Introduction

Central venous stenosis and obstruction is a common and significant problem in the long term management of the access circuit for patients undergoing haemodialysis. Central Venous Stenosis (CVS) disrupts the haemodialysis access circuit by causing venous hypertension and access flow dysfunction with or without debilitating symptoms ***(Kundu, 2010).***

The exact incidence of central venous lesions in the dialysis population is unknown. It is estimated that between 5% and 20% of dialysis patients develop central venous stenosis. The incidence of significant stenosis following subclavian vein catheter placement is 42% to 50%, it is 10% in patients with internal jugular catheters ***(Dosluoglu et al., 2010).***

One suggested mechanism for the development of central venous stenosis includes central venous catheter-induced central trauma to the venous endothelium and secondary inflammatory damage within the vessels wall at the time of insertion. Other proposed mechanisms include the presence of a foreign body in the vein, along with increased flow and turbulence from the creation of an arterio-venous (AV) access ***(Peden, 2011).***

A history of central venous access placement or central vein intervention in patients undergoing haemodialysis is the most common risk factor for central venous occlusion. Placement of multiple central venous catheters indwell times, have been associated with an increased risk of CVS. The access site for central venous catheter placement is also an important risk factor for central venous occlusion ***(Kundu, 2010).***

There are three possible outcomes of central venous occlusion in the presence of ipsilateral AV access: the patient remains asymptomatic due to adequate venous collaterals, thrombosis of the AV access, or venous hypertension due to patent access with inadequate collaterals .the sequelae of venous hypertension include distal extremity swelling that can be severe, bluish discoloration, pigmentation of the skin and in advanced cases, ulceration of the finger tips and neuralgia ***(Neville et al., 2004).***

Central venous occlusion can be asymptomatic and detected on diagnostic venogram or fistulogram before access placement for an immature fistula .Most occult Central venous occlusion cases become clinically apparent after development of a functioning AV access in the ipsilateral extremity. The diagnosis of Central venous occlusion is based on clinical and imaging findings. Depending on the location of the access, a proportion of patients will have evidence of AV access dysfunction, with decreased access flow rates. On physical

examination, there may be numerous dilated collaterals vessels in the neck and chest and arm edema on the side of the Central venous occlusion. It is difficult to visualize the central veins with duplex in patients with a high body mass index or significant chest musculature. Digital subtraction central contrast venography is the current gold standard for the diagnosis of Central venous occlusion ***(Kundu, 2010).***

Treatment options to date include balloon angioplasty, bare metal stents, and recently, placement of covered stents and surgical bypass. Endovascular intervention is the present mainstay of treatment in the haemodialysis of patients with CVS. The National Kidney Foundation disease outcomes Quality Initiative guidelines recommend PTA with or without stent placement as the preferred treatment approach to Central venous occlusion ***(Kundu, 2010).***

Chapter (1):

Surgical Anatomy of Upper Limb Venous System

ANATOMY OF THE UPPER LIMB VEINS

Cephalic vein

The cephalic vein originates in the web space between the thumb and the index finger. It often has a fairly constant position near the anatomic snuff-box between the tendons of extensor pollicus brevis and extensor pollicis longus. The cephalic vein travels further on to the antero-lateral aspect of the forearm at the level of the wrist where it usually runs parallel to the radial artery, usually within a centimeter for a variable distance (4-5 cm length) and is joined by a tributary that often drains medial aspect of the back of the hand. This dorsal tributary of the cephalic vein can often be the major component of the cephalic vein ***(Sekar, 1993).***

When the dorsal tributary is the major component of the cephalic vein it is observed very well on the posterior-lateral aspect of the hand (using this vein on the back of the hand for venipuncture saves rest of the cephalic venous system for access creation in ESRD patients).The junction of this prominent dorsal tributary to the main cephalic vein is usually

located 7-10 cm from the proximal wrist crease The cephalic vein continues further in the superficial fascia medially towards the cubital fossa ***(Johnson et al., 2008).***

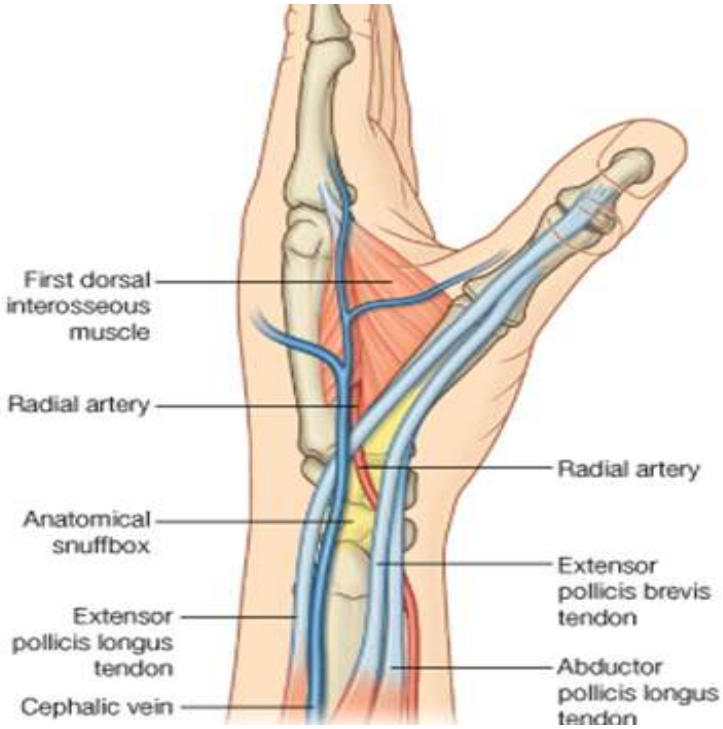

Fig. (1): Cephalic vein formation in wrist ***(Patrick and Thomas, 2009).***

Near the cubital fossa, the forearm cephalic vein branches into the median cubital vein and the upper arm cephalic vein.The median cubital vein travels above the elbow towards the medial aspect of the lower third of the upper arm where it joins the basilic vein ***(Johnson et al., 2008).***

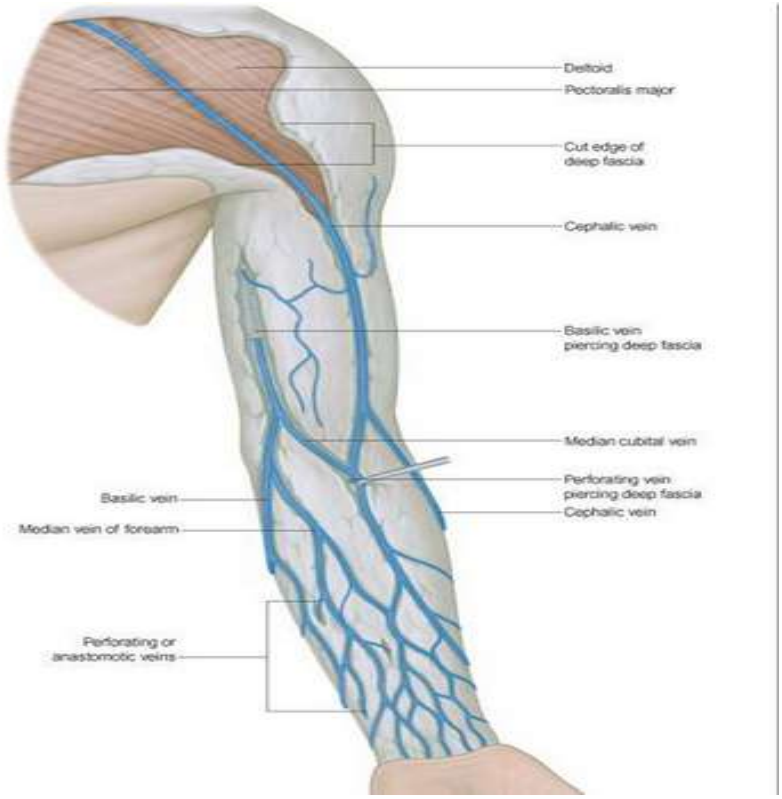

Fig (2): Illustration of cephalic and basilic veins in arm and forearm ***(Patrick and Thomas, 2009).***

Basilic vein

The basilic vein is a superficial vein in the forearm. It originates in the ulnar aspect of the dorsal venous network of the hand near the wrist. As it travels on the posterior aspect of the ulnar side of the forearm in the mid and lower third, it often receives couple of communicating branches from the cephalic vein ***(Moore, 2006).***

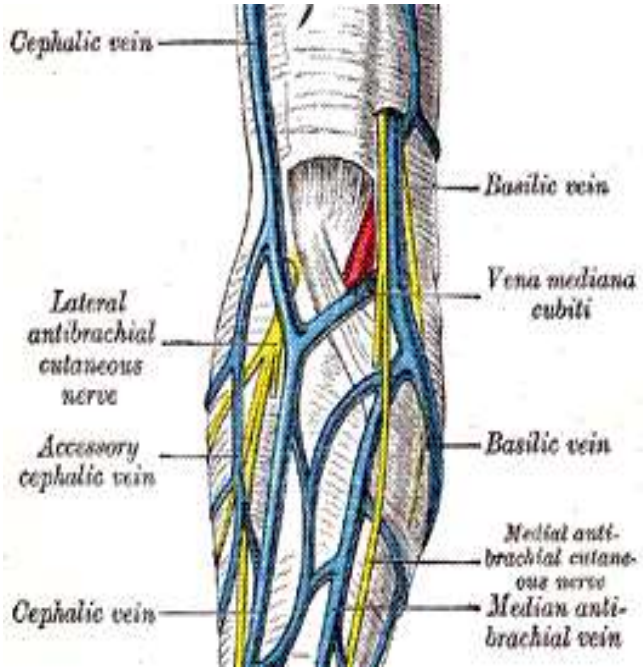

Fig. (3): Median Cubital vein communicating basilic and cephalic veins ***(Bittl 2010).***

The basilic vein runs on the medial aspect of the upper part of the forearm as it approaches the elbow. At the level of the elbow, it is always anterior to the medial epicondyle (important landmark to locate the basilic vein during difficult vein mapping with US). In the lower third of the upper arm, the basilic vein often has a reasonable caliber (3-4 mm) but is well masked by subcutaneous fat. In this location it is joined by the median cubital vein. As it travels higher, it pierces the deep fascia and becomes a deep vein that accompanies the brachial artery and usually joins the deep brachial venae comitans in the mid upper arm ***(Moore, 2006).***

Axillary Vein

Its origin is at the lower margin of the teres major muscle and a continuation of the brachial vein. This large vein is formed by the brachial vein and the basilic vein. At its terminal part, it is also joined by the cephalic vein. Other tributaries include the subscapular vein, circumflex humeral vein, lateral thoracic vein and thoraco-acromial vein. It terminates at the lateral margin of the first rib, at which it becomes the subclavian vein. It is accompanied along its course by a similarly named artery, the axillary artery ***(Moore, 2006).***

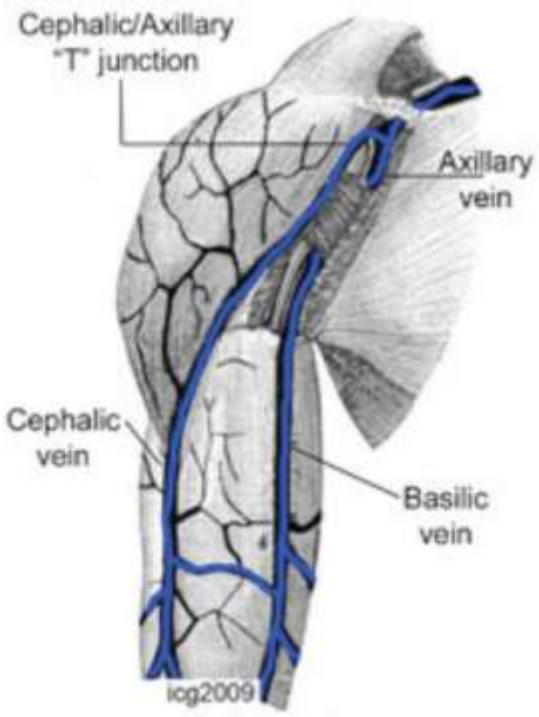

Fig. (4): Formation of axillary vein ***(Bittl, 2010).***

ANATOMY OF THE CENTRAL VEINS

Relatively detailed anatomical knowledge of the central veins is a prerequisite for safe placement of CVCs, and to enable identification of abnormalities when they occur. A description of the normal anatomy of the central veins within the thorax is as follows ***(Ellis et al., 2004).***

Brachiocephalic veins

The brachiocephalic (innominate) veins are two large trunks, placed one on either side of the root of the neck and formed by the union of the IJ and SCVs of the corresponding side; they are devoid of valves. The right brachiocephalic vein is ≈ 2.5 cm long, and begins behind the sternal end of the clavicle and passes almost vertically downwards, joining with the left brachiocephalic vein, just below the cartilage of the first rib, close to the right border of the sternum. Here it forms the SVC. It lies anteriorly and to the right of the brachiocephalic artery ***(Stonelake and Bodenham, 2006).***

The right brachiocephalic vein, at its commencement, receives the right vertebral vein and, lower down, the right internal thoracic (mammary) and right inferior thyroid veins. Sometimes the vein from the first intercostal space also joins here. Owing to its relatively straight course into the SVC, in terms of catheter placement, it could be functionally considered

as a proximal limb of the SVC. The left brachiocephalic vein, some 6 cm long, begins posterior to the sternal end of the left clavicle and runs obliquely downwards and to the right, behind the upper half of the manubrium sterni to the sternal end of the first right costal cartilage. Here, it unites with the right brachiocephalic vein to form the SVC. Behind it are the three large arteries, the right brachiocephalic, left common carotid, and the left subclavian artery, arising from the aortic arch, together with the vagus and phrenic nerves ***(Mallick and Bodenham, 2003).***

The left brachiocephalic vein may occupy a higher level, crossing the jugular notch and lying directly in front of the trachea. Its tributaries are the left vertebral, left internal thoracic (mammary), left inferior thyroid, and the left highest intercostal veins, and occasionally, some thymic and pericardiac veins. Its angle of approach to the right brachiocephalic vein is very variable and this is an important determinant of the ease of central catheter positioning from the left IJ and SCV routes. The more acute the angle is, the longer the distal section of catheter needed to be able to traverse the corner, and to lie in the longitudinal axis of the SVC or upper RA. This is important to avoid acute angulation against the wall of the SVC or RA, with the attendant risks of thrombosis, catheter failure, or perforation. Typically, catheter tips need to lie at the caval/atrial junction or upper RA after left-sided CVC placements ***(Mallick and Bodenham, 2003).***

The right brachiocephalic vein receives lymph from the right lymphatic duct, and the left brachiocephalic vein receives lymph from the thoracic duct. Such anatomy is very variable ***(Stonelake and Bodenham, 2006).***

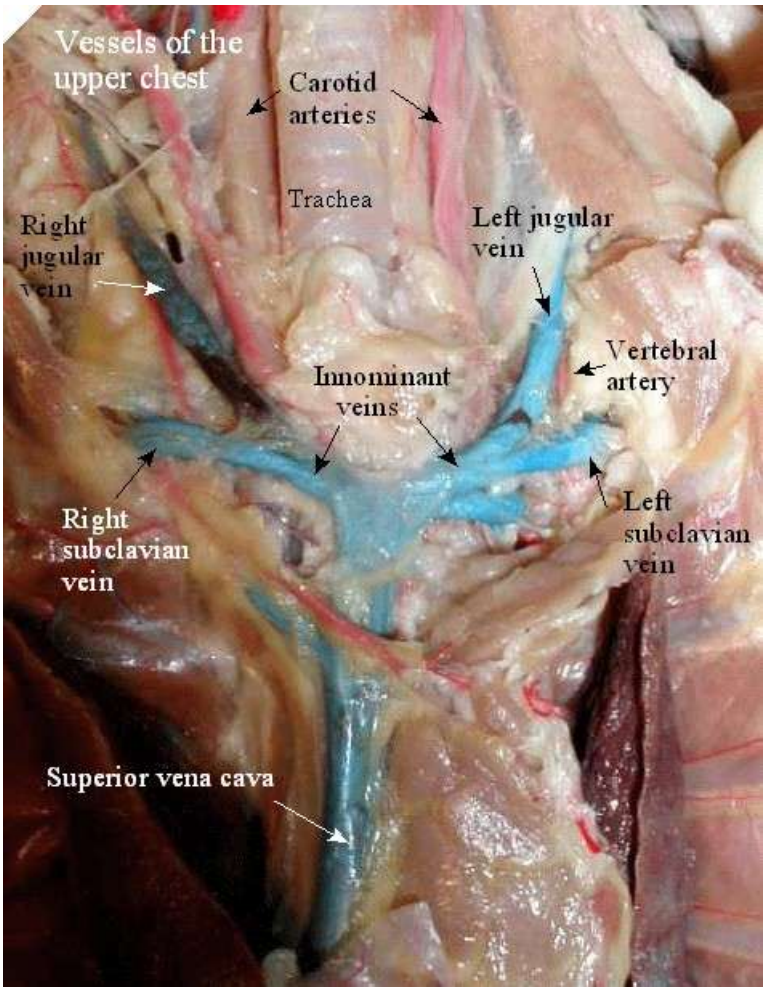

Fig. (5): Formation of Superior Vena Cava by right and left innominate veins ***(Gray, 1918).***

Superior vena cava

The SVC drains venous blood from the upper half of the body. It measures ≈7 cm in length and is formed by the junction of the two brachiocephalic veins behind the lower border of the first right costal cartilage near the sternum. It descends vertically behind the first and second intercostals spaces, ending in the upper part of the RA, opposite the upper border of the third right costal cartilage. In its course, it describes a slight curve, the convexity of which is posterior and to the right side. The SVC lies anterolateral to the trachea and posterolateral to the ascending aorta. The right phrenic nerve lies between the SVC and the mediastinal pleura ***(Albrecht et al., 2004).***

The terminal half of the SVC is in the middle mediastinum, where it lies beside the ascending aorta. The lower half of the vessel is within the pericardial sac. Just before it pierces the pericardium, it receives the azygous vein and several small veins from the pericardium and other structures from within the mediastinal cavity. The upper level of the pericardial sac, as it traverses the SVC, lies below the level of the carina. Hence, the use of the carina as an X-ray landmark to identify the placement of a CVC tip outside of the pericardium, therefore minimizing the small but serious risk of cardiac tamponade if the tip perforates the vessel wall ***(Albrecht et al., 2004).***

The SVC lies in close anatomical proximity to the mediastinal pleura in the upper thorax. Perforation of the vein wall here, with a guidewire, dilator, or catheter, may cause uncontrolled bleeding into the low pressure pleural space. A catheter while left in situ may control such bleeding which then becomes evident on removal. With age or disease, the SVC may become increasingly tortuous, which can cause difficulty when attempting to advance a guidewire or catheter. The SVC has no valves ***(Schuster et al., 2000).***

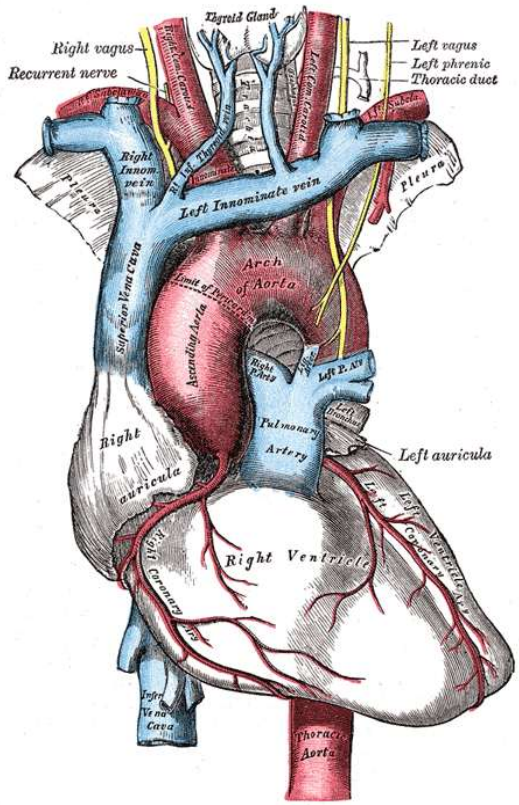

Fig. (6): Illustration of central veins draining in the right atrium ***(Gray, 1918).***

Chapter (2):

Pathophisiology and Clinical Picture of Central Venous Stenosis

PATHOPHYSIOLOGY

There has been a strong association of CVS, with the previous placement of central venous catheters and pacemaker wires In addition, there is a very high incidence of CVS in patients with a history of subclavian catheters of 42-50% compared with central venous catheters placed via the internal jugular vein route . One suggested mechanism for the development of CVS includes central venous catheter induced trauma to the venous endothelium and secondary inflammatory damage within the vessel wall at the time of insertion. Other proposed mechanisms include the presence of a foreign body in the vein, along with increased flow and turbulence from the creation of an arteriovenous (AV) access ***(Peden, 2011).***

It is rare for CVS to occur in HD patients, without a history of previous central venous access placement or previous intervention. Placement of multiple central venous catheters, with increased duration of catheter dwell times, has been associated with a higher risk of CVS. The access site for central venous catheter placement is also an important risk factor for CVS. Central venous catheters placed by a subclavian access,

have a particularly high risk, with a 42% incidence of CVS comparedto a 10% rate with catheters placed via an internal jugular vein access ***(Schon and Whittman, 2003).***

There is also an increased predilection for CVS to occur with left sided access for catheter placement, which may be related to the more tortuous course catheters have to traverse from a left sided access. The high incidence of CVS with HD catheters may berelated to the large caliber of these catheters or possibly the high flow rates required for HD. Peripherally inserted central catheters (PICC) and central venous port catheters are also becoming an increasingly important risk factor for CVS. Most patients with CVS secondary to peripherally inserted catheters and central venous port catheters, are usually asymptomatic, and present clinically after a hemodynamic challenge, such as placement of an ipsilateral AV access. Pacemaker and defibrillator wires can also lead to CVS, with development of clinical symptoms after the placement of an AV access in the ipsilateral extremity ***(Salgado et al., 2008).***

In addition, the status of the valves plays an important role in the development and severity of the symptoms. Increased venous pressures in the presence of incompetent venous valves distally in the arm lead to higher distal venous pressure and development of symptoms. This was frequently seen after side to- side arteriovenous anastomoses ***(Rutherford, 2000).***