Introduction
Epidermal venous engorgement due to epidural collaterals can occur due to various causes such as inferior Vena Cava agenesis [1,2], abdominal malignancy [3,4], inferior vena cava occlusion/thrombosis secondary to contraceptive use [3,5,2], pregnancy [3,6] and protein C deficiency [3]. Patients with such epidermal venous plexus engorgement usually present with low backache, radiculopathy or neuropathic claudication [4,8,9]. In rare circumstances patient develop neurological deficits or cauda equina syndrome [10]. Bladder & bowel involvement and weakness of lower limbs are the most serious consequences of such epidural collaterals. We present here a case of a 30 year old female who presented with abdominal pain, the causes of which was found to be IVC thrombosis. Patients also had lower backache and radiculopathy which on MRI of spine was found to be the result of extensive epidural collaterals leading to thecal sac and foraminal compression, the cause of which was inferior vena cava thrombosis in this patient.

Case report
A newly married female was admitted to our institution with the complaints of severe pain abdomen for 10 days which aggravated further for last 2 days. Patient was worked up in medicine department and had Hb 9 gm , TLC -7200/cumm,normal LFT&RFT's and Total bilirubin -1.0 mg/dl. Patient was referred to our department for Ultrasonography and CECT of the whole abdomen, on which she was investigated by the neurologist and was found to have radiculopathy at L5 level. The neurologist referred the patient for MRI spine (Image 2).

As the patient had severe backache radiating to right leg, she was investigated by the neurologist and was found to have radiculopathy at L5 level. The neurologist referred the patient for MRI spine (Image 2).

MR imaging showed the presence of the tortuous dark structures in the anterior epidural space as well as in the bilateral neural foramina in the dorsolumbar region. The signal loss of these structures lead us to think of the vascular nature of these structures. Prominent collaterals were also noted in paravertebral location. Inferior vena cava and proximal portions of bilateral common iliac veins were attenuated in caliber and were thick walled with loss of flow voids suggestive of chronic thrombosis. In addition there was excess amount of epidural fat at L4-S1 level i.e epidural lipomatosis. The epidural and foraminal venous collaterals along with epidural lipomatosis was causing compression of thecal sac and nerve roots, being most marked at L5-S1 level. With these findings the cause of backache in this patient was found to be epidural varicosis secondary to chronic inferior vena cava thrombosis leading to thecal sac and foraminal stenosis. The patient was continued on anticoagulation therapy. The inferior vena cava stenting was also recommended to the patient in the event of non-relief of her symptoms.

Discussion
The commonest causes of radiculopathy is disc pathology [11]. Chronic Inferior vena cava thrombosis as a possible cause of low backache and radiculopathy is very unexpected like was in our case. Vascular malformations are rare causes in the spectrum of low backache [12]. Still rare is the venous etiology for the backache. The epidural collaterals itself mimic the protruded/ extruded disc [11]. Till date small number of cases had been reported in the literature who had neurologological symptoms in the setting of Inferior vena cava involvement, the likely etiologies in these patients were found to be Inferior vena cava agenesis [1,2]; and thrombosis secondary to oral contraceptive use [3,5,2], pregnancy [3,6], Behcet's disease [3], malignancy [3,4], Budd-Chiari syndrome [7], and protein C deficiency [3]. The patient should be evaluated for potential etiologies of IVC occlusion, including structural abnormalities, portal hypertension, oral contraceptive use, thrombophilias, and
malignancies.

Congenital anomalies of the inferior vena cava and its tributaries had also been associated with epidural venous plexus engorgement(1,2). These include agenesis of infrarenal IVC, azygous continuation of IVC, left sided IVC, double IVC etc. These patients may present with venous insufficiency or idiopathic deep venous thrombosis.

In the setting of inferior vena cava occlusion, venous drainage is redirected into the azygous system from the lower extremities, as well as the abdomen and pelvis, by way of paravertebral collaterals and the vertebral plexus. The vertebral plexus becomes engorged. This elevated venous pressure is transmitted into the valvless epidural and radicular veins may result in thecal sac and neural foraminal compression resulting in neurological symptoms. In rare circumstances, venous congestion may also cause myelopathy (13).

Epidural venous congestion is more common in lumbar region, however cervical root compression due to cervical epidural venous plexus has also been reported (14).

Anticoagulation has been proposed as symptomatic treatment in patients with chronic Inferior vena cava thrombosis. In the literature, the alternative methods to achieve thrombolysis as well as surgical intervention have been used. The thrombolysis was found to be unsatisfactory hence it is rarely used [15]. The need of surgical treatment of an epidural varix is required only if there are intractable neurological symptoms. Surgical thermoacoagulation of the venous plexus, surgical compression of the venous plexus with a resorbable gelatin sponge, interventional techniques have all been used with good symptomatic relief to the patients. However severe epidural collaterals due to Inferior vena cava thrombosis should not be undertaken for surgery due to surgical risks (16).

Conclusions

The epidural venous collaterals may cause neurological symptoms mimicking lumbar disc herniations or spinal canal stenosis. The findings of epidural collaterals on MRI should raise the possibility of chronic inferior vena cava occlusion/thrombosis. Vis-a-versa in patient with neurological symptoms in the setting of inferior vena cava thrombosis, the possibility of epidural varicosis should be ruled out. Knowledge of this condition may help in early recognition and clinical management of the patients so that potentially treatable etiologies like IVC thrombosis be identified at the earliest to prevent permanent neurological deficits and patient be offered the best treatment option.

Authors contribution

NS, CG, KS, AA participated in acquisition of data, conception and design of manuscript. All authors participated for literature search and approved the final manuscript for the publication.

Conflict of interest

All authors declare that they don’t have any conflict of interest.

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