placement in the right internal jugular vein.

In this article, we will discuss the factors that increase the risk of this complication, the measures that can be adopted in our protocols to reduce the incidence, and the therapeutic approaches to use when faced with a suspected CT.

Clinical case

Female patient, 26 years old, 150 cm tall, 55 kg, ASA IV, admitted for elective left nephrectomy due to pyonephrosis refractory to antibiotic therapy. The patient relevant clinical history included polycystic kidney disease (hydrocephalus, spina bifida, interventricular communication, vesicoureteral reflux, neurogenic bladder, dysfunctional colon, and imperforate anus), with various surgical repair interventions, epilepsy, and chronic renal failure on hemodialysis (renal transplantation in 2001). Laboratory tests showed hemoglobin – 102 g.L\(^{-1}\); hematocrit – 0.311 L.L\(^{-1}\); platelets – 193 \times 10^9 L\(^{-1}\); prothrombin rate – 60%; aPTT – 45.4 s; INR – 1.45; creatinine – 9.8 mg.dL\(^{-1}\). Electrocardiogram (ECG) showed sinus tachycardia with a frequency of 106 beats per minute.

The patient had no peripheral venous access, reason why a CVC was placed before the induction of general anesthesia. The right internal jugular vein was punctured at first attempt by an experienced anesthesiologist. A 3-lumen catheter, 15 cm long (Certofix\(^\text{®}\) Trio; B\(\)BRAUN) was inserted using the Seldinger technique, without any changes in ECG. The correct insertion into the venous system was confirmed by ultrasound visualization of the needle, guidewire and catheter and free aspiration of dark red blood through the three lumens. During surgery, the patient required vasopressor support with norepinephrine, blood transfusions (two units of fresh frozen plasma and seven units of cryoprecipitate), in addition to fluid maintenance/blood loss replacement, an estimated 1000 mL of infused fluids through the CVC.

A chest X-ray was performed at the Post-Anesthesia Care Unit (PACU) immediately after surgery (Fig. 1), with intracardiac view of the catheter tip. Due to the poor technical conditions of radiography, the finding was neglected and the catheter was not externalized.

The patient returned to the operating room seven days later due to a retroperitoneal hematoma, in the nephrectomy bed, without active bleeding focus. During surgery, the patient required vasopressor support with norepinephrine and, through the same CVC, two units of packed red blood cells and isosmolar fluid were infused, with a volume of 1200 mL, uneventfully.

Figure 1 Postoperative chest X-ray performed after CVC placement in the right internal jugular vein.
During hospitalization, 14 days after CVC placement, the patient developed dyspnea and precordial pain, and another chest X-ray was performed (Fig. 2), again without appraising the tip of the CVC positioning. At day 16, the patient presented with hemodynamic instability with significant worsening on the 17th day and progression to cardiopulmonary arrest (CPA) with asystole, with return of spontaneous circulation 10 min after the start of advanced life support (ALS).

Immediately after transfer to the Intensive Care Unit (ICU), she had a new episode of bradycardia with progression to asystole, with pulse recovery after five minutes of ALS. Subsequently, a transthoracic echocardiogram showed CT and the CVC tip into the pericardial space. We contacted the cardiology department, and pericardiocentesis was performed, with immediate output of 400 mL of hematic liquid, resulting in improved hemodynamics. Pericardial drainage was performed with output of 750 mL of hematic liquid in 24 h. The jugular CVC was removed after a new chest X-ray, which confirmed the intracardiac placement of the catheter tip (Fig. 3).

The patient was discharged from the ICU two days later, hemodynamically stable, with spontaneous breathing and without sequelae.

**Discussion**

A study based on 6440 medical records of complications associated with medical practice, performed between 1979 and 2000, reveals that 110 (1.7%) of such complications were related to CVC placement and that of 16 registered CT, 13 were associated with prolonged use of catheters, with a high mortality rate (81%) compared to other complications.

In fact, CT caused by central venous catheterization is a well-documented entity, with clinical cases reported since 1958. However, its variable incidence in the literature remains low because CT cases are not recognized or not disclosed.

There are different mechanisms involved in post-CVC CT: direct trauma at the time of insertion, catheter migration, and mechanical and chemical erosion.

Upon insertion, the guidewire (despite its flexible J-tip), introducer or catheter may cause a detachment of the superior vena cava (SVC) wall at the atrium junction (SVC intrapericardial portion) or perforation of the heart chambers and frequently causing acute symptoms.

Noteworthily, the complications directly resulting from CVC insertion are closely related to the physician’s experience. Inexperienced physicians have failure rates with the technique and complications that are twice those of experienced physicians. CVC material (polyethylene, silicone or polyurethane) also has influence on CT origin. The more stiff and less flexible it is, the greater the risk of perforation.

CVC repeated contact with the endocardium leads to thrombus formation and CVC adherence to the myocardium, myocardial wall erosion, necrosis, and perforation up to the pericardial space, causing CT after several days. Perforation is more likely to occur if the catheter tip is located in the heart, in contact with the wall, because the cutting edge moves in correspondence with the respiratory movements and cardiac contractility. The angle formed by the catheter tip and the vessel or heart wall is also a major risk factor. The more perpendicular the angle is (>40°), the greater the possibility of erosion and necrosis perforation of the vessel intima or heart wall, which is more evident when CVC is placed via subclavian or left internal jugular vein due to its more tortuous path.

Chemical erosion happens when hyperviscous fluids in contact with the endocardium lead to erosion and osmotic injury perforation, with transmural diffusion of fluids leading to pericardial effusion. From the moment in which there is hypertonic fluid present in the pericardial space, there will be a rapid build-up of fluid by osmotic gradient.
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The most common sites of perforation are right atrium (RA) and right ventricle (RV) in 80% of cases, followed by the SVC. There is also reports of left atrial (LA), SVC/RV junction, and left subclavian vein perforations.

CT may not always be prevented, but certain measures can reduce its incidence and mortality. Our conduct during central venous catheterization can be changed and some precautions can be taken.

A catheter with the shortest length possible, appropriate to the clinical situation, should be selected. According to the literature, catheters with 15–16 cm significantly minimize CVC intracardiac placement via subclavian or jugular vein.

Whenever possible and available, real time ultrasound should be used to guide the insertion of CVC via internal jugular vein, in order to improve the success rate and reduce the incidence of complications. Regarding catheter placement via subclavian or femoral vein, the evidence is ambiguous to recommend its routine use. The use of ultrasound has been suggested for vein and local anatomy screening prior to puncture (static ultrasound), particularly in high risk patients.

At the time of puncture, the blood color or absence of pulsatile flow should not preclude the later confirmation of the CVC location.

The catheter tip position may be confirmed by three methods: chest X-ray, fluoroscopy, and endocavitary ECG. The major advantage of fluoroscopy and endocavitary ECG is the possibility to confirm the initial position of the catheter tip on the bedside monitor. The literature reports that placing a CVC without a fluoroscopic or continuous ECG monitoring of the catheter tip results in at least 15% of erroneously positioned catheters. In the operating theater of our hospital, the protocol requires a chest X-ray in the immediate postoperative period.

It is also important to check the blood backflow through the CVC lumens and, particularly, aspirate the blood from the distal lumen to confirm its position in the vascular system, and perform it regularly and aseptically whenever the catheter is used. Noteworthy, the distal lumen should not be used to infuse hyperosmolar solutions.

The movement of arms, head, neck, and trunk exacerbated by the large anatomic variations in the VCS length may cause the catheter migration. So it must be securely fixed to the skin.

Several anatomical landmarks have traditionally been used to assess the proper final position of the catheter tip using radiological control. However, although there is no consensus on its position in the SVC, its intracardiac location should certainly be avoided because it is associated with higher mortality.

Regarding diagnosis, 36% of the CT secondary to CVC placement occur within the first 24 h, which suggests that the perforation occurred at the time of insertion and not by migration or erosion, and 82% occur in the first week after CVC placement.

The symptoms include chest or epigastrum pain or discomfort, nausea, dyspnea, tachycardia, engorgement of neck veins, paradoxical pulse, hypotension, low ECG voltage, and increased cardiac silhouette. The classic Beck’s triad (hypotension, muffled heart sounds, and jugular engorgement) is not present in over 29% of cases, and death from cardiovascular collapse may be sudden with "vague" signs/symptoms.

In any patient with CVC, presenting with chest pain and any of the above mentioned symptoms, a CT should be suspected until proven otherwise. At this stage, an ECG may not show changes; a chest X-ray will show changes only if a significant amount of fluid is accumulated in the pericardial space; an echocardiogram that, although a diagnostic test, is not always promptly available; or any other test that helps the diagnosis must be dismissed to avoid treatment delay.

A successful CT approach depends on early diagnosis and prompt and proper treatment. So, if the probable diagnosis of CT is admitted, the catheter infusion should be immediately stopped. The infusion container must be lowered below the level of the patient’s heart, and any pericardial content must be carefully aspirated through the catheter. Note that the backflow of blood through the CVC does not exclude the CT diagnosis, particularly if the CVC tip is already in the pericardial space. CVC should be carefully removed and an echocardiography performed. If CPA is imminent, emergency pericardiocentesis should be performed and, as a last resort, thoracotomy.

In this clinical case, the catheter selection was adequate (15 cm flexible polyurethane catheter); the procedure was ultrasound-guided monitored with ECG, without causing arrhythmia by the guidewire; and the backflow of blood was observed through the three lumens of the catheter, which confirmed CVC position in the vascular system. However, the chest X-ray performed in the immediate postoperative period showed the catheter tip within the heart, but as it was an anteroposterior incidence and technical conditions were bad, the finding was underestimated. The catheter permanence for 17 days in an anomalous position (possibly in RA), in a patient with polycystic kidney disease and several previous surgeries, resulted in the CT by an increasing mechanical erosion mechanism and migration to the pericardial space.

Although the causes of pericardial disease, and consequently CT, are very diverse, in this case seeing the catheter tip in the pericardial space defined the etiologic diagnosis of CT. There were no objective clinical, analytical or imaging criteria to support other causes of subacute CT, especially complications from medical conditions, such as infectious pericarditis, acute myocardial infarction (Dressler’s syndrome), congestive heart failure, uremia, inflammatory bowel disease, hypothyroidism, connective tissue diseases, metastases, or drug side effects (procainamide, isoniazid, hydralazine, minoxidil, phenytoin, anticoagulants, methysergide).

Interestingly, a multicenter US study of intensive care units found intracardiac placement of CVC tip in 47% of patients and that the CVC position was not corrected after the chest X-ray. These numbers and our reported case alert us for a careful consideration of the control X-rays after CVC placement, in order to detect anomalous positions and effectively correct it after visualization.

However, we consider that the importance of the catheter tip exact position in the chest radiography is overestimated in the literature, because no position will be realistic in anteroposterior radiographs and even if patients are immobile, the physiological movements of the cardiac and respiratory cycle may change the catheter tip position at any time.
In fact, there is no evidence in the literature that radiography can prevent late complications such as CT. Moreover, we advocate that the CVC position should be confirmed by radiography and should be reviewed whenever chest X-rays are performed for other reasons, mainly to prevent intracardiac placement and hidden catheter migration.

Conclusion

The selection of more flexible and short catheters should be an option. Real-time ultrasound reduces the number of complications associated with the technique, but it does not allow the catheter tip view or prevent late complications. Fluoroscopy and endocardial ECG, if available, are effective methods to identify the catheter tip initial position at the bedside, but should not dismiss control radiographs. Radiologic confirmation of the catheter tip position is an important measure to prevent intracardiac placement, but it does not guarantee that vessel or heart wall erosion or migration occur, so its position should be reviewed whenever chest X-rays are performed for other reasons. The backflow of blood through lumens should be checked whenever fluids or drugs are infused through the catheter.

CT should be considered in all patients with CVC, presenting with deterioration in their clinical condition. The key to therapeutic success depends on early clinical suspicion, and echocardiography is a surplus in confirming the diagnosis.

Conflicts of interest

The authors declare no conflicts of interest.

References