INTRODUCTION

Venous outflow obstruction can lead to ambulatory venous hypertension and chronic venous insufficiency. Their treatment represents a large amount of health care expenditure, estimated at over 1 billion dollars per year in USA ***(Titus et al., 2011).***

Chronic outflow obstruction after an episode of acute DVT manifests itself as a constellation of symptoms known as the postthrombotic syndrome (PTS). This includes symptoms of persistent pain, swelling, skin changes, and ulceration. As many as 29% to 82% of patients will develop PTS after an episode of DVT ***(Titus et al., 2011).***

These symptoms are thought to result from the chronic outflow obstruction after thrombosis and occlusion of the venous lumen. During this process, inflammatory changes take place that can result in vein wall fibrosis, leading to valve dysfunction, reflux, and insufficiency ***(Titus et al., 2011).***

Studies have shown that early thrombus removal and decreasing the incidence of recurrent thrombotic events can reduce the likelihood of developing PTS ***(Neglén et al., 2010).***

Chronic outflow obstruction can also result from extrinsic forces compressing the iliac vein. These include entities such as

May-Thurner syndrome (compression of the left iliac vein from an overriding right common iliac artery) or pelvic tumors, fluid collections, or fibrosis ***(Titus et al., 2011).***

Iliac vein compression syndrome was described by May and Thurner in 1957, they observed compression of the left common iliac vein between the overriding right common iliac artery and an underlying vertebral body in 22% of 430 autopsies. Chronic compression results in the formation of venous ‘‘spurs’’ that lead to acute or chronic thrombosis of the left iliac veins ***(Moudgill et al., 2009).***

Although the majority of cases are generally believed to be related to venous valvular incompetence and abnormal reflux, awareness of the importance of iliac venous obstruction as a cause of lower extremity symptoms is increasing ***(Marston et al., 2011).***

It has previously been reported that patients with severe symptoms of chronic venous disease have a high incidence of compression of the iliocaval system when studied with intravascular ultrasound (IVUS) ***(Marston et al., 2011).***

Although occlusions of the femoropopliteal venous system may be well tolerated because of the presence of duplicated femoral and popliteal veins or the collateralization to the saphenous and accessory saphenous systems, more proximal

obstructive processes are not always well tolerated **(*Brian et al., 2013).***

The Chronic occlusion of the inferior vena cava (IVC) is a condition, with a variety of underlying diseases. Patients have a myriad of signs and symptoms, which vary dependent on collateral venous drainage. Symptoms at presentation include bilateral lower extremity pain and edema; skin changes such as eczema, pigmentation, and ulceration; back pain; inguinal, abdominal, and thoracic varicosities; and occasionally, impaired kidney or liver function if the renal veins or intrahepatic vena cava are involved ***(Mark et al., 2005).***

This variable clinical presentation relates to the rich collateral pathways that may develop in these patients. Collateralization from the common iliac vein via the thoracolumbar vein is a particularly important collateral pathway and associated occlusion of the common iliac vein is particularly important in the development of symptoms ***(Raju et al., 2006).***

Patients at the end stage of chronic venous insufficiency (CEAP clinical class 5 and 6) have complex venous disease, with involvement of deep, superficial, and perforator veins in the majority of cases ***(Marston et al., 2011).***

The diagnosis of occlusive or non-occlusive obstruction is therefore based on morphological studies (50% stenosis is

considered significant, which has been arbitrarily chosen based on clinical outcome) Since ultrasound scanning of the pelvic outflow is suboptimal in the detection of obstruction, venography (transfemoral, magnetic resonance, or computed tomography), should be performed in patients with severe chronic venous insufficiency (CEAP class C3–6) ***(Neglén et al., 2010).***

However, The diagnostic sensitivity of venography for nonthrombotic iliac vein type lesions is known to be poor. Liberal use of the more sensitive IVUS yields a more diverse picture of the syndrome than previously appreciated; particularly, we found the incidence of nonthrombotic iliac vein type lesions to be very high in symptomatic CVD cases ***(Raju et al., 2006).***

The standard treatment for deep venous thrombosis is anticoagulation with low molecular weight heparin followed by oral warfarin but the fact that the removal of thrombus is not one of the effects accomplished by anticoagulation may have a sever impact in the long term disability caused by DVT ***(Rosales et al., 2010).***

The treatment options for chronic vein occlusion have changed in the 1990 with the introduction of endovascular techniques. Previously, open surgical bypass surgery for chronic

venous obstruction was done, but with disappointing result at long term follow up ***(Alhadad et al ., 2011).***

Surgical therapy has been reserved for the most severe cases, and although some reports show beneficial results, this patient population tends to have significant comorbid conditions and long segment central venous occlusion, making surgical intervention risky and associated with a high failure rate ***(Mark et al., 2005).***

The pattern of spontanoeus recanalization after DVT varies according to the anatomical segment as shown by colour duplex ultra sound studies.While recanalization occurs in up to 90% of femoro-popliteal veins after one year. This is rarely the case after iliofemoral thrombosis (5%). Persistent chronic venous outflow obstruction in the iliofemoral vein leads to the development of venous claudication in about 43% of these patients ***(Rosales et al., 2010).***

However, the development of percutaneous endovascular techniques has made possible the recanalization and stenting of chronic venous occlusion with successful clinical outcomes and low morbidity ***(Rosales et al., 2010).***

Regardless of the cause, interventional treatment of outflow obstruction in the form of angioplasty and stenting has

been shown to relieve symptoms and improve quality of life ***(Raju et al., 2005).***

It has also been found to have comparable if not better patency rates than the currently available open surgical alternatives (bypass) ***(Gloviczki et al., 2005).***

Endovenous angioplasty, combined with stenting, is a sure, safe, effective and very minimally invasive technique, which provides good long-term patency rates. Currently, it is recognized as the technique of choice for the treatment of ilio-caval obstructive lesions ***(Hartung et al., 2009).***

AIM OF THE WORK

To evaluate the efficacy of endovascular treatment techniques as the primary choice for management of patients with chronic iliocaval occlusion and higlight it's role in decreasing the morbidity and improving the quality of life for this type of patients.

ANATOMY

- **The Inferior vena cava normal anatomy**

The IVC, the largest vein in the body, has no valves except for a variable, non-functional one at its orifice in the right atrium of the heart. The IVC returns poorly oxygenated blood from the lower limbs, most of the back, the abdominal walls, and the abdominopelvic viscera. Blood from the abdominal viscera passes through the portal venous system and the liver before entering the IVC via the hepatic veins ***(Moore et al., 2011).***

The IVC typically has an oval shape in cross- section but is easily deformed by adjacent abdominal or retroperitoneal masses. The average diameter of the infrarenal IVC is approximately 23mm, although the intrarenal segment is usually slightly larger. The IVC is an elastic structure that responds to decrease volume or increaesed intraabdominal pressure by collapsing.The dynamic nature of The IVC should be considered when interpreting imaging studies or contempling interventions ***(Kaufman et al., 2014).***

The inferior vena cava begins at the confluence of the common iliac veins and ascends on the right side of the vertebral column, passes through the tendinous portion of the diaphragm, and after a short course (approximately 2.5 cm) in the chest it

terminates in the right atrium at the level of T8 ***(Moore et al., 2011).***

In the upper abdomen the IVC is located posterior to the duodenum, the head and neck of the pancreas, the lesser sac, and the liver. The intrahepatic portion of the IVC lies in a groove along the posterior aspect of the caudate lobe ***(Moore et al., 2011).***

Tributaries of the IVC are the paired lumbar and renal veins and the hepatic veins, additionally on the right side the right gonadal, suprarenal, and inferior phrenic veins also drain into the IVC. The left gonadal and suprarenal veins join the left renal vein, the left inferior phrenic vein drains into the left suprarenal vein ***(Moore et al., 2011).***

The branches corresponding to the paired visceral branches of the abdominal aorta include the right suprarenal vein, the right and left renal veins, and the right gonadal (testicular or ovarian) vein. The left suprarenal and gonadal veins drain indirectly into the IVC because they are tributaries of the left renal vein ***(Moore et al., 2011).***

Paired parietal branches of the IVC include the inferior phrenic veins, the 3rd (L3) and 4th (L4) lumbar veins, and the common iliac veins. The ascending lumbar and azygos veins connect the IVC and SVC, either directly or indirectly providing collateral pathways ***(Moore et al., 2011).***

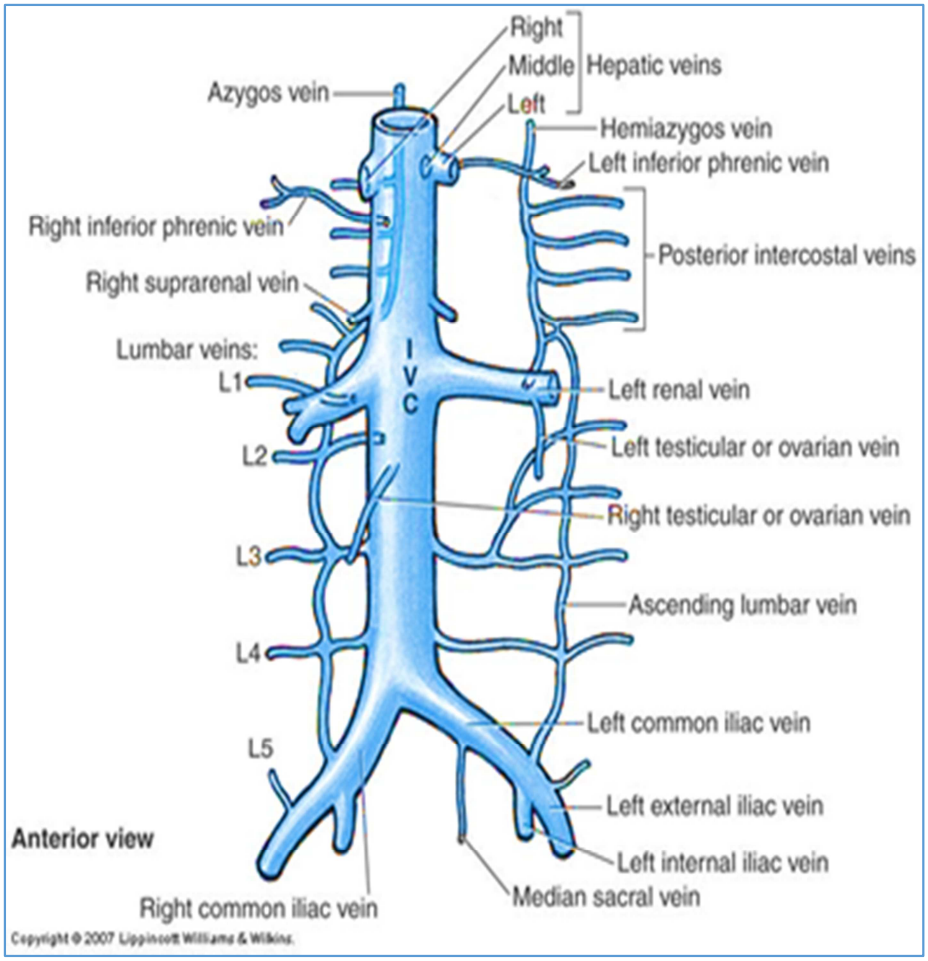

Fig. (1): IVC anatomy ***(Moore et al., 2011).***

- **Embryological Development and anomalies of the IVC**

The inferior part of the IVC has a complicated developmental history because it forms from parts of three sets of embryonic veins.Therefore, IVC anomalies are relatively common, and most of them, such as a persistent left IVC, occur

inferior to the renal veins.These anomalies usually result from the persistence of embryonic veins ***(Moore et al., 2011).***

The three pairs of fetal vein that become the IVC are the posterior cardinal, the subcardinal and the supracardinal. The posterior cardinal veins normaly involutes completely, althougt persistence on the right result in retrocaval ureter, the subcardinal veins form the intrahepatic IVC, and contribute to the renal veins and suprarenal segment of the IVC. Regression of the right subcardinal veins results in azygos or hemiazygos continuation of the IVC. The infrarenal IVC and the azygos veins derived from the supracardinal veins ***(Kaufman et al., 2014).***

Duplication of the infrarenal results from failure of regression of the left supracardinal veins, while a left sided IVC results from regression of the right supracardinal veins. When there is caval duplication each iliac vein is usually isolated drains through its own IVC, although communication at the normal of the iliac vein conflunce may also occur. When there is only a single left sided IVC, both iliac vein drains into the IVC, which usually crosses the aorta at the level of left renal vein to form a normally located suprarenal vein. Thus unless there is an associated anomaly of the subcardinal veins, duplicated or left sided IVCs usually revert to a normal location above the level of renal veins ***(kaufman et al., 2014).***

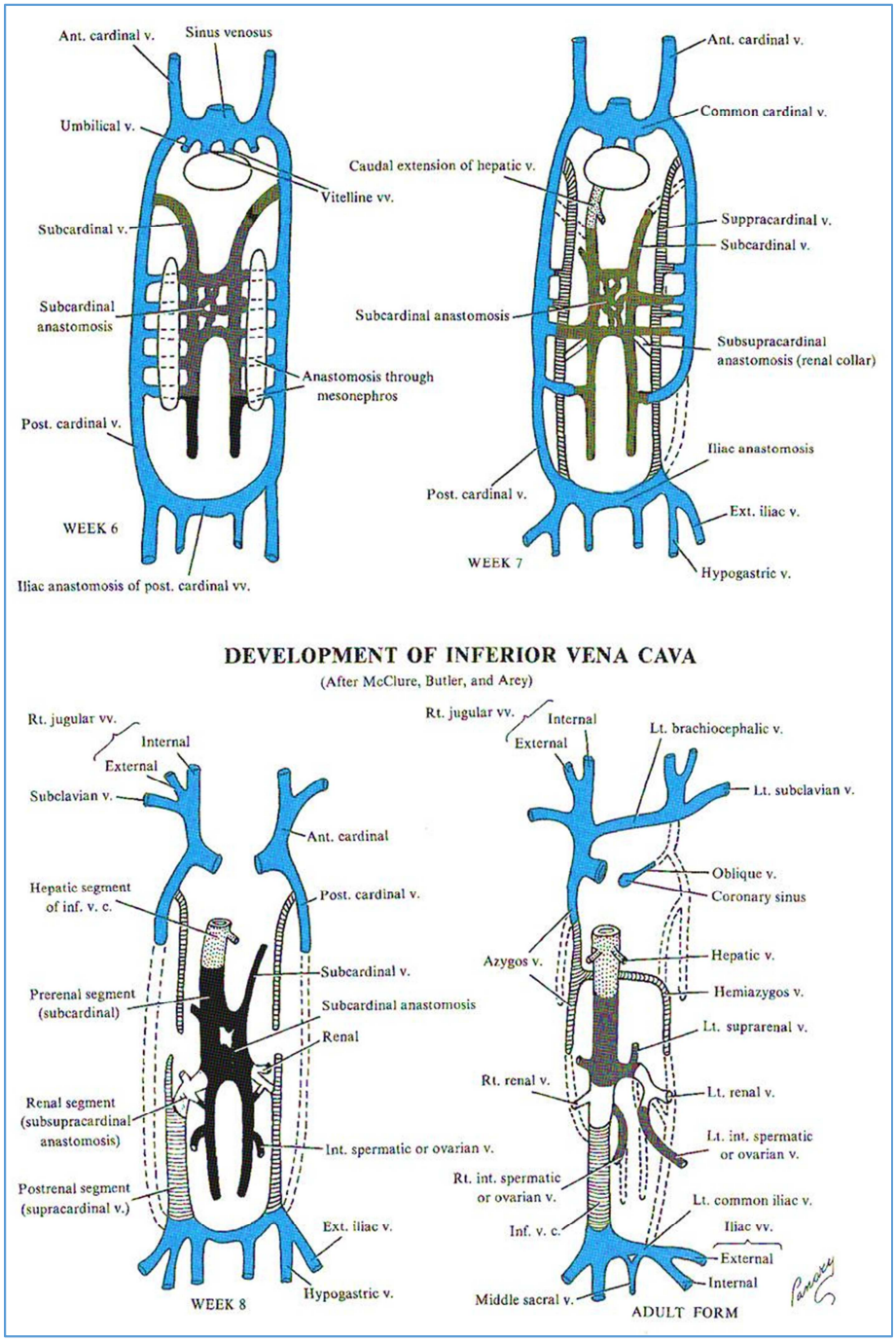

Fig. (2): Embryological Development of the IVC. ***(Moore et al., 2011).***

- **The Iliac veins**

The common iliac veins begin at the sacroiliac joint on both sides and end at L5, where they form the IVC. The only tributary of the right common iliac vein is the right ascending lumbar vein; the left common iliac vein drains the left ***(Moore et al., 2011).***

The right common iliac vein lies postero-lateral to the right common iliac artery. The distal segment of the left common iliac vein is medial and posterior to the left common iliac artery, the proximal segment is posterior to the right iliac artery and distal aorta ***(Moore et al., 2011).***

Compression of the proximal left common iliac vein may occur due to the overlying arterial structures. The external iliac vein starts at the level of the inguinal ligament, it courses along the pelvic brim and ends anterior to the sacroiliac joint where the external and internal iliac veins form the common iliac vein. On the right the distal external iliac vein is medial to the artery; however, as it ascends, more proximally, it courses posterior to it. The left external iliac vein remains medial to the artery along its entire course ***(Bergan et al., 2007).***

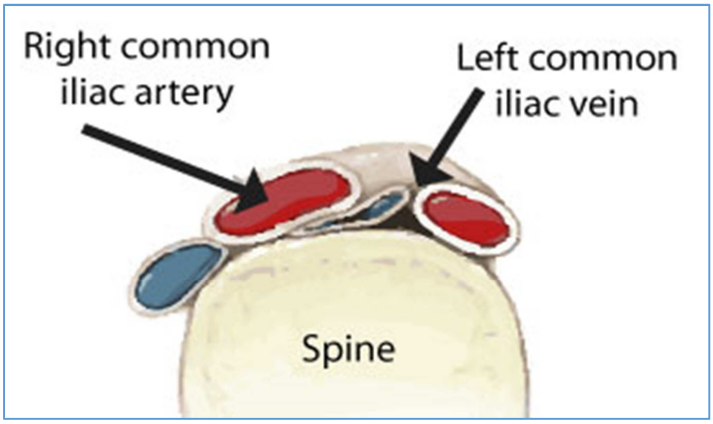

Fig. (3): Transverse view show iliac vein compression syndrome. ***(http://www.quazoo.com 2013).***

Tributaries of the external iliac vein are the inferior epigastric, deep circumflex iliac, and pubic veins. The internal iliac vein runs postero-medial to the internal iliac artery on both sides. The short trunk of internal iliac vein is formed by the confluence of extra and intrapelvic venous tributaries ***(Bergan et al., 2007).***

The extrapelvic tributaries include the gluteal (superior and inferior), internal pudendal, and obturator veins, which drain the pelvic wall and the perineum. The intrapelvic tributaries of the internal iliac vein are the lateral sacral and visceral (middle rectal, vesical, uterine, and vaginal) veins, which drain the presacral and pelvic visceral venous plexuses (rectal, vesical, prostatic, uterine, and vaginal.Both the IVC and the common iliac veins are valveless. There is usually one valve in the

external iliac vein, however often it is without any valves ***(Bergan et al., 2007).***

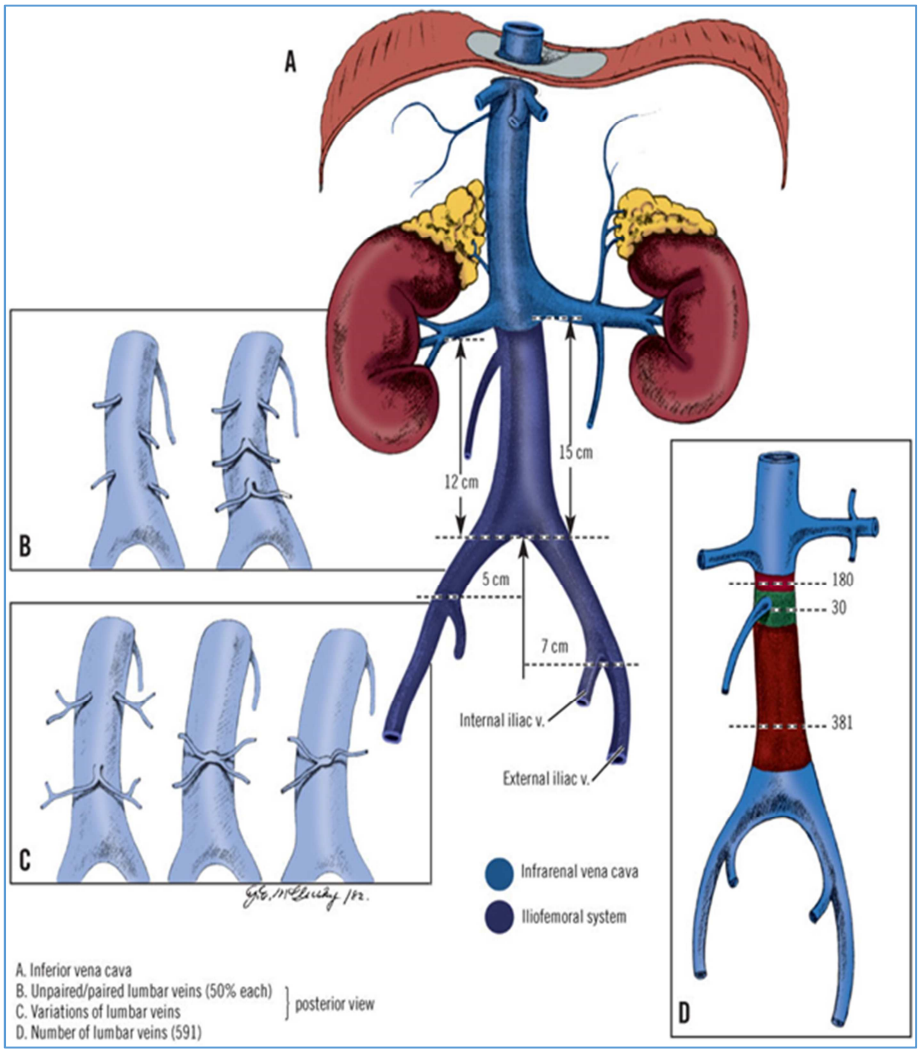

Fig. (4): Anatomical variations of IVC, Iliac veins and it's tributaries ***(Moore et al., 2011).***