Pictorial CME

Inferior Vena Cava Syndrome in Hepatic Trauma and Pyogenic Liver Abscess

Ashwani K Dalal[1], Usha Rani Dalal[2], Hari Kishan Rathi[3], Ravinder Kaur[4]

Vena cava syndrome presents with features of central venous obstruction due to occlusion of superior or inferior vena cava. Clinically, SVC syndrome is well described whereas there are only few reports on IVC syndrome. IVC occlusion may occur either by direct physical invasion or compression by various pathological processes of the organs in its vicinity and primary congenital or acquired lesions of IVC. Ignorance about this entity due to rarity and overlapping of its clinical features with the clinical course of the underlying aetiology causes significant diagnostic and management delays. Early recognition and prompt management is crucial for a favourable outcome. CECT abdomen is an important tool for timely diagnosis. Only few cases of IVC syndrome due to IVC thrombosis (IVCT) in liver trauma and IVC compression by a pyogenic liver abscess have been reported in literature. We hereby report two cases of IVC syndrome where the clinical course of hepatic trauma and pyogenic liver abscess was complicated by the IVC occlusion due to IVCT and external compression and managed successfully with anticoagulant therapy and by the drainage of the liver abscess respectively.

[*J Indian Med Assoc* 2021; **119(5):** 54-7]

Key words **: Inferior vena cava Syndrome, Hepatic Trauma, Pyogenic Liver Abscess.**

Superior and inferior vena cava syndrome is a rare group of symptoms produced by central venous obstruction. Superior vena cava syndrome is characterised by facial plethora, jugular venous distension and arm swelling. The manifestations of IVC syndrome are not well described resulting in poor recognition and hence under-reporting of this entity. Early recognition and management of IVC occlusion remains a challenge for clinicians. Occlusion due to IVC thrombosis (IVCT) is an uncommon complication of trauma[1-4]. In general the Virchow's triad of "stasis, vessel injury and hypercoagulability" remain the main underlying pathophysiology in the development of IVCT. Other causes are external compression due to lymphadenopathy, hypertrophy and regenerating nodule, trauma, abscess, hydatid disease and malignancy of liver and adjacent structures, aortic aneurysm, pregnant uterus, raised intra-abdominal pressure, pneumo-peritoneum during laparoscopic procedures[5-9]. Internal luminal obstruction may occur due to congenital anomaly, primary tumours of vena cava, direct tumour extension of renal or uterine tumour in its lumen, hydatid cyst and emboli from DVT in of lower limbs[10-12]. Clinical manifestations depend on the speed and degree of occlusion, status of collateral veins, stasis in the venous flow. IVC occlusion usually complicates the clinical course of the underlying primary aetiology and the diagnosis is missed initially due to its diverse non-specific presentation and ignorance about this syndrome. Sonography, color-doppler, CECT and MR imaging studies are important diagnostic tools. Treatment of IVCT is individualised according to the cause and severity of the occlusion; anticoagulants alone or a multimodality management by combination of anticoagulants and intervention or surgical procedures. Management of external compression by underlying primary pathology include (1) Excision or surgical debulking of the tumour alone or in combination with chemotherapy or radiation (2) drainage of abscess (3) extraction of tumour emboli. We report two cases of IVC syndrome where IVC occlusion complicated the clinical course of hepatic trauma 3 weeks after the injury and pyogenic liver abscess due to thrombosis and external compression respectively. CECT abdomen revealed the thrombosis of vena cava due to trauma and compression of vena cava by liver abscess. Obstruction was relieved by anticoagulants in thrombotic occlusion and by surgical drainage in case of liver abscess.

Editor's Comment :

- Clinicians should be watchful when faced with complicated liver abscesses, or liver trauma especially with uncommon presentations like IVC syndrome resulting in swelling of both lower limbs, ascites and scrotal oedema.
- High index of suspicion, repeated radiological examinations, prompt early aggressive management with timely surgical intervention, antibiotics and drainage results in life threatening complications of IVCT due to trauma or liver abscess.

Department of Surgery, Government Medical College, Chandigarh 160030

[1]MBBS, MS, MRCS (Glasgow), Professor

[2]MBBS, MS, Professor and Corresponding Author

[3]MBBS MS, Ex Senior Resident

[4]MBBS, MD, Professor and Head, Department of Radiodiagnosis, Government Medical College, Chandigarh 160030

Received on : 15/02/2021

Accepted on : 22/02/2021

Case Report 1 (Case of IVC Thrombosis Following BTA) :

A 28-year-old male patient presented to surgery emergency with blunt trauma abdomen sustained when his motorcycle was hit by a car. On arrival to surgery emergency, his GCS was 15/15, blood pressure was 128/88 mm of Hg, pulse rate was 92 / min and respiratory rate was 22/min. CECT abdomen showed laceration of the caudate of liver, perihepatic haematoma and minimal haemoperitoneum (Fig 1 CECT abdomen showing injury of the caudate lobe of liver). Patient was treated by non-operative management protocols by I/V fluids along with parentral antibiotics and analgesics. He was discharged in satisfactory condition after 7 days.

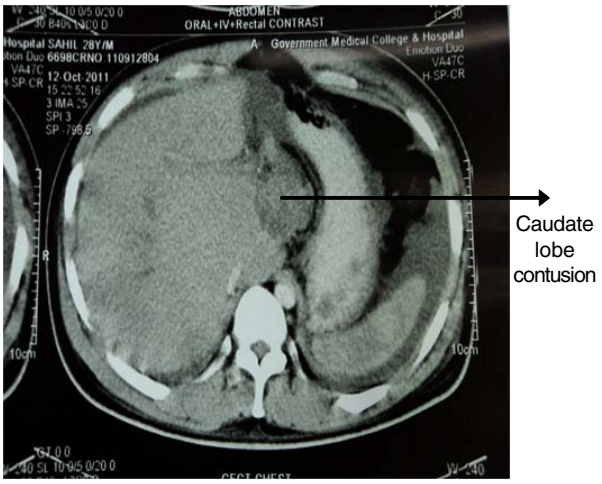


Fig 1 — CECT abdomen showing injury of the caudate lobe of liver (arrow)

After a period of 2 weeks patient again reported in surgery emergency ward with complaint of back ache and abdominal pain and distension. There was no history of vomiting and constipation. His blood pressure was 132/90 mm of Hg, pulse 98/ min. On general physical examination bilateral pedal oedema was present. Abdomen was distended with tenderness all over with evidence of free fluid and sluggish bowl sounds. Investigations like haemogram, serum electrolytes, renal function tests and X-ray of chest and abdomen were normal. Serum bilirubin and proteins level were 1.9 (0.1-1.2 mg/dl) and 7.7 g/dl (6-8 g/dl) respectively. USG abdomen showed significant free fluid in abdomen. With a suspicion of peritonitis, emergency laparotomy was carried out. The intra-operative finding were; significant ascites, gut oedema and liver contusion. After surgery worsening of clinical condition continued with added development of significant abdominal wall oedema and hypotension. A repeat CECT abdomen was done on postoperative day 10 which revealed a large thrombus in the supra-renal part of IVC along with liver injury and perihepatic fluid collection (Figs 2,3 CECT abdomen showing thrombus in the supra-renal part of IVC).

Patient was managed successfully with low molecular weight heparin (LMWH) and warfarin for 6 months. Thrombus disappeared completely and patient recovered fully without any further complication.

Case 2 (Pyogenic Liver Abscess Causing IVC Compression) :

A 3-year-old male presented to paediatric emergency with 4 days history of pain right upper abdomen, high grade fever and non-bilious vomiting. On general physical examination heart rate, blood pressure respiratory rate were 114/min, 112/72mm of Hg, 41/min respectively. Abdominal examination revealed tender hepatomegaly. Ultrasound, CECT and MRI studies of abdomen showed 8.7* 6.4 cm size lesion involving VI, VII, VIII segments of liver with partial liquefaction causing significant compression of IVC and ascites (Figs 4,5 CECT abdomen showing IVC compression due to liver abscess). Peritoneal fluid showed lymphocytes. In serology tests ANA was negative and amoebic IgG level was 0.51 U.

Around 30 ml of thick, creamy pus was aspirated under USG guidance and culture & sensitivity revealed growth of klebsiella. Antibiotics according to culture sensitivity were started. Even after aspiration and appropriate broad spectrum antibiotics treatment, there was no resolution of symptoms. His clinical condition worsened with development of hypotension refractory to vasopressors, oedema of extremities, abdominal distension, ascites and pleural effusion. There was no decrease in size of the abscess as revealed by repeated ultrasound examination. So under general anaesthesia, open exploration of the abscess cavity via right sub-costal

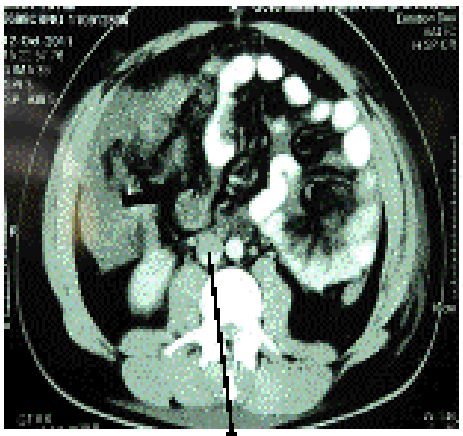


Fig 2

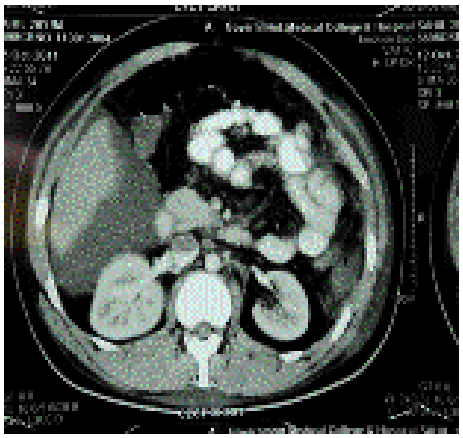
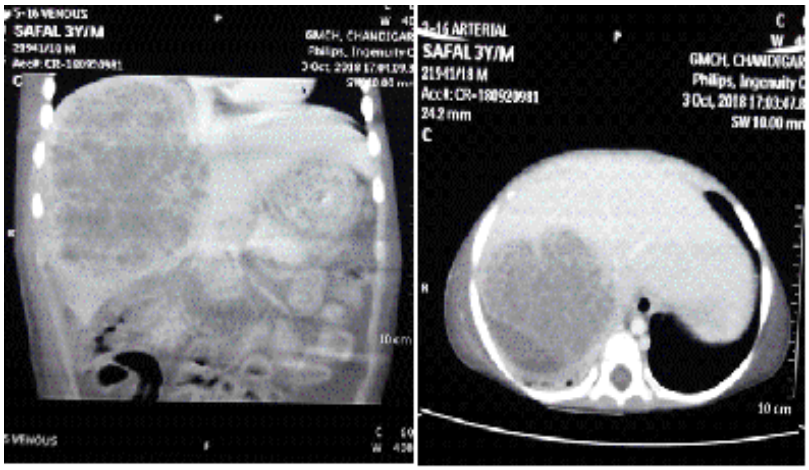


Fig 3

Figs 2 & 3 — CECT abdomen showing thrombus in the supra-renal part of IVC (arrow)

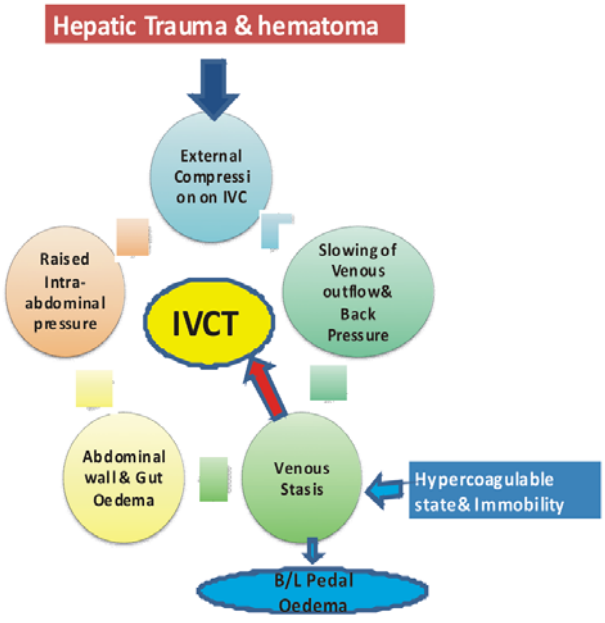
incision was carried out and around 200 ml of frank thick, creamy pus was drained along with placement of corrugated drain in the abscess cavity. Antibiotics were administered as per culture sensitivity and vasopressors were weaned off on the second PO day. Drain was removed on 4th postoperative day and he was discharged in satisfactory condition after 10 days of surgery.



Figs 4 & 5 — CECT abdomen showing IVC compression due to liver abscess

Discussion

Syndrome due to obstruction of superior vena cava is a well known, however, the syndrome related to occlusion of inferior vena cava is not commonly described. IVC thrombosis (IVCT) in abdominal trauma is uncommon and potentially life threatening complication[13,14]. The risk factors include; congenital anomaly of IVC, advanced age, obesity atherosclerotic patch, prolonged bed rest, hypercoagulable states, prior history of DVT, prolonged bed rest and malignancy[1,5,10,13,14]. Following mechanisms have been attributed in the pathophysiology of post traumatic IVCT; (a) Endothelial injury of the caval wall with secondary thrombus formation, (b) Caval stasis secondary to compression by a pericaval or retroperitoneal hematoma, (c) Hepatic vein thrombosis after liver laceration extending into the IVC, (d) Hypercoagulable state after major trauma.[2,3] In our case external compression on IVC by pericaval haematoma and liver trauma resulted in stasis of venous flow which was compounded further by bed rest, gut oedema and laparotomy leading on to IVCT. The vicious cycle of IVCT development in the present case is explained in the flow chart.



The IVCT may occur within few hours, few days, few weeks or 4 years after trauma[15,16]. IVCT developed in 3rd week in our case. Serial imaging studies should be done in abdominal trauma if there is ongoing nonspecific clinical course. The clinical presentations depend on the speed and degree of occlusion, presence of collaterals and flow of the venous system. Patients with slow and partial occlusion with intact collaterals may be completely asymptomatic, however, cases with sudden occlusion usually present with acute abdominal or back pain, sudden development of lower limb and abdominal wall oedema, hypotension due to decreased venous return, dilated superficial veins, brownish discoloration and ulceration of skin due to venous stasis. Extension of thrombus in hepatic veins may results in Budd-Chiari syndrome. Extension of thrombus in right atrium and pulmonary embolism are the potentially life threatening complications. Among various radiological studies USG, CECT, Doppler and MR imaging with angiography are important diagnostic tools.

Treatment of IVCT is decided carefully based on the underlying aetiology, degree of occlusion and severity of the clinical condition. Once identified, prompt treatment must be initiated for the dissolution of the thrombus, to avoid clot migration and chronic complications. Treatment approach with anticoagulants only or combination of anticoagulants with intervention procedures is titrated carefully as per individual patient and underlying aetiology. Anticoagulation with low molecular weight heparin with close monitoring in ICU setting followed by warfarin is an effective treatment, however, It should be avoided in case of active bleeding & uncontrolled hypertension. Known side effects of anticoagulant therapy are bleeding and heparin induced thrombocytopenia. Prophylactic insertion of a Greenfield caval filters with anticoagulants may be undertaken as a precautionary step to trap any large emboli that might have been produced during dissolution, to avoid fatal pulmonary embolism [4,14,15]. Other thrombectomy modalities are;

catheter-directed thrombolysis, AngioJet rheolytic thrombectomy, the Trellis peripheral infusion system, amplatz thrombectomy device and the angiovac for aspiration thrombectomy[15,16].

The first CECT abdomen in our case of BTA showed only injury of caudate lobe of liver and mild haemoperitoneum without any evidence of IVC lesion. After two weeks of discharge he was again admitted with abdominal tenderness, distension, free fluid, sluggish bowel sounds and oedema of extremities. USG abdomen showed gross free fluid. Laparotomy undertaken in view of peritonitis revealed; gut oedema, liver contusion and ascites. Repeat CECT abdomen after 10 days of surgery revealed a large thrombus in IVC. The case was managed with low molecular weight heparin (LMWH) and warfarin.

Case 2 (Liver Abscess Causing Ivc Compression) :

Amoebic or pyogenic liver abscesses or rarely hydatid cyst in endemic areas may result in occlusion of portal vein, IVC or hepatic venous outflow by thrombosis or external compression.[7,13-20] The vascular involvement may result in high morbidity and mortality due to the potentially life threatening complication. Children with large liver abscess are more prone to develop complete IVC obstruction as they have relatively small liver tissue mass. The location of the abscess rather than its volume or size is more important than its volume or size. Abscess in vicinity of IVC or portal vein is more likely to compress these vessels than the one away from it. Doppler study (sensitivity of 85-95%) may demonstrate reduced calibre and flow of IVC, portal vein or hepatic veins. In our case CECT and MRI abdomen revealed a large pyogenic abscess in VI, VII and VIII segments of liver causing complete IVC compression. The patient did not improve with medical management by antibiotics. Due to thick pus and multiple septa in the abscess cavity, complete aspiration under USG guidance failed and hence open surgical drainage was done in combination with appropriate antibiotic therapy. The patient recovered completely after the drainage.

Conclusion :

Development of post traumatic IVCT is a challenging clinical scenario from diagnostic and management point of view. In abdominal trauma, combination of a high index of suspicion and repeated radiological examination may help in early recognition and prompt aggressive management of IVCT to prevent potentially life threatening complications of IVCT. In IVC compression due to liver abscess, decompression by early drainage along with appropriate medical treatment results in excellent outcome.

Acknoledgments : None
Funding : Nil
Conflict of interest : None

References

1 Coon WW — Epidemiology of venous thromboembolism. *Ann Surg* 1997; **186:** 149-64.
2 Grmoljez PF, Donovan JF, Willman VL — Traumatic inferior vena cava obstruction. *J Trauma* 1976; **16(9):** 746-8.
3 Campbell DN, Liechty RD, Rutherford RB — Traumatic thrombosis of the inferior vena cava. *J Trauma* 1981; **21(5):** 413-5.
4 Nagy KK, Duarte B — Post-traumatic inferior vena caval thrombosis: case report. *J Trauma* 1990; **30(2):** 218-21.
5 Glen PM — Thrombosis of the iliac artery from the seat belt injury. (letter). *West J Med* 1985; **142:** 400-1.
6 Yun M, Kim GH, Cho H, Kim BU — Hypotension due to compression of inferior vena cava by intrathoracic herniation of peritoneal fat during laparoscopic surgery. *Anesth Pain Med* 2018; **13:** 72-6.
7 M Soriano Hervas, D Robles-Hernandez, L Herraro Bernuz, M Almena Marjalizo, I Laros Beltran IL, *et al* — Inferior vena cava compression by an hydatid cyst. EC Anaesthesia **5.6(2019):** 169-72.
8 Sharma MP, Sarin SK — Inferior vena caval obstruction due to amoebic liver abscess. *J Assoc Physicians India* 1982; **30:** 243-4.
9 Sodhi KS, Ojili V, Sakhuja V, Khandelwal N. Hepatic and inferior vena caval thrombosis: vascular complication of amebic liver abscess. *J Emerg Med* 2008; **34:** 155-7.
10 Kallafallah AA, Renu S, Sharp C, Hannan T — Extensive venous thrombosis in a healthy young man with a short inferior vena cava syndrome treated successfully with revaroxaban. *BMJ Case Rep* 2012; doi:10.1136/bcr-2012-007313.
11 Guiu XJ, Villegas AR, Batalla SR, Marti-Mestre FJ, Mestre AR, *et al* — Inferior vena cava syndrome of uncommon aetiology. *Surg Rehabil* 2018; **2(2):** 1-3.
12 O'Connor NTJ, Fletchner EW, Sharp AA — Significance of idiopathic deep venous thrombosis. *Postgrad Med J*1984; **60:** 275-7.
13 Roehm EF, Twiest MW, Willaims RC — Abdominal aortic thrombosis in association with an attempted Heimlich maneuver. *JAMA* 1983; **249:** 1186-7.
14 Frank MS, Stern EJ, Hoy HM — Occult complication of non-operative treatment of blunt liver injury: detection by CT, *AJR* 1994; **163:** 333-4.
15 Castelli P, Caronno R, Piffaretti G, Tozzi M — Emergency endovascular repair for traumatic injury of the inferior vena cava. *Eur J Cardiothorac Surg* 2005; **28(6):** 906-8.
16 Salloum C, Lim C, Hillion ML, Azoulay D — Reconstruction of the inferior vena cava due to blunt hepatic trauma. *J Visc Surg* 2016; **153(1):** 75-6.
17 Huddle KR — Amoebic liver abscess, inferior vena-caval compression and the nephrotic syndrome. A case report. *S Afr Med J* 1982; **61:** 758-60.
18 Hodkinson J, Couper-Smith J, Kew MC — Inferior vena caval and right atrial thrombosis complicating an amebic hepatic abscess. *Am J Gastroenterol* 1988; **83:** 786-8.
19 Schmid BD, Lalyre Y, Sigel B, Kiani R, Layden TJ — Inferior vena cava obstruction complicating amebic liver abscess. *Dig Dis Sci* 1982; **27:** 565-9.
20 Yang DM, Kim HN, Kang JH, Seo TS, Park CH, Kim HS — Complications of pyogenic hepatic abscess: computed tomography and clinical features. *J Comput Assist Tomogr* 2004; **28:** 311-7.