Introduction

Venous Hypertension is a significant problem for the patients on regular haemodialysis that result in disabling upper extremity oedema and impairment of arteriovenous access function. The problem seems to be increasing in clinical importance as the ability to care medically for patients on haemodialysis continues to improve, resulting in patients living longer. This may be due to an actual increase in incidence of, or a greater awareness and recognition of, the problem ***(Elseviers et al., 2003).***

Venous hypertension after access construction is due to central venous stenosis or occlusion or valvular incompetence in the more peripheral arm veins with retrograde flow. The exact incidence of central venous lesions in the haemodialysis population is unknown. It is estimated that between 5% and 20% of haemodialysis patients develop central venous stenosis. The incidence of significant (>50%) central venous stenosis following subclavian vein catheter placement is 42% to 50%; it is 10% in patients with internal jugular catheters ***(Dosluoglu et al., 2010).***

Several factors have an impact on the development of central venous lesions, including longer catheter indwelling times, multiple catheterizations, and longer functioning

ipsilateral arterio-venous access after ipsilateral catheter placement ***(Dosluoglu et al., 2010).***

The pathophysiology is straightforward but is frequently misunderstood or misdiagnosed. The essential concept pairs a functioning arterio-venous access, which increases arterial blood flow to an extremity, with an obstruction to venous outflow. Pre-existing venous obstruction may not be readily recognized. To produce venous hypertension, the obstruction must be located close enough to the arterio-venous access outflow to permit filling of branch veins. If there were no intervening branches between the access and the obstruction, thrombosis would occur; thus, obstruction may produce a localized venous hypertension or affect an entire extremity. Central vein obstruction may affect one or both extremities. Normal blood flows are generally accommodated by the development of extensive collateral drainage beds. A slightly different variant is seen with the valvular incompetence that occurs with the less frequently constructed side-to-side autogenous arterio-venous access; dilation of the vein may render the valves in the branches incompetent, permitting high volume retrograde flow to be redirected distally. Progressive intimal hyperplastic stenosis in an outflow vein may produce similar volume redirection ***(Frank et al., 2008).***

Subclinical venous obstruction is often unmasked by the increased flow of a well functioning arterio-venous access ***(Frank et al., 2008).***

Vneous hypertension after arterio-venous access construction is important because of unilateral symptoms may reflect regional or central vein obstruction, superior vena cava obstruction may eliminate consideration of both upper extremities and venous hypertension may produce typical symptoms of venous insufficiency ***(Frank et al., 2008).***

Symptoms of venous hypertension were recognized in its complete clinical form within a decade of the initial reports advocating arterio-venous access for hemodialysis. The most common manifestation of venous hypertension is regional edema, although other typical signs and symptoms such as pigmentation, induration, dermatosclerosis, and ulceration may be produced ***(Frank et al., 2008).***

With the presence of central venous stenosis and ipsilateral dialysis access creation, the patient may remain asymptomatic owing to good collateral development, the access may thrombose owing to poor outflow, or the patient may experience a rapid onset of venous hypertension, with arm swelling and pain. The arm swelling can lead to cyanosis and even ulcerations in extreme cases ***(Dosluoglu et al., 2010).***

The NKF-KDOQI (The National Kidney Foundation Kidney Disease Outcomes Quality Initiative) guidelines recommend prompt treatment of central vein occlusions in haemodialysis patients when suspected preferably by endovascular minimally invasive techniques ***(Dosluoglu et al., 2010).***

As regard central venous stenosis/obstruction management, angioplasty alone is most appropriate for subclavian sites due to the potential for extrinsic compression. Angioplasty with or without stent is preferable as initial therapy while surgical bypass is more durable but incurs considerably greater risk ***(Frank et al., 2008).***

Localized venous insufficiency from retrograde venous flow patterns may be resolved either by ligation of veins with retrograde flow or correction of localized outflow obstruction ***(Frank et al., 2008).***

To address outflow obstruction leading to venous hypertension after an access has been placed, there are a variety of potentially feasible options, including access sacrifice, endovascular treatments to recanalize the occluded central vein, and venovenous bypass. Ligation of the access, though successful at relieving symptoms, results in loss of the limb for dialysis access ***(Sidawy et al., 2002).***

Aim of the Work

The first aim of this work is to detect the prevalence of venous hypertension among population on haemodialysis patients in Ain Shams University Hospitals and its relation to central venous catheterization.

The second aim of this work is to evaluate the value of early detection of patients with venous hypertension on the short term outcome, to suggest a protocol of management of post-access venous hypertension regarding method of investigation and timing of intervention.

Anatomy of Central Veins

Relatively detailed anatomical knowledge of the central veins is a prerequisite for safe placement of central venous catheter, and to enable identification of abnormalities when they occur. A description of the normal anatomy of the central veins follows. The anatomy of the internal jugular, subclavian, axillary, femoral, and other routes of access to the central veins are well described elsewhere. Such anatomy is complicated and variable. We provide a schematic overview in the form of a roadmap in Figure 2. This shows the major vessels and branches, and some more common variants. Such simplified images may be helpful when performing procedures or reviewing images. We also show volume rendered reformatted computed tomography (CT) anatomy images later in the text showing normal and variant anatomy ***(Ellis et al., 2004).***

Venous walls are relatively thin and fragile in comparison with arterial walls, rendering them at greater risk from iatrogenic injury. Structurally, they are composed of three layers: an inner endothelial layer (tunica intima), a middle muscular layer (tunica media), and an outer connective tissue layer (tunica adventitia) (Fig. 1) ***(Gibson et al., 2013).***

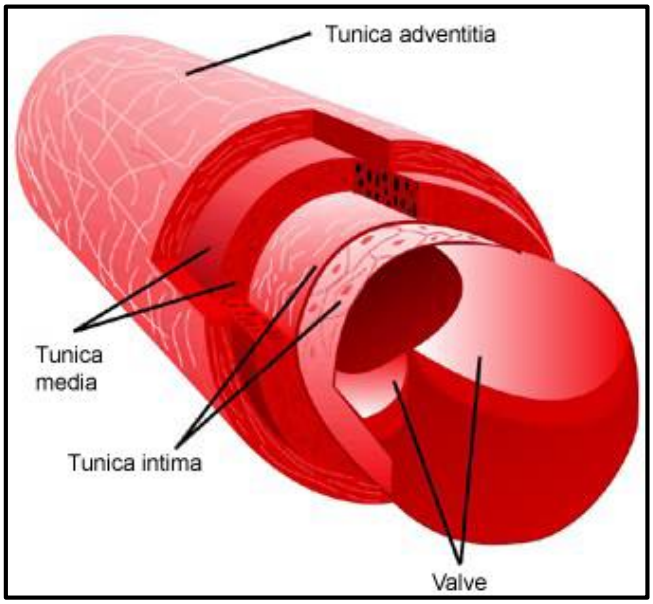

Figure (1): Venous wall structure
(http://www.quidditymedia.co.uk/access/anatomy.html)

These layers are not distinct in all veins, but relative to arteries, there is a larger proportion of the outer connective tissue layer and a smaller proportion of the middle muscular layer. This allows the venous system to be distensible and compliant and act as a blood reservoir, but is the reason for their relative fragility. The longitudinal organization of the layers means that tears in the vein walls tend to extend along the long axis, causing larger defects with the potential for serious bleeding ***(Gibson et al., 2013).***

Vein walls are composed of three layers the intima, media, and adventitia. The intima is a single layer of cells resting on a thin layer of connective tissue. Valves are lined on both sides with a layer of intima over a thin connective

tissue skeleton. Adjacent to the intima is the internal elastic lamina, which is more developed in larger veins and may be absent in small veins. The media consists of smooth muscle cells and connective tissue such as collagen. In the great saphenous vein it is thick and has a great capacity for muscular contraction. This conveys protection from dilatation and varicosity formation ***(Shenoy, 2009).***

In contrast, tributaries of the great saphenous vein have little media and are prone to the formation of varicosities. In the deep system, the content of collagen also varies greatly; calf veins have a large amount, which provides great wall strength and resistance to varicosity formation. The central veins have fewer smooth muscle cells and an increasing amount of connective tissue. The adventitia is not well differentiated from the media and contains loose connective tissue, vasa vasorum, and adrenergic nerve fibers ***(Shenoy, 2009).***

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 internal jugular and subclavian veins 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 Superior vena cava (SVC). It lies anteriorly and to the right of the brachiocephalic artery ***(Stonelake et al., 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 ***(Stonelake et al., 2006).***

The left brachiocephalic vein, 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 ***(Stonelake et al., 2006).***

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 internal jugular and subclavian veins routes ***(Stonelake et al., 2006).***

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 right atrium. This is important to avoid acute angulation against the wall of the SVC or right atrium, with the attendant risks of thrombosis, catheter failure, or perforation. Typically, catheter tips need to lie at the caval/atrial junction or upper right atrium after left-sided central venous catheter placements. 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 et al., 2006).***

Superior vena cava

The SVC drains venous blood from the upper half of the body (Fig. 2). 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 ***(Albrecht et al., 2004).***

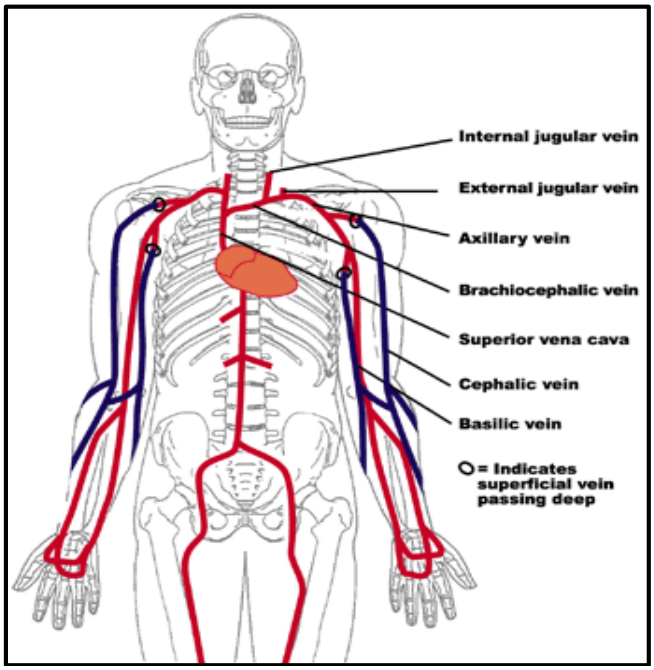

Figure (2): Veins of the thorax
(http://www.quidditymedia.co.uk/access/anatomy.html) Vein wall anatomy

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. 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 ***(Albrecht et al., 2004).***

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 central venous catheter tip outside of the pericardium, therefore minimizing the small but serious risk of cardiac tamponade if the tip perforates the vessel wall. The SVC lies in close anatomical proximity to the mediastinal pleura (Fig. 3) in the upper thorax ***(Albrecht et al., 2004).***

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 ***(Albrecht et al., 2004).***

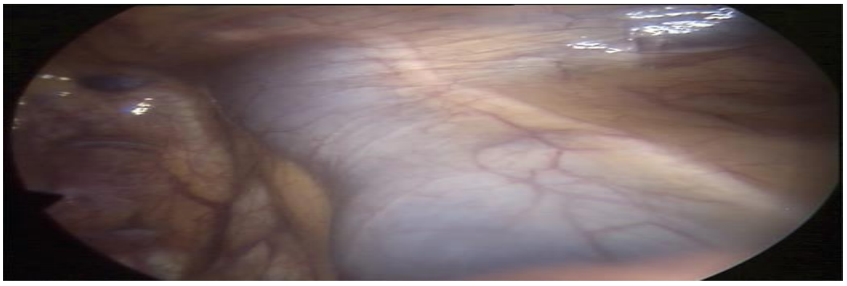

Figure (1): Right-sided thoracoscopic image of the superior vena cava. Note the close anatomical proximity of the vein wall to the pleura, with the vein bulging laterally into the pleural space. A tear of the vein at this site risks massive bleeding into the low pressure pleural space ***(Albrecht et al., 2004).***

Azygous veins

The azygous and hemiazygous venous systems drain the back, thoracic, and abdominal walls. They exhibit much variation. The azygous vein usually arises from the posterior aspect of the inferior vena cava (IVC) at the level of the first or second lumbar vertebra, and connects the IVC to the SVC. It enters the thorax through the aortic hiatus in the diaphragm and ascends in the posterior mediastinum, passing close to the right sides of the bodies of the inferior eight thoracic vertebrae ***(Gibson et al., 2013).***

It arches over the superior aspect of the root of the lung to enter the posterior aspect of the SVC just before it pierces the pericardium. It bulges into the pleural space or may even lie free within the pleural space. The azygous venous system offers an alternative means of venous drainage from the lower body (thoracic, abdominal, and back regions), when there is obstruction of the IVC, and can offer a route of access for the catheter to enter the SVC. The smaller hemiazygous system provides venous drainage for the left chest and upper abdomen and anastomoses with the azygous system ***(Gibson et al., 2013).***

Inferior vena cava

The IVC drains blood from the lower half of the body. It is formed from the junction of the common iliac veins. It is

about 2.5 cm wide and ascends along the anterior of the vertebral column to the right of the aorta. It then perforates the diaphragm and continues cranially and medially for about 2.5 cm. Here, it pierces the fibrous pericardium and opens into the lower part of the RA. There are no functional valves in the IVC ***(Gibson et al., 2013).***

Venous return to the upper extremity is provided by two sets of veins namely the superficial and the deep veins. The main superficial veins are superficial to the deep fascia and are often located at or below the investing layer of superficial fascia in the subcutaneous tissue (Fig. 4). Deep veins are situated deep to the deep fascia and often accompany the artery and the nerves supplying the limb forming a neurovascular bundle ***(Shenoy, 2009).***

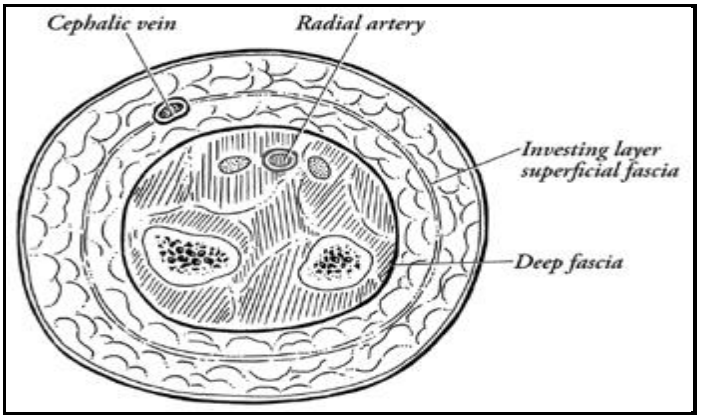

Figure (2): Anatomic location of superficial and deep veins ***(Shenoy, 2009).***

Small superficial veins that drain blood into the named main superficial veins are referred to as venous tributaries.

Blood flows from superficial veins into the deep veins. This unidirectional flow is aided by the valves within the veins. The major named superficial veins join the deep veins at fairly constant anatomical locations, for example, the cephalic vein joins the axillary vein in the infra clavicular fossa ***(Shenoy, 2009).***

In addition to this direct drainage of the named superficial vein into the deep veins, there are several small veins (vein branches) that periodically arise from the superficial venous system and join the deep venous system. These are called the perforating veins (they perforate or travel through a defect in the deep fascia). While some of the perforators can be found at constant locations (for example, the perforating vein at the cubital fossa), the locations of many perforators are inconsistent ***(Shenoy, 2009).***

Venous blood flow is a passive flow (not supported by a smooth muscle pump such as the heart). It is assisted to some extent by the peripheral muscle contraction and smooth muscle in the vein wall. The direction of the flow is maintained by the valves within the vein which prevents flow reversal. Location of the valves within the veins is highly variable. They are commonly found in the deep veins near the entry of a tributary. They are also in the tributary close to the site of its entry into the deep vein (Fig. 5) ***(Wellen et al., 2009).***