Contents lists available at ScienceDirect

Trauma Case Reports



journal homepage: www.elsevier.com/locate/tcr

Hickman catheter-induced cardiac tamponade-related cardiac perforation management by mediastinotomy in children and a review of the literature

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ARTICLE INFO

Keywords: Cardiac perforation Hickman catheter Insertion Complication Cardiac tamponed

ABSTRACT

Objective: Hickman catheter placement rarely causes cardiac tamponade due to cardiac perforation in children. Cardiac perforation can be managed with timely perceive and appropriate approach. We present a case of the cardiac perforation related to Hickman catheter insertion in a 7-year-old girl and a review of the supporting literature.

Case report: The patient had previous history of Thalassemia and admitted to hospital for Hickman catheter placement for bone marrow transplantation. The catheter was placed in the right internal jugular vein by ultrasonography. During the postoperative period she had hypoxia, hypotension and tachycardia. The patient underwent an emergency surgery and there was a small perforation between vena cava superior and right atrium. That wound was managed by mediastinotomy. The patient was discharge on the 8th postoperative day.

Conclusion: Cardiac perforation is one of the rare and unexpected complications of Hickman catheter placement. Unfortunately, the complication may cause significant mortality. The prompt diagnosis and early intervention provides satisfactory results.

Introduction

The Hickman catheter (HC) affords many advantages, including application of intense therapy via lumina of different diameters if medications are incompatible and infusion of large fluid volumes. HCs are often inserted via the jugular or subclavian veins of pediatric patients [1–3]. However, HCs may be associated with various complications, one of which is cardiac tamponade (CT)-related cardiac perforation (CP) [4]. Complications associated with HC insertion are similar to those associated with other central catheters and include arterial puncture, CP, hemo-/pneumothorax, thrombosis, and infections [5]. The mechanical complication such as; vascular issues and CP are the most serious life-threatening complications after central venous catheter insertion [6].

Findings indicative of problems with HC insertion include pulsus paradoxus, hypotension, tachycardia, and/or severe dyspnea. The goals of treatment are to restore the hemodynamic balance, drain any pericardial effusion, prevent complications, and improve the hemodynamics. The treatment options are limited and frequently unsuccessful. In the absence of prompt treatment, CP may cause cardiogenic shock, cardiac arrest, or death.

Here, we describe a case of HC insertion that triggered acute-onset CT caused by CP.

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https://doi.org/10.1016/j.tcr.2021.100436

Accepted 12 February 2021 Available online 18 February 2021 2352-6440/© 2021 Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).



Case report

A 7-year-old girl required percutaneous HC insertion after bone marrow transplantation. She had a medical history of thalassemia. The procedure was performed under general anesthesia employing guided ultrasonography (USG). After puncture of the right vena jugularis interna, a J guidewire was placed feasibly, followed by a pleural sheet and 9-Fr catheter. After recovering from surgery, she was hemodynamically stable and was transferred to the clinic for postoperative follow-up. Soon thereafter, the patient then became hypotensive and developed dyspnea and tachycardia 4 h after HC insertion. There was no bleeding. Blood analysis showed a no decrease in the Hb level (to 8 g/dL) and increasing leukocytosis (leukocyte count, 27,000/mL). Blood gas analysis revealed low oxygen saturation, increased CO₂ pressure, and acidosis. The symptoms progressed. On a chest X-ray, the catheter tip was observed within the heart, and the cardiac silhouette was enlarged and exhibited massive bilateral pleural effusion (Fig. 1a). Thorax and abdominal USG revealed massive pulmonary effusion and free fluid in the abdomen. Echocardiography revealed a large amount of pericardial effusion. Computed tomography revealed massive bilateral pleural effusion and massive amounts of free fluid in the abdomen (Fig. 1b). We considered the possibility that a cardiac or large-vessel perforation had caused CT. Red blood cells and fluid were infused. The patient was rushed to the theater, and sternotomy was performed via a midline incision approximately 8 h after HC insertion. Approximately 600 mL hemorrhagic fluid were distributed diffusely around the heart and inside the pericardial recesses; a 0.5-cm-diameter perforation lying between the right atrium and vena cava superior was closed, a drain placed in the pericardial area, and the HC removed percutaneously. On postoperative day 2, the pericardial drain was removed (Fig. 2). On postoperative day 8, she was discharged without any complications.

Discussion

We searched the literature using the PubMed MEDLINE database. We searched titles and abstracts using a combination of the terms "cardiac perforation", "central venous catheter", and "children". No data limit was imposed. We evaluated papers on 20 cases, including our case, published in the English language. Most of these reports referred to neonates (most of whom were of low birth weight, female, and premature) [7] and infants treated in pediatric intensive care units [8]. Patients with chronic diseases, and newborns, are more likely to have weak myocardial muscles and are therefore at a higher risk of perforation [5]. Our patient had thalassemia, and had received many blood transfusions. Most cases were diagnosed postmortem, and all had catheter tips within the cardiac silhouettes. CT had developed late after catheter insertion; some patients survived after pericardiocentesis or mediastinotomy. When we included guidewires in our search, all three reports referred to adults [5,14]. We excluded adult cardiac perforations caused by catheters (Table 1) [3,7,10,13,18–22]. This is the first reported case of CP caused by the J-tipped guidewire of an HC in a patient with thalassemia. In a previous study, HC insertion caused rapid CT in an adult who died [9]. We followed our patient closely, and early diagnosis allowed rescue.

Complications associated with the use of HCs are early or late is a serious but rare complication [8,10]. Complications can be divided into those that develop early (<24 h after HC insertion; hemothorax, pneumothorax, catheter malpositioning, and CP) and those that develop late (>24 h after insertion; sepsis and thrombosis) [10,11]. Early complications are usually mechanical in nature and more serious than late complications [10,11]. The complication rate associated with central catheter insertion in children is 2.4–4.6% [1]. The CT incidence is quite variable, ranging from 0.0001 to 1.4% [2,10–12]. CT constitutes approximately 5% of all mechanical complications [4,17]. Mortality caused by CT also varies (30–100%) [3,10,11,13]. Diagnosis is usually not determined until postmortem [13]. We inserted more than 500 HCs over a 5-year period and encountered only this single CT incident. The time from central catheter insertion to CT ranges from hours to days [8]. In our present case, signs of iatrogenic CT developed 4 h after HC



Fig. 1. a Chest X-rays. A. The course of the central venous catheter commencing at the right jugular insertion site. The widening silhouette in the cardiac area. b Representative computed tomography images showing CT after HC insertion.



Fig. 2. A chest X-ray taken after repair of the atrial wall.

insertion. Rapid diagnosis and treatment are essential. Other patients developed symptoms between 8 h and 15 days after HC insertion [3,7,10,13,18–22].

Catheter insertion aided by fluoroscopy and USG has improved child safety; these guidance modalities are now the recognized standard of care. We insert HCs employing USG guidance in patients under general anesthesia.

CP caused by CT may be affected by the time of catheter insertion, movement of the catheter tip, angle of insertion, movement of the cardiac chambers, and direct trauma [5]. CP associated with central catheter placement features puncture of the cardiac wall, superior vena cava, or inferior vena cava by the guidewire, dilator, or catheter per se [2,6,11,14,20]. In our present case, the wound was located in the right atrium, as in many other cases. All venous insertions feature a risk of CP. J-tipped guidewires are safe and rarely cause complications such as CP [5]. The J-tip is blunt; no sharp guidewire tip can contact or perforate any structure during travel [5]. Usually, CT is associated with the placement procedure per se¹¹ and may develop immediately after catheter insertion or after several days depending on the rate of fluid accumulation [3]. In our present case, the diameter of the right atrial perforation was approximately 5 mm. We did not force the dilator; we therefore suspect that the guidewire caused the perforation. Late CT complications associated with accumulation of hyperosmolar fluid include osmotic injury, shallow breathing, catheter displacement to the myocardial wall, erosion, thrombosis causing transmural necrosis, and local inflammation [3,7,8,13,20]. Polyethylene catheters are more rigid than silicone catheters and thus more likely to cause CP. Serious/fatal complications may occur despite careful placement and follow-up [15].

A small amount of fluid is present in the pericardial sac (between the smooth-surfaced viscera and parietal pericardial layer) to facilitate heart lubrication [16]. If that fluid accumulates (either slowly or rapidly), CT may follow. Any pressure increase in the pericardium and/or thoracic cavity may affect the cardiac chambers [16]. As the pressure within the pericardium increases, the central vascular pressure increase because of the resistance imparted by the pressure associated with right atrial filling. Thus, blood returning from the veins fills the right heart chambers with difficulty, triggering cardiogenic shock and death [12,16].

CP symptoms and signs are nonspecific and various. CP onset may be sudden or progressive, and CT may develop at various times after catheter insertion [17]. The symptoms include nausea, dyspnea, chest pain, tachypnea, hemodynamic instability, and confusion [16,20]. The objective findings include hypotension, tachycardia, soft heart sounds that vary in intensity with respiration, venous engorgement, pulsus paradoxus, and shock [20]. The presentation of Beck and classic triad-related acute CT features hypotension, increased vascular pressure, and muffled heart sounds [12,16]. The present and other patients lacking symptoms or dyspnea suddenly entered crises.

CP is survivable if detected early and treated promptly [4]. Electrocardiography (both direct and Doppler) facilitates rapid CP diagnosis, assessment of the presence, volume, and distribution of pericardial fluid, and evaluation of intrapericardial tissue [12,16]. However, diagnosis is not always obtained by electrocardiography. Chest X-ray has been used to locate the catheter and pleural effusion, but it may not reveal any abnormality until considerable fluid has accumulated in the pericardial sac [12]. Computed to-mography and magnetic resonance imaging can detect the presence of pericardial fluid and may afford some insight into the causative pathology.

Treatment is influenced by multiple factors such as the lesion size, hemodynamic effect, and etiology [16]. A small traumatic ventricular septum may close spontaneously, but an atrial defect may not [6]. Because our patient had an atrial defect, we scheduled surgery. CP should be surgically treated immediately after diagnosis, as cardiac lesions are fatal [18].

CT temporarily protects against massive volume loss, affording a window for diagnosis and surgical intervention [18]. Once a diagnosis has been made, prompt treatment is required. The catheter should be removed. Pericardiocentesis and drain placement may

How many case Reference	Site of perforation	Initial route	Liquid (cc)	Clinical signs	Time since catheterization	Course	Treatment	Age	Gender	Cause
3 cases [11]	-	RSV	30	Non	8 h	Died	Unknown	14-day old	Girl	Tip
	RA	RFV	-	Dyspnea	24 h	Died		Premature	Girl	Tip
	RA	LSV	95	Sweating	7 day	Died		3-year old	Girl	Tip
2Cases [22]	RA	RSV	Unknown	Sudden	2, 4 day	Death	Pericadrdiocentesis	Neonate	Girl	Tip
1 case [21]	RA					Survived		2-day-old		Tip
5 cases [7]		PICC		Sudden	1,2,6,7 day	Survived	Percutaneous Aspiration	24,24,30,33	Girl	Tip
2 cases [20]	Unknown	FV	Unknown	Shock	12 h	Survived	Aspiration	4-year-old	Unknown	Tip
	Unknown	Unknown	Unknown	Sudden	3 day	Death	Mediastnomy	12-day-old		
2 cases [3]	VCI	RIJV	Large	Sudden	5 day	Death	-	25-day-old	Boy	Tip
	RA	RSV	TPN		5 day	Death	-	4-year-old	Boy	
1 case [10]	RV	RIJV	large	Apnea	12 h	Death	Mediastnomy and aspiration	12-year-old	Girl	Tip
1 case [13]	Unknown	R SaV	23 ml	Tachypnea	12 day	Survived	Perikardiosentesis	Premature	Girl	Tip
3 cases [15]	RV	PICC	8 ml	Apnea	15 day	Death	-CVC removed	Premature	Boy	Tip
			20 ml	-	5 day	Survived	-		boy	
11cases ¹⁸	Unknown					Death	_	Infant	Unknown	Tip
Our case	RA	RIJV	600 ml	Dyspnea	4 h	Survived	Mediastinomy cardioraphy	7 year-old	Girl	Guide- wire

 Table 1

 Clinical characteristics of the present case and literature cases featuring cardiac perforations in children.

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RA: Right atrium, RV; Right Ventricule, VCI: Vena Cava Inferior, RSV; Right Subclvian Ven, RFV; Right Femoreal Ven, LSV; Left Subclavian Ven, RIJV; Right Interneal Jugular Ven, RSaV; Right sapheneous Ven, PICC; Peripherally inserted central catheter.

be performed in an emergency [4,12]. After catheter withdrawal, failure to improve necessitates pericardiocentesis followed by mediastinotomy or thoracotomy, pericardiotomy, and a cardiac procedure [8,19]. In our case, we did not know the side of perforation, and the condition of our patient worsened after accumulation of intra-abdominal, intrathoracic, and intrapericardial fluid. She therefore underwent mediastinotomy to identify the site of trauma.

In conclusion, CP associated with HC placement is rare, serious, and associated with high mortality, and it can occur any time after insertion. Strategies should be formulated to avoid the risk of CP. If CP is suspected, close follow-up and early treatment are essential. The guidewire should not be forced, and fluoroscopic guidance should be employed in cases of doubt.

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