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Successful retrieval of a knotted Swan--Ganz catheter using interventional approach in an adult: a case report

Xiao-ke Shang[1], Yan Yan[2], Bin Wang[1]

[1]*Union Hospital, Tongji Medical College of Huazhong University of Science and Technology, Wuhan, Hubei, China*
[2]*SinoScript LLC., Vernon, USA*

Sir,

Swan-Ganz catheterization, also known as the balloon tipped pulmonary artery catheterization, is widely used as a diagnostic and hemodynamic tool to monitor patients in the intensive care unit [1]. Knot formation is one of the rare complications with the incidence of 0.2–2.5%, which in general resulting in entrapping the catheter in the superior vena cava [2]. Surgical intervention is necessary. In recent years, due to the advances in radiology, interventional approaches are utilized to non-surgically extract the catheter [3]. We describe a case in which we succeeded removing the entrapped catheter using non-invasive procedure.

A 58-year-old male was admitted to our center with a 36-month chest pain and 12-month exacerbation. Using ultrasonic cardiogram (UCG), he was diagnosed congenital septal defect, severe aortic regurgitation and cardiac function capacity II. The patient underwent aortic valve replacement and temporary pacemaker insertion. To monitor hemodynamic measurements, a Swan-Ganz catheter was passed into the pulmonary artery through the jugular vein on the following day in the intensive care unit. After obtaining hemodynamic indexes, attempts to withdraw the catheter were unsuccessful. A chest X-ray was performed to identify an obvious knotting at the rostral end (Fig. 1), which entrapped the catheter. X-ray also revealed the annuloplasty ring and the temporary pacemaker (Fig. 2). The diameter of the knot was 6 mm, and it was possible that the catheter was coiled to form a knot like structure. We designed three strategies to remove the knotted Swan-Ganz catheter: 1. Utilizing a snare to grasp the twisted end, which stabilizes the external portion, so we could turn the snare clockwise or counter clockwise to straighten the catheter and remove it from the vein. 2. A delivery sheath (congenital heart disease interventional delivery sheath) to capture the knot, cut off the catheter at other end and use the delivery sheath to retract. 3. Surgical extraction. We started with the option 1. In the hybrid operation room, under fluoroscopy the angiographic catheter (Cordis Coporation, 0.038", 100 cm, MPA1) was inserted into superior vena cava along the guide wire (Medtronic, 0.035', 145 cm) (Fig. 3). After retracting the guide wire, the snare was delivered through end-hole catheter to capture the Swan-Ganz catheter knotted end till atrium. The

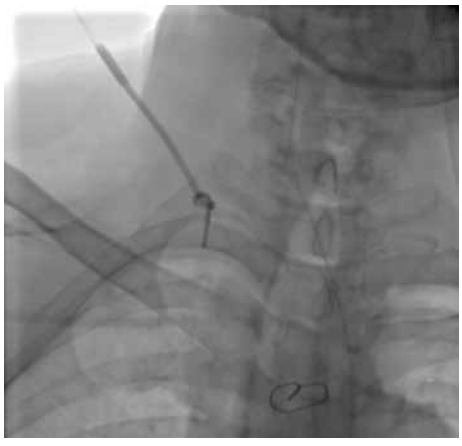

Figure 1. X-ray demonstrating the knotted Swan-Ganz catheter

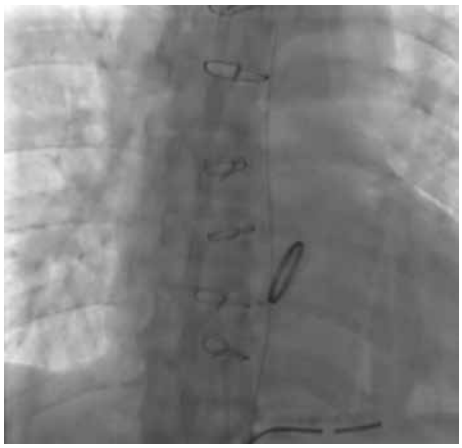

Figure 2. The annuloplasty ring and the temporary pacemaker in position

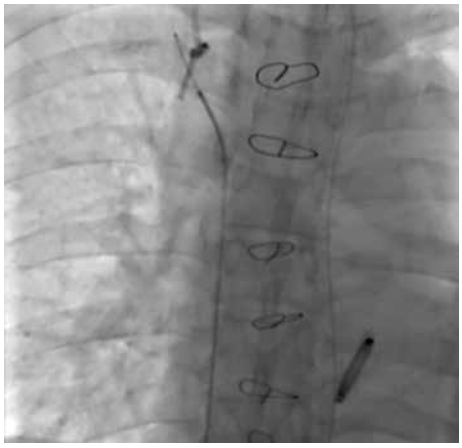

Figure 3. The angiographic catheter and the guide wire in the superior vena cava, in touch with the knotted catheter

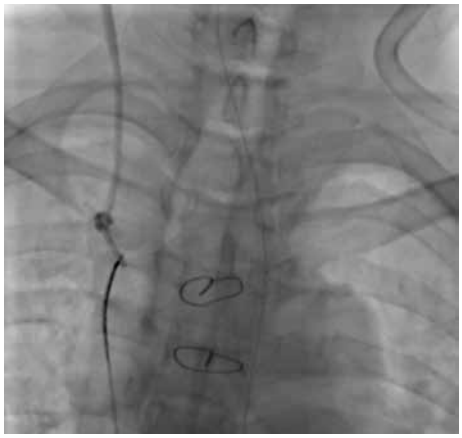

Figure 4. The snare cannot pull the catheter straight

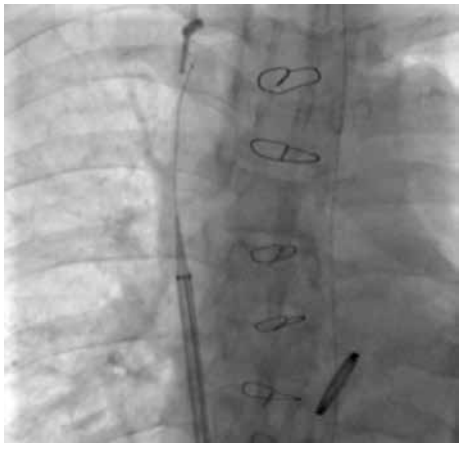

Figure 5. 14F Septal occluder into the superior vena cava

catheter was unable to be straightened after turning clockwise or counter clockwise, confirming that it was knotting instead of coiling (Fig. 4). We turned to option 2 by retracting the snare to replace with another guide wire (Terumo radifocus, 0.035", 260 cm), which guided the septal occluder delivery sheath (Shanghai Shape Memory Alloy, ODS-A-14F) to reach the superior vena cava (Fig. 5). The knotted Swan-Ganz catheter was re-captured by the end-hole catheter and the snare (Fig. 6). The attempts to bring the catheter into the delivery sheath did not succeed, because of the angle between the catheter and the sheath entrance. Another re-capture successfully guided the knotted catheter into the delivery sheath (Fig. 7), with 27 N forces applied (Fig. 8). The distal end was cut off from the jugular vein end (Fig. 9), and hemostasis was achieved by mechanical compression. The knotted catheter was visualized after extraction (Fig. 10).

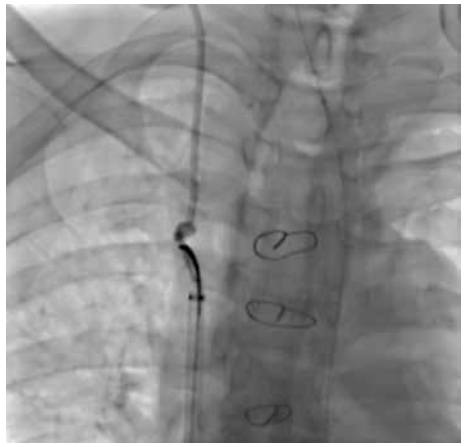

Figure 6. The snare captures the Swan-Ganz catheter

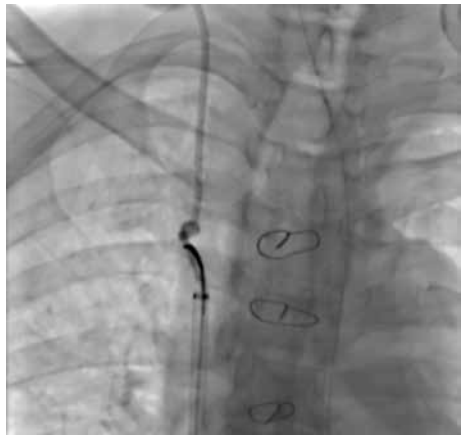

Figure 7. The knotted Swan-Ganz catheter inside the delivery sheath

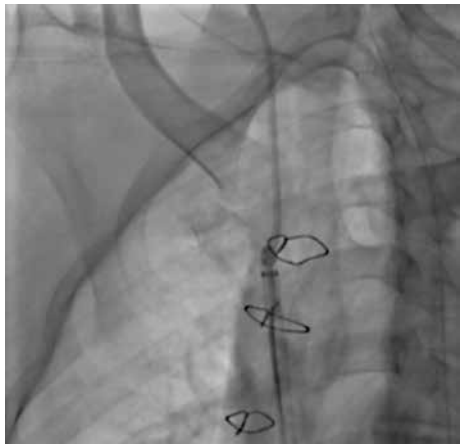

Figure 8. The knotted Swan-Ganz catheter inside the delivery sheath

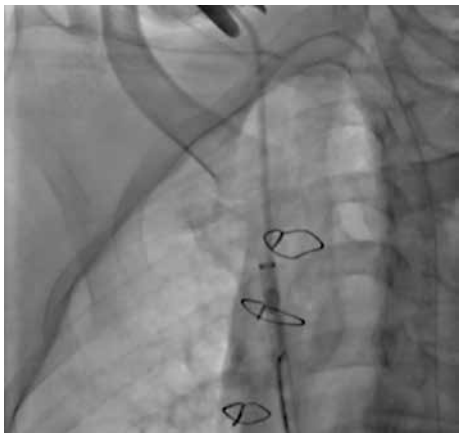

Figure 9. A cut at the jugular vein end

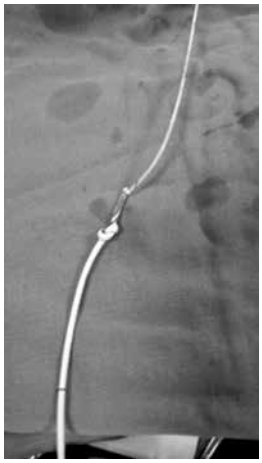

Figure 10. The knot of the Swan-Ganz catheter with the snare after extraction

First developed by Dr. Jeremy Swan and Dr. William Ganz in 1970, Swan-Ganz catheter is an essential diagnostic and hemodynamic monitoring method. Nowadays Swan-Ganz catheter is commonly used for critically unstable patients in intensive care units perioperatively [4, 5]. Although the causal connection between utilization of Swan-Ganz catheterization and improvement of clinical outcomes is not fully established, Swan-Ganz is one of the leading medical devices to provide hemodynamic and cardiac output evaluations [6, 7]. Well-documented complications include arrhythmias, infection, thrombophlebitis, thrombosis, cardiac perforation and pulmonary artery rupture [8]. The lattermost is the most severe complication due to catheterization, with an estimated incidence of 0.03–0.2%, mortality of 70% [9, 10]. Catheter knotting is a rare but known complication [11, 12], here we present a case involving a knotted an entrapped Swan-Ganz catheter that was successfully removed by a snare using interventional approaches.

Previously, knotting formation of the catheter was reported in 2013 [11]. In that case, a 59-year-old patient with myocardial infarction was implanted with a Swan-Ganz catheter (110 cm 7 French 4-luman like Baxter). The Swan--Ganz catheter reached the pulmonary artery correctly and obtained the hemodynamic parameters. However, attempts to open the knot percutaneously using radiological approach failed, the knotted catheter was extracted with surgery. In sharp contrast, we report a case that interventional approach succeeded removing the knotted catheter. In addition, we did not introduce additional complications during the procedure, thus our successful experience provides a novel alternative to deal with catheterization complications.

Knotting of the Swan-Ganz catheter formation is unknown, we hypothesize several reasons: 1. Enlarged right atrium causes catheter curls in the blood flow. 2. Catheter reaches at the distal portion of the pulmonary artery, the catheter end loops to form a knot 3. Catheter is used repetitively and the line is softened to coil. X-ray visualized the knotting formation in our case and in the previous case [11], which indicates that the knotted catheter may occur if retraction is not smooth, thus X-ray is useful to diagnose the problem. Retracting the knotted Swan-Ganz catheter from the jugular vein is extremely dangerous, because this might cause jugular vein rupture. Our experience suggests a few points to pay attention: 1. Measure the knot size, choose the proper diameter of delivery sheath (≥ knot diameter). 2. Personalize the delivery sheath, this catheter is entrapped in the superior vena cava, thus we chose the septal defect sheath with a straight portion. 3. The snare aims at the proximal end of the catheter, otherwise it is difficult to guide the catheter into the delivery sheath. 4. A neat cut at the jugular vein side is necessary to avoid vessel damage. 5. Surgical intervention is essential if the interventional approach fails, thus the procedure performed in hybrid operation room is recommended.

To sum up, not formation of the Swan-Ganz catheter is a rare complication. We reported that interventional approaches can be utilized to remove the knotted Swan-Ganz catheter in the superior vena cava. Compared to surgical removal, interventional procedures with improved resolution result in smaller lesions and better recovery.

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2. Source of funding: none.
3. Conflict of interest: none.

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Corresponding author:
Bin Wang
Union Hospital, Tongji Medical College
of Huazhong University of Science and Technology
Wuhan, Hubei
e-mail: yxsxs2015@sohu.com

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Isolated lower limb gangrene: a caveat of terlipressin therapy

Ankur Khandelwal, Devendra Gupta, Rudrashish Haldar, Anindita Rai

Department of Anaesthesiology, Sanjay Gandhi Post Graduate Institute Of Medical Sciences (Sgpgims), Lucknow, Uttar Pradesh, India

Sir,

Terlipressin is a synthetic analogue of the natural hormone arginine–vasopressin. It is often employed for the management of bleeding esophageal varices (BEV) and hepatorenal syndrome (HRS), both of which are catastrophic complications of advanced liver disease. Being a vasoconstrictor with preferential action on the splanchnic circulation, it aids the lowering of portal venous pressure. Terlipressin usage during BEV has been shown to decrease mortality, the failure rate of initial hemostasis, as well as the number of emergency procedures to stop uncontrolled bleeding or rebleeding [1]. In spite of its relatively safer pharmacological profile as compared to vasopressin, complications attributed to systemic vasoconstrictor properties have been occasionally reported. We encountered a case of isolated lower-limb gangrene following terlipressin therapy and wish to report it after obtaining informed written consent from the relatives.

A 67-year-old male patient with a history of alcohol-related chronic liver disease (CLD) and portal hypertension (7 years duration) was admitted to our ICU following one episode of haemetemesis, deteriorating sensorium and reduced urine output (approx. 300 mL day^{-1}) during the previous 48 hours. The patient was an active smoker with a history of 45 pack-years. His daily intake of alcohol had been 150 g for 38 years. He had discontinued alcohol consumption 7 years ago following the initial identification of alcohol-related cirrhosis. On admission, the patient was afebrile, haemodynamically stable and icteric. After initial examination and investigations, patient was diagnosed provisionally as acute-on-chronic liver disease with decompensation and HRS. Since the concern of aspiration existed due to an ongoing oesophageal bleed and depressed sensorium secondary to hepatic encephalopathy, the patient was intubated and put on mechanical ventilation using the Continous Positive Airway Pressure (CPAP) mode.

Subsequently, endoscopic variceal band ligation (EVL) was attempted but the procedure failed due to a persistent haemorrhage. Therefore, terlipressin therapy was considered. The patient was administered an initial bolus of terlipressin (2 mg stat) followed by 1 mg every 4 hours. Concomitantly, a 20% albumin solution infusion was commenced at a dose of 1 g kg^{-1} day^{-1} for the 1st day followed by 20 g day^{-1} in view of HRS. Packed Red Blood Cells (PRBCs) and blood products were transfused based on existing haemoglobin levels, the coagulation profile and thromboelastography. After the initial conservative measures had stabilised the patient, EVL was again attempted on the 3rd day and was carried out successfully.

On the 4th day, however, a new onset of blackish discolouration of the skin of all the toes of the left foot, along with the distal part of the foot on both the dorsal and ventral aspects, were noticed. Similar changes, but of a lesser

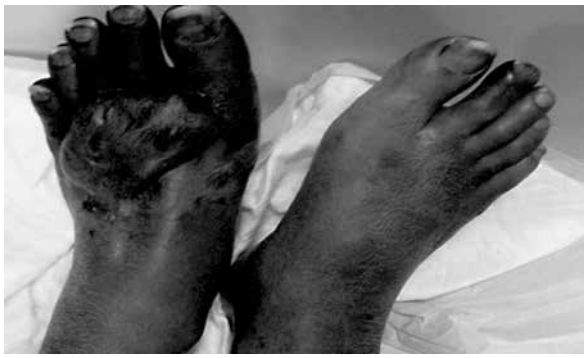

Figure 1. Blackish discolouration of skin of all the toes of the left foot, along with distal part of the same foot. Similar changes, but of lesser magnitude, may also be noticed on the great toe and 2nd toe of the right foot

magnitude were also noticed on the great toe and the 2nd toe of the right foot (Fig. 1). An urgent Doppler ultrasound was performed which demonstrated normal blood flow in the major arteries (superficial femoral, popliteal, anterior tibial, posterior tibial, peroneal and dorsalis pedis) of both lower limbs, while also confirming the patency of the venous channels. Such changes were, however, absent on other body parts. Terlipressin injections were stopped immediately and oral sildenafil (50 mg twice a day) was started on the same day. However, the gangrenous changes did not resolve until the 14th day when the patient expired due to systemic complications of ongoing severe sepsis and acute respiratory distress syndrome (ARDS).

Since its introduction in the early 1990s, terlipressin has emerged as a frontline therapy in order to manage BEV and HRS. Its advantages include its potency, prolonged half-life (6 hours), relative safety and easy administration in intravenous boluses. While terlipressin acts selectively on the splanchnic circulation, it can exert vasoconstrictor effects on the systemic circulation. Therefore, systemic sequelae ranging from mild ischaemic complications to serious complications like ischaemic colitis, myocardial infarction and skin necrosis can be attributed to terlipressin usage. The frequency of ischaemic complications after terlipressin therapy for HRS is reported to be 5% [2]. Le Moine *et al.* [3] reported the absence of ischaemic complications following high doses of terlipressin (1 mg every 4 hours) administration to a patient with HRS over 2 months. Conversely, gangrenous changes on the toes have been reported to appear on the very first day of terlipressin therapy [4]. Ischaemic events, therefore, are probably independent of the duration of terlipressin therapy. This necessitates the recognition of certain risk factors like hypovolemia, the concomitant administration of pressor drugs and the mode of terlipressin administration [5]. Generally, continuous intravenous infusion of terlipressin is not recommended as the mode of administration, since it causes cutaneous (at the infusion site) and scrotal necrosis [6]. The ischaemic complications secondary to terlipressin therapy are probably related to the particular distribution of the target receptor of terlipressin — the vasopressin receptor type 1 (V_1 receptor) — which is located in the smooth muscles of the blood vessels, mainly in the territory of the splanchnic circulation, kidney, myometrium, bladder, adipocytes and skin circulation [7]. However, preferential involvement of a particular site has not yet been fully explained.

In our case, terlipressin was administered as an intravenous bolus while the risk factors involved were chronic alcoholism and smoking. The peripheral vasoconstrictive changes secondary to prolonged smoking may have exaggerated and accelerated the development of limb gangrene.

Terlipressin should be stopped immediately once the ischaemic events are suspected. Ischaemic changes in both the lower limbs have been reported to have regressed and recovered in 2 weeks after the discontinuation of terlipressin [8]. In contrast, Coskun *et al.* [1] reported that skin necrosis on the forearm progressed for 1 week even after terlipressin discontinuation. Thus, cessation of terlipressin does not always necessarily result in the regression of gangrenous changes.

Various vasodilators have been tried as rescue therapy with variable success rates. These include alprostadil (PGE1 analogue) [9], sildenafil [10] and nitrates [11] as reported by various authors. Nevertheless, amputation remains the last resort in limb gangrene. In our patient, terlipressin-induced ischemia led to necrosis and gangrene of both feet. Despite the timely cessation of terlipressin and the initiation of vasodilator therapy, gangrene did not subside in our patient until his death on the 14th day.

This case suggests that despite its rarity, the possibility of ischaemic complications caused by terlipressin, must be borne in mind by clinicians. Recognising the risk factors, the immediate cessation of terlipressin and concomitant initiation of vasodilators can be helpful, albeit not always successful forms of treatment.

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Corresponding author:
Ankur Khandelwal
Department of Anaesthesiology
Sanjay Gandhi Post Graduate Institute Of Medical Sciences (Sgpgims), Lucknow, Uttar Pradesh, India
e-mail: ankurchintus@gmail.com