



Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Post traumatic inferior vena cava thrombosis: A case report and review of literature



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ARTICLE INFO

Article history:

Received 13 January 2017

Received in revised form 3 May 2017

Accepted 6 May 2017

Available online 15 May 2017

Keywords:

Inferior vena cava

Post traumatic

Thrombosis

Case report

ABSTRACT

INTRODUCTION: Post traumatic inferior vena cava (IVC) thrombosis is a rare and not well described entity with nonspecific clinical presentation. It remains a therapeutic challenge in traumatic context because of haemorrhagic risk due to anticoagulation.

PRESENTATION OF CASE: We report a case of IVC thrombosis in an 18 year-old man who presented with liver injury following a traffic crash. The thrombosis was incidentally diagnosed on admission by computed tomography. The patient was managed conservatively without anticoagulation initially considering the increasing haemorrhagic risk. IVC filter placing was not possible because of the unusual localization of the thrombus. Unfractionated heparin was started on the third day after CT scan control showing stability of hepatic lesions with occurrence of a pulmonary embolism. The final outcome was good.

DISCUSSION: The management of post traumatic IVC thrombosis is not well described. Medical approach consists in conservative management with anticoagulation which requires the absence of active bleeding lesions. Surgical treatment is commonly based on thrombectomy under extracorporeal circulation. Interventional vascular techniques have become an important alternative approach for the treatment of many vessel lesions. Their main advantages are the relative ease and speed with which they can be performed.

CONCLUSION: Post traumatic IVC thrombosis is a rare condition. Its management is not well defined. Early anticoagulation should be discussed on a case-by-case basis. Other alternatives such IVC filter or surgical thrombectomy may be used when the bleeding risk is increased. The most serious risk is pulmonary embolism. Outcome can be favorable even with non surgical approaches.

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1. Introduction

The present case is compliant with the SCARE guidelines [1].

Post traumatic venous lesions are dominated by injuries and aneurysms. Another entity, not well described through the medical literature, is post traumatic venous thrombosis which can affect various locations such as jugular vena, renal vena or inferior vena cava. Only few cases of post traumatic inferior vena cava (IVC) thrombosis have been reported so far in the literature [2], which suggest diagnosis and management difficulties. The major risk of this condition is pulmonary embolism [3]. We report a case of post traumatic IVC thrombosis following closed hepatic injury,

diagnosed accidentally on admission, rapidly complicated by pulmonary embolism and managed only by anticoagulation. IVC filter placing was not possible because of unusual thrombus localization. This case of post traumatic IVC thrombosis was managed in a Tunisian academic hospital.

2. Presentation of case

An 18-year-old healthy man was admitted to the ICU of Sahloul teaching hospital, 13 h after a traffic crash. His BMI was about 28 kg/m². The vital signs were as follows: Glasgow Coma Scale was 15, arterial blood pressure was 130/80 mmHg, heart rate was 94 bpm and oxygen saturation was 100%. The patient complained about diffuse abdominal pain with sensibility of the right hypochondrium. The findings of initial blood tests showed: hemoglobin level 9,6 g/dl, WBC count 13,600/mm³, platelet count 247,000/mm³, prothrombin time 69%, activated partial thromboplastin time 37"/32", alanine aminotransferase 450 IU/L, total

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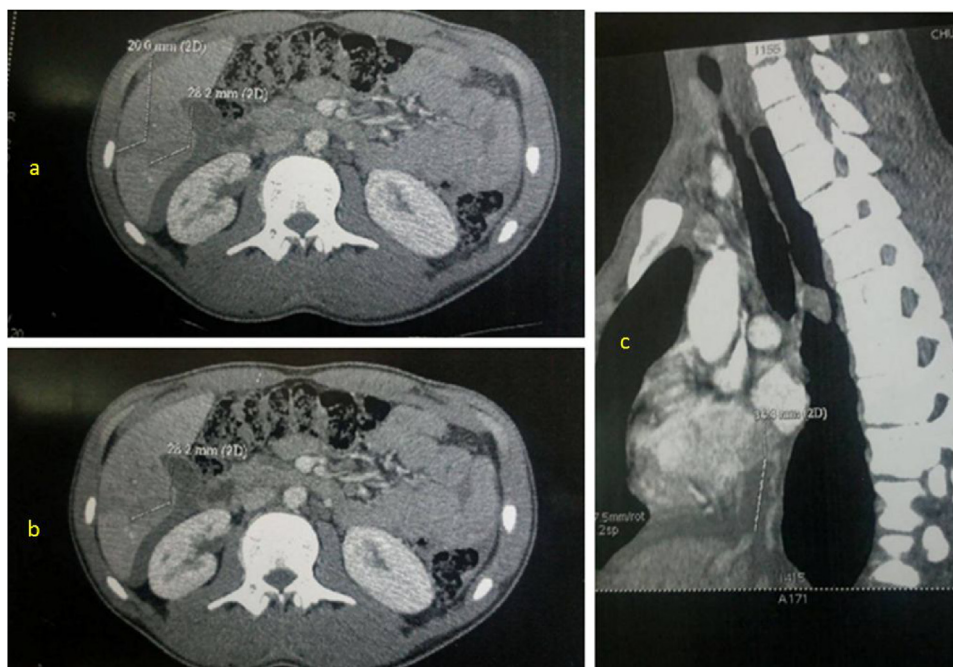


Fig. 1. Thoraco-abdominal CT findings (a and b: hepatic contusion and peritoneal effusion; c: IVC thrombosis).

bilirubin 20 $\mu\text{mol/L}$ and conjugated bilirubin 4 $\mu\text{mol/L}$. The body spiral computed tomography (CT) revealed hepatic contusion of the 6th segment with a moderate peritoneal effusion, a 35 mm endoluminal thrombosis of the thoracic IVC and a thrombosis of the middle hepatic vein (Fig. 1). The CT acquisition protocol consisted in arterial time (at 30 s) and portal time (at 70 s) after injection of 2 ml/kg of iodixanol (320 mg/ml) with 0.6 mm slice thickness.

The patient had no history of hypercoagulopathy or nephrotic syndrome. Thrombophilia tests returned without abnormalities.

The patient was managed conservatively in the intensive care unit (ICU) because he was hemodynamically stable without anticoagulation for fear inducing bleeding within hepatic contusions. By day 3, there were neither changes in the vital signs, nor decrease in hemoglobin and hematocrit levels, and then we decided to check hepatic injuries stability and thrombosis extension to start anticoagulation. The thoraco-abdominal CT scan showed a regression of the peritoneal effusion and the endoluminal IVC thrombosis. A pulmonary embolism in the right inferior lobar artery and a complete thrombosis of the middle hepatic vein were diagnosed. Unfractionated heparin, which is the mainstay of treatment for patients with IVC thrombosis was then started targeting an activated partial thromplastin time twice the normal value. The next day, we added the acenocoumarol. The outcome was favorable allowing the transfer to the visceral surgery ward on day 13 and the discharge from hospital on day 28. A CT scan control at month 3 after the trauma showed a total resolution of thrombus. The used protocol for the CT scan control was the same as that used for the initial diagnosis. No residual luminal stenosis of the IVC was found. Anticoagulation with antivitamin k was continued for 6 months.

3. Discussion

Post traumatic IVC thrombosis is an extremely rare condition. Only few cases have been reported so far in the literature [2–19] (Table 1). It has been attributed to transmurular laceration of the vena cava secondary to crushing forces, with formation of a pericaval or a retroperitoneal hematoma compressing and narrowing the vena cava to such an extent that venous stasis develops. This stasis

leads to distal thrombophlebitis and then antegrade caval thrombosis. In cases where a retroperitoneal or a pericaval hematoma is not obvious, different mechanisms of traumatic thrombosis are involved. Endothelial injury of the venous wall, secondary to compression or shear forces, leads directly to caval mural thrombosis resulting in occlusion [17]. Hepatic parenchymal injury may cause hepatic vein thrombosis that eventually extends into the IVC. Hypercoagulability associated with suppression of fibrinolysis is a normal physiologic response after trauma [17]. These different mechanisms reported in the literature and explaining the pathophysiology of post traumatic IVC thrombosis are summarized in Table 2. In our case, IVC thrombosis is likely to be caused by direct endothelial injury of the caval wall given the thrombus rapid formation and size.

The delay of diagnosis varies through the literature from 3 days to 4 years (Table 2). Besides our case, only two others were diagnosed on the first day of admission. The first one, published in 2005, was diagnosed 4 h after admission [14] and the second one, published in 2016, was diagnosed by the initial body CT scan [19]. In this regard, we have to emphasize that the imaging progress, the availability of the Body CT scan and its broadening indications in the management of polytrauma, should reduce the diagnosis delay. However, a normal initial CT scan does not exclude the possibility of a thrombus formation later. In fact, IVC thrombosis is usually found 2–7 weeks after injury in the reported cases [3].

The clinical manifestations of post traumatic IVC thrombosis are unclear and involve nonspecific signs as abdominal or back pain. It can also involve ascites, bilateral leg edema, phlebitis of lower limb, pulmonary embolism and Budd-Chiari syndrome (Table 2).

The therapeutic strategy is not well described in the literature. Once the diagnosis is confirmed, immediate treatment must be started to avoid clot migration and chronic complications. It is based on either medical or medico-surgical approach. Medical approach consists in conservative management in ICU with anticoagulation which requires the absence of active bleeding lesions. Surgical treatment is commonly based on thrombectomy under extracorporeal circulation (ECC). The indication of a veno-venous or arterio-venous ECC depends on the level of the IVC throm-

Table 1
Description of the cases of post traumatic IVC thrombosis over the literature.

Authors	Age/Sex	Revealing signs	Delay	Vascular lesion	Associated lesions	Contributory factors	Treatment	Outcome
Little [4]	57/M	Abdominal pain, leg edema	2 months	Diaphragmatic constriction	Hepatic vena	None	Antibiotic	Death
Grmoljez [5]	57/M	Phlebitis of lower limb	6 weeks	Transmural IVC laceration. Hematoma	Lower limb thrombosis	None	CPB, thrombectomy, AC	–
Campbell [6]	21/M	Abdominal pain, fever	19 days	Endothelial lesion	Hepatic laceration	None	AC, Bed rest	–
Nagy [7]	55/M	Abdominal and back pain	18 days	Endothelial lesion	None	VTE history	IVC filter, Anticoagulation	–
Mayzlik [8]	49/M	Nephrotic syndrome	2 months	–	Thrombosis of renal vena and lower limb	None	Thrombectomy	–
Nau [9]	30/M	PE	6 weeks	Endothelial lesion	Thrombosis from renal vena to IVC	None	AC then fibrinolysis	Death
Takeuchi [10]	21/M	Abdominal pain, PE	14 days	–	Retroperitoneal hematoma	None	–	Death
Kimoto [11]	35/M	None	35 days	–	Hepatic laceration	Hyper coagulability	Thrombectomy	–
Balian [12]	31/M	Asthenia	3 years	Intravascular membrane	–	Myeloproliferative neoplasm	AC, percutaneous angioplasty then surgery after recurrence	obstruction recurrence
	17/M	Abdominal pain. Budd-Chiari syndrome	3 days	Intravascular membrane	Renal and hepatic vena	Plasminogen congenital deficiency	Thrombectomy, membranous resection, AC	obstruction recurrence
Cellarier [13]	53/M	PE	3 months	Endothelial lesion	None	None	Thrombectomy, AC	–
Fujii [3]	40/F	Incidental finding	30 days	–	Intrahepatic hematoma	None	CPB, atriotomy and thrombectomy, AC and antiplatelet therapy	–
Mouaffak [2]	19/M	Incidental finding	3 days	Left renal vena partial thrombosis	Hepatic and renal contusion, splenic hematoma	None	AC	–
Castelli [14]	65/F	Hemorrhagic shock	4 h	IVC leaking injury	Retroperitoneal hematoma	None	Stent-grafting	Death
Ushijima [15]	22/M	Incidental finding	4 years	Intravascular membrane	Pancreatic laceration	None	CPB. Right atriotomy, thrombectomy, AC	–
Hamamoto [16]	32/M	leg edema, abnormal LFT, Budd-Chiari syndrome	1 month	–	Hepatic laceration extended to the middle hepatic vena	None	AC, Thrombectomy under CPB	–
Kim [17]	26/M	Incidental finding	15 days	–	Hepatic laceration, parenchymal hematoma	None	IVC filter, AC	–
Sabzi [18]	30/M	back pain, leg and scrotal edema, dyspnea, fever	10 days	–	–	None	CPB: atriotomy, pulmonary embolectomy, AC	–
Salloum [19]	33/M	Incidental finding	admission	Retrohepatic IVC thrombosis	Left liver fracture, left hepatic artery lesion	None	CPB: thrombectomy. Left liver lobectomy	–

IVC: inferior vena cava; CPB: cardio-pulmonary bypass; AC: anticoagulation; VTE: venous thrombo-embolism; PE: pulmonary embolism; LFT: liver function tests.

Table 2
Pathophysiological mechanisms of post traumatic IVC thrombosis (5).

Mechanisms
Endothelial injury of the caval wall with secondary thrombus formation
Caval stasis secondary to compression by a pericaval or retroperitoneal hematoma
Hepatic vein thrombosis after liver laceration extending into the IVC
Hypercoagulable state after major trauma

bus. Venovenous ECC is indicated when the thrombus is located retro-hepatically, requiring caval reconstruction with prolonged infra-diaphragmatic caval clamping. When the thrombus extends to the right atrium, arterio-venous ECC, with or without cardiac arrest and deep hypothermia is necessary [19]. Besides, interventional vascular techniques have become an important alternative approach for the treatment of many vessel lesions. Ten years ago, Castelli et al. successfully used this technique for a patient who underwent endovascular stent-grafting for traumatic injury of the inferior vena cava. The main advantages of this endovascular approach include the relative ease and speed with which it can be performed [14]. It is particularly attractive in terms of bleeding control following injuries at the level of the suprarenal vena cava or at the level of the ilio-caval bifurcation, where hemostasis requires extensive dissection. However, there are several concerns with stent-graft repair of IVC traumatic injury such as the postoperative anticoagulation strategy and the long term outcome [14]. Other endovascular treatment modalities exist for patients with IVC thrombosis such as catheter-directed thrombolysis, AngioJet rheolytic thrombectomy, the Trellis peripheral infusion system and the angiovac for aspiration thrombectomy [20].

IVC filters can be used to prevent pulmonary embolism when anticoagulation is risky. Most operators opt to place this device in the infrarenal or suprarenal segments of IVC only in high-risk patients especially those with large floating thrombus or diminished lung reserve [20].

In our case, the patient was hemodynamically stable after the clot migration. In certain cases, massive pulmonary embolism causes hemodynamic instability with right ventricular failure and circulatory collapse. According to the 2016 American College of Chest Physicians (ACCP) Antithrombotic Guidelines, therapy for massive pulmonary embolism should include systemic thrombolytic therapy in association with anticoagulation and supportive care. Patients with contraindication to systemic thrombolytics, extracorporeal membrane oxygenation (ECMO) and/or surgical embolectomy may be used to improve oxygenation, achieve hemodynamic stability and successfully treat massive pulmonary embolism. The most common complication from ECMO and pulmonary embolectomy with cardiopulmonary bypass is bleeding due to systemic anticoagulation, thrombocytopenia and platelet dysfunction [21].

In our case, we did not opt for surgical approach because of the good clinical tolerance and the risk of anticoagulation during the ECC. The endovascular approach was not considered because of many thrombi locations. The IVC filter wasn't possible because of the unusual thrombus localization in the intra thoracic portion. The patient was managed conservatively in the ICU. Anticoagulation was started on the third day for fear of bleeding risk initially from hepatic laceration. The final outcome was good like the majority of published cases. In fact, we deplored only 4 deaths in the literature, caused by pulmonary embolism in 2 cases (Table 2). If our patient was hemodynamically unstable, the most reasonable solution would be surgical embolectomy under general anesthesia and cardiopulmonary bypass while keeping in mind the high risk of hepatic bleeding.

Because of a very early diagnosis and treatment of the IVC thrombosis, there was no chronic fibrotic transformation of the clot.

Thereby, total thrombus resolution was found in the 3 months post traumatic CT scan.

4. Conclusion

Post traumatic IVC thrombosis is a rare condition and its therapeutic approach is still not well defined. Early anticoagulation is the mainstay of treatment. When it is at high risk of bleeding, other alternatives may be used such IVC filter or surgical thrombectomy. The most serious risk is pulmonary embolism which can be fatal. Outcome can be favorable even with non surgical approaches.

Conflict of interest

The authors declare that they have no conflict of interest.

Funding

This study was not funded by any organization or institution.

Ethical approval

This manuscript was approved by Ethics Committee of Sahloul teaching Hospital.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Authors contribution

Study concept or design: Mohamed Kahloul, Mohamed Said Nakhli

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Data interpretation: Walid Naija, Mohamed Kahloul

Literature review: Mohamed Said Nakhli, Amine Chakroun

Drafting of the paper: Amine Chakroun, Mohamed Kahloul

Editing of the paper: Mohamed Said Nakhli, Walid Naija

Guarantor

Walid Naija is the guarantor.

Acknowledgement

None.

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