Internal jugular vein thrombosis is an extremely rare vascular disease. It is usually secondary to intravenous drug abuse, prolonged central venous catheterization or deep head-neck infections or trauma. Associated malignancies are uncommon and not well documented in the etiology of IJV thrombosis. A 50-year-old female presented with complaints of neck swelling on left side for past two weeks and with no ear or nose complaints. On examination of neck presented swelling along the anterior border of the left sternocleidomastoid muscle. An ultrasound scan of the neck neck revealed left IJV thrombosis. The patient was referred to vascular surgeon for further treatment.

Keywords: Thrombosis, Internal jugular vein, pseudo pharyngeal abscess, neck abscess, septicemia

Introduction

Internal Jugular Vein (IJV) thrombosis is a rare but potentially fatal condition. The cause of IJV thrombosis is the use of the IJV for venous access and central venous catheterization. Other causes include oropharyngeal infection (Lemierre Syndrome), deep neck space infection and trauma [1]. It can also occur as a complication of head and neck malignancy, and rarely can be the first manifestation of the disease. Diagnosis of IJV thrombosis requires a high degree of clinical suspicion, and the use of appropriate investigations. Potentially serious complications can ensue and prompt appropriate management is imperative [2]. The incidence of IJV thrombosis is not known exactly but the incidence appears to be increasing every year. The complications associated with IJV thrombosis are potentially lethal and it is therefore important to recognize and treat this condition early. Here, we are presenting a case of left internal jugular vein thrombosis in a middle aged woman.

Case presentation

A 50-year middle aged lady Mrs XX presented to outpatient department of surgery, of our hospital with the presence of neck swelling on left side in short duration of two weeks which is painful, and not much of systemic symptoms. History revealed no significant symptoms of severe pyrexia or sore throat. There was no history suggestive of any ear or nose disease and no history suggestive of intravenous drug abuse or trauma. On physical examination the swelling was found to be soft, mobile, semi fluctuant and measuring around 7c.m.Swelling was superficial
to strap muscles started off in submandibular region and became bigger. Tongue dental hygiene normal. Other group of cervical lymph nodes like anterior posterior supra clavicular not clinically palpable.

Figure 1: Swelling and neck abscess in pharyngeal region.

Physical examination showed low grade fever (99°F), pulse rate 100/min, blood pressure 130/90, respiratory rate 22/min, with prominent accessory muscles of respiration. The patient had no pallor, clubbing or peripheral edema. There was no cervical, axillary or inguinal lymphadenopathy. There was no purulent discharge from ear or nose. No h/o of any previous surgery/scar, weight loss. Mucosal surfaces of the oral cavity and otoscopic examination of the both ears normal. Neck swelling is not pulsatile and no transmitted pulsations or bruit on auscultation. Examination of head and neck like abnormalities of thyroid gland was not found and ENT examination laryngoscopy, pharyngoscopy, general examination revealed no significant NAD.

Features of the neck swelling was severely tender, possibility of the abscess was thought as swelling over the fluctuant in the beginning and it does not seem to transmit any pulsation from the nearby vascular structure. Based on clinical presentation of the neck abscess, lateral side of the neck (submandibular region) was further evaluated. Ultrasound of neck revealed – ill defined collection of fluid in left submandibular region and in subcutaneous plane. Clinically it was considered to be submandibular in the sub cutaneous plane abscess. On scan finding it is, hyperchoic thrombus in left internal jugular vein completely obstructing the lumen. Proximal or distal flow is so minimal. Laboratory findings revealed T.W.B.C count 8.7x 10³/mm³, T.R.B.C count 3.8x 10⁵, hemoglobin 10.0gms, AEC 3.8 million/mm³ and a platelet count of 3.2 lakh/mm³ with raised ESR (22 mm/hour). Biochemical investigations include random blood sugar (RBS) – 90mg/dl, serum urea – 29 mg/dl, serum creatinine – 0.8 mg/dl. Routine examination showed urine sugar – Nil, urine albumin – Nil, urine microscopy revealed so significant abnormality.

Figure 2: swelling in the left side of neck region.

She does not have any history of disturbances in salivary, mastication, notable primary symptoms in neck or ear. She is not known diabetic or HTN. Patients other CVD conditions observation revealed - no dysnea, no obstructive symptoms or compressive symptoms related to neck or dilated veins, significant lymphadenopathy and no ear symptoms. Her respiratory system was clinically normal.

Case discussion

The many causes of internal jugular vein thrombosis include complication of surgical
procedures, presence of an indwelling venous catheter, tumor invasion, hypercoagulability, caudal extension of sigmoid sinus thrombosis, compression from adjacent tumor or nodes, reaction to an adjacent infectious process, and direct venous injection [3].

Diagnosing internal jugular vein thrombosis requires a high degree of suspicion. In the differential diagnosis of a painful neck swelling or neck mass, deep neck infection, cellulitis, painful lymphadenopathy, and head and neck tumor with necrosis are commonly encountered causes [4]. However, internal jugular vein thrombosis should be included in any list of causes, particularly where there is a history of previous head and neck infection, venous catheterization, or drug use.

The present patient displayed sudden onset of painful swelling of the left neck with local heat. The differential diagnosis for this patient included local infection, cellulitis, and internal jugular vein thrombosis because of its occurrence over several hours to days. Furthermore, no fever or signs of systemic inflammatory response syndrome were noted during this period of time, thus making internal jugular vein thrombosis the most likely diagnosis. However, from the history of this patient, it was clear that she was not an IV drug user and had not received central venous catheterization before admission. Consequently, the etiology of internal jugular vein thrombosis merited further investigation [4]. Suspected internal jugular vein thrombosis can be rapidly diagnosed using duplex ultrasonography. Ultrasound has the key advantage of providing a bedside diagnosis, with high sensitivity and specificity, and may achieve superior resolution to CT in superficial areas. In the present patient, total occlusion of the left internal jugular vein was clearly identified using ultrasound, which also provided a convenient method for follow-up under anticoagulation therapy.

A CT scan and magnetic resonance image (MRI) can also be used for diagnosis. These methods are especially useful for excluding a local mass effect from an unsuspected malignancy. Furthermore, these methods can help identify the possible cause of internal jugular vein thrombosis. The CT of the present patient revealed abnormal soft tissue infiltration over the left lung apex which may have compressed the lower part of the internal jugular vein. One aspect of the pathophysiology of thrombosis in malignancy is venous stasis or abnormality of blood flow. Immobilization or bed rest or vascular compression by a tumor mass may cause venous stasis, which can delay the clearance of activated coagulation factors and cause venous valve damage due to hypoxia [5].

The retropharyngeal space lies in the midline of the neck between the pharynx (visceral space) and the prevertebral space. The alar fascia, which is the anterior and lateral split of the deep layer of deep cervical fascia, is the anterior border of the danger space and contributes to the combined facial layers making up the carotid sheath. Because the completeness of the facial coverings and thus the relative communication of these spaces with each other and the parapharyngeal space are debatable [6], the ability of free fluid to pass among the spaces either directly or along vascular or lymphatic sheaths is debatable. Whether or not fluid (or pus) can freely move among these spaces is not the most important issue in this setting, as we do not believe this pathologic process in the retropharyngeal space is created by free-flowing fluid.

Lemierre syndrome, internal jugular vein thrombosis with anaerobic septicemia, causes metastatic lung abscesses, septic arthritis to osteomyelitis, skin and soft tissue abscesses, liver and splenic abscesses, cerebral abscess formation, cranial nerve paralysis, and so on, and is potentially fatal [7]. Although the cause of the syndrome is unknown, it is thought that involvement of the internal jugular vein occurs by direct extension through the facial plane between the tonsil and the parapharyngeal space or by hematogenous or lymphatic spread from peritonsiller vessels, and then septic emboli can arise and spread to distant sites and organs [8]. After the advent of antibiotic therapy, as penicillin was frequently used for patients with
pharyngitis, cases of Lemierre syndrome gradually decreased and became known as “the forgotten disease.” However, recently, cases of the Lemierre syndrome have increased, and it is thought that the reason is the decreased use of antibiotics for upper respiratory infections [9]. Prompt diagnosis and appropriate therapy, intravenous antibiotics with good anaerobic coverage with or without anticoagulation, and supportive care are needed. It is difficult to diagnose Lemierre syndrome based only on the clinical symptoms because the clinical course of Lemierre syndrome is variable and complications can involve almost any symptom in the body. Radiographic findings, particularly contrast-enhanced CT, are characteristic and facilitate the diagnosis of Lemierre syndrome[10].

Conclusion

Cases of true isolated spontaneous IJV thrombosis are very rare. Unfortunately there is little work published regarding internal jugular vein thrombosis. To help and guide the clinician when managing the disease the present case was reported. The evidence of treatment is diverse and drawn from experiences from individual clinicians. Advice from other clinicians should be sought where necessary to obtain a consensus view on the management of individual cases. In the present case because of lack of advanced technology and facilities for further treatment patient was referred to a vascular surgeon of other super specialty hospital.

References