Review Article

The jugular-subclavian junction and venous drainage of the brain

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Abstract: Lung cancers and mediastinal masses can invade the veins in the upper mediastinum and neck. It can be challenging to determine management options and the feasibility of resection particularly when tumors involve the major venous junctions. Furthermore, impaired flow in these veins can have devastating complications such as Paget-Schroetter syndrome, which describes a constellation of symptoms (arm swelling, cyanosis, pain) due to stenosis of the subclavian vein. This section will provide an overview of venous drainage of the brain, which can be divided into two major systems—superficial medullary venous system and deep medullary venous system. The anatomy and function of the great veins of the neck and upper mediastinum, including the internal jugular vein, subclavian vein, and brachiocephalic (i.e., innominate) vein will be described. Also discussed will be principles of ligation of the venous structures and the importance of keeping the venous junctions intact to facilitate and maximize the development of collateral flow. This section will also discuss ensuing complications when blood flow is impaired, such as development of upper extremity deep venous thrombosis and cerebral venous thrombosis (CVT). CVT can result in a stroke and is an umbrella term that refers to problems in cerebral venous outflow due to numerous etiologies.

Keywords: Jugular-subclavian junction; cerebral venous thrombosis (CVT); venous ligation; mediastinal veins; innominate vein

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Venous drainage of the brain

The meninges are composed of three layers: pia mater (inner layer), arachnoid (the middle layer that's filled with fluid and cushions the brain), and the dura mater (the tough outer layer). The cerebral veins are in the subarachnoid space and lack muscular tissue and valves (1). Blood from these cerebral veins along with cerebrospinal fluid (CSF) drains into the dural venous sinuses—essentially, venous channels—located in the dura mater. Depending on the location (anterior, midline, posterior or lateral), this fluid then drains into one of the larger venous sinuses (1,2).

This drainage pathway can be divided into two major systems: superficial medullary venous system and deep medullary venous system. The superficial system is composed of sagittal sinuses and cortical veins, which drain the surfaces of the cerebral hemispheres. The deep system drains the deeper cortical veins and comprises the lateral, sigmoid, and straight sinus (1).

This anatomy is important to understand because thrombosis of the major veins in the neck, which can be caused by underlying lung malignancy, can lead to cerebral venous thrombosis (CVT) and potentially result in a stroke. Symptoms are varied based on the area of venous infarct. This will be discussed later.

Anatomy and the jugular-subclavian junction

Lung cancers can invade or abut the thoracic inlet, affecting the adjacent structures. These include the major veins such as internal jugular veins, subclavian veins, and brachiocephalic, i.e., innominate, veins. The internal jugular veins drain the blood from the cerebrum, skull, neck, and superficial parts of the face. These veins originate in the posterior compartment of the jugular foramen at the base of the skull, travel caudally in the carotid sheath, and join with the subclavian veins at the base of the neck (venous junction) to form the brachiocephalic veins. The internal jugular veins contain a valve just proximal to their connection with the subclavian veins, which prevents retrograde flow of venous blood (3). The internal jugular veins are important for drainage primarily in the supine position. In an erect position, they contribute to approximately 10% of the outflow and rather valveless channels become more important for outflow. These channels include the vertebral veins, internal vertebral venous plexus and external vertebral venous plexus. However, if there is an obstruction in the cerebrospinal system, the internal jugular veins provide collateral circulation and can thus become important for outflow in the erect position as well (2,4).

The subclavian veins are valveless and are a continuation of the axillary veins at the outer border of the first rib. They drain the upper extremities. Impairment in flow due to stenosis or thrombosis can result in arm swelling, cyanosis, and pain (Paget-Schroetter syndrome) (5).

Blood from the brachiocephalic veins drains into the superior vena cava, emptying into the right atrium. These are valveless veins that also receive drainage from the left and right inferior thyroid veins, left superior intercostal vein, and left and right internal thoracic veins (6).

Management of tumors involving the jugular-subclavian venous junction can be challenging. Tumor involvement of this junction can be seen in thymic, head and neck, and lung cancers. This is often regarded as an ominous sign because there is a proclivity for distant vascular metastasis. Management of many of these patients frequently involves chemoradiation with the hope of following with surgery. Surgical management may require resection of the involved subclavian vein and brachiocephalic vein (7).

Ligation of veins and development of collateral flow

Venous obstruction can be attributed to a variety of factors including thrombosis, extrinsic mass effect (e.g., from a tumor) or stenosis (e.g., from prolonged central catheter placement). Ligation of the veins may be necessary during surgical resection. Acute symptoms of central vein obstruction may develop, such as unilateral arm swelling. However, these can be managed conservatively (e.g., arm elevation) and often subside with adequate development of collateral flow over 3 weeks. Collateral circulation can be assessed via duplex ultrasound of the neck (8). If revascularization is needed, it can be done via primary anastomosis, interposition graft or transposition of another vessel (e.g., using the ipsilateral internal jugular vein to reconstruct a resected subclavian vein) (9,10).

Ligation of the left innominate (brachiocephalic) vein is well tolerated and is often necessary during standard surgical approaches. For instance, given the anterior location of the left innominate vein in relation to the aortic arch, it can be safely divided at its midpoint during a sternotomy to gain better exposure of the aortic arch (6). Collateral pathways develop through the azygous system and the internal thoracic vein (11). In general, the surgeon should attempt to keep the innominate-caval and the innominate-jugular subclavian junction intact to maximize collaterals. The subclavian vein can often be safely ligated as well, with subsequent collateralization to the ipsilateral shoulder via the subscapular and suprascapular veins and to the neck via the anterior and posterior ipsilateral neck veins (11). Again, venous junctions should be preserved when possible.

Although uncommon, the first sign of underlying malignancy can be internal jugular vein thrombosis, which may necessitate ligation of the vein (12-14). Complications associated with internal jugular vein ligation are rare and can include stroke, pulmonary embolism, pseudotumor cerebri or transverse sinus thrombosis. Compared to unilateral vein ligation, bilateral ligation, performed during bilateral radical neck dissection for example, is associated with increased morbidity and mortality, partly because of the ensuing cerebral venous congestion and increased intracranial pressure. Therefore, a two-step staged operation with an interval of 2 weeks is preferred to allow for adequate collaterals to develop in the interim (15,16). If simultaneous bilateral ligation must be performed, one of the internal jugular veins should be reconstructed via an anastomosis between the internal jugular vein and external jugular vein or via an interposition graft (17).

Investigation of collateral flow in patients with multiple sclerosis, who often have stenosis of the internal jugular vein, and in patients who have undergone bilateral jugular

vein ligation reveals re-routing of blood flow from the jugular veins to the vertebral venous system and superficial veins (i.e., external jugular veins, anterior jugular veins, and posterior external jugular veins) (15,18).

Upper extremity deep vein thrombosis (UEDVT) and risk of stroke

The pathophysiology of deep vein thrombosis is well described by Virchow's triad: vessel wall damage, stasis of flow, and hypercoagulable stage. UEDVT is an uncommon phenomenon, accounting for only 10% of all cases (19). In the last several years, there has been an increased effort to investigate the etiologies, complications, and treatment of UEDVT, especially internal jugular vein thrombosis.

In general, the etiology of UEDVT can be categorized as primary or secondary. Primary causes include idiopathic, occult malignancies or effort thrombosis (Paget-Schroetter syndrome). Secondary causes are more common and include multiple factors such as catheterization, surgery, otolaryngological infections (e.g., Lemierre's syndrome), trauma, implantable cardiac rhythm devices, and malignancies (Trousseau's syndrome). The two most common malignancies contributing to UEDVT include ovarian carcinoma and lung adenocarcinoma (19).

Diagnosis can be challenging because these patients can present with a myriad of non-specific symptoms, especially those with internal jugular vein thrombosis. Presentations for UEDVT include unilateral arm erythema, edema, dyspnea, and low-grade fevers. Similarly, internal jugular vein thrombosis can present as sudden onset painful neck swelling and erythema, face swelling, and fevers. Less commonly, it can manifest as a palpable cord beneath the sternocleidomastoid muscle, superior vena cava syndrome, chylothorax, and jugular foramen syndrome (20). Jugular foramen syndrome (Vernet syndrome) is characterized by paralysis of the cranial nerves which traverse the jugular foramen (IX, X, XI). The most common presenting symptoms are hoarseness or dysphagia. Other symptoms include loss of gag reflex, drooping of the shoulder and loss of sensation to the posterior ipsilateral aspect of the tongue. Patients may also present with headaches or papilledema due to venous congestion caused by obstruction of the venous sinuses and veins that also traverse the jugular foramen. As alluded to previously, it is important to keep underlying malignancy in mind in patients with UEDVT, especially when diagnosed with internal jugular vein thrombosis.

Ultrasound is the preferred diagnostic modality, but computed tomography (CT), magnetic resonance imaging (MRI) or direct venography can also be used when the diagnosis is uncertain. Prognosis of UEDVT varies depending on the vessel(s) involved and the etiology. Cancer-induced thrombosis compared to other etiologies has greater morbidity with increased risk of complications including post-thrombotic syndrome, superior vena cava syndrome, and septic thrombophlebitis. Internal jugular vein thrombosis especially can be life-threatening due to the greater risk of pulmonary embolism. This risk increases if other veins are also thrombosed. The rates of pulmonary embolism can vary from 0.5% for isolated internal jugular vein thrombosis to 2.4% if subclavian and axillary veins are thrombosed as well (19). Anticoagulation is the mainstay of treatment with low molecular weight heparin often followed by warfarin. Other interventions may also be needed depending on the etiology, including antibiotics, removal of central venous catheter or drainage of neck abscess.

Propagation of an internal jugular vein thrombus can also lead to the development of CVT, which can then result in a stroke. CVT is an umbrella term that describes problems with cerebral venous outflow due to numerous etiologies, and it comprises approximately 0.5% of all strokes. It has been linked to pro-thrombotic states and is more commonly associated with pregnancy and contraceptive use. It is a rare disorder seen primarily in young adults and females and describes the thrombosis of cerebral veins and dural sinuses that leads to increased venous pressure and obstruction of the cerebral sinuses. This, in turn, results in decreased cerebral perfusion and increased intracranial pressures. Symptoms are varied and can range from focal headaches to seizures to ischemic/hemorrhagic stroke. MRI/magnetic resonance venography (MRV) is the gold standard for diagnosis, but CT venography and non-contrast CT can also be used (21). Prompt recognition and management of life-threatening symptoms (i.e., seizures, signs of intracranial hypertension) is critical.

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