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A Case of Superior Venacaval (Svc) Syndrome

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CLINICAL HISTORY:

An 85year old male patient who is a known case of type 2 diabetes and hypertension and COPD on medication came with the complaints of Easy fatigability since 2 months. Cough since 30 days , Reduced appetite since 30 days. Giddiness since 7 days. Facial puffiness since 1 day.

The patient developed cough associated with expectoration which was whitish, scanty in amount, and non-blood tinged, and non-foul smelling since 30 days. He developed increased breathlessness since 20 days.

No history of fever, chest pain/palpitations. No history of orthopnea/ PND. No history of syncope, pain abdomen, decreased urinary output.No history of pedal edema. No h/o exposure to covid patient or trauma

Past history

No history of thyroid disorders, cardiac disorders

EXAMINATION AND INVESTIGATIONS:

GENERAL PHYSICAL EXAMINATION:

Afebrile- 97.6°F

Bilateral supraclavicular lymph nodes are palpable, hard in consistency, discrete, adherent to deeper structures, largest measuring 4x4cms.

Pallor and icterus present

Puffiness of face was present

JVP- raised; non-pulsatile neck veins

RS:

On percussion dullness present over areas on the right side

Right-sided air entry is reduced with crepts present over the right infrascapular, infra axillary, and interscapular area.

Bilateral ronchi+

PER ABDOMEN:

All findings within normal limits

CVS: S1, S2 heard. No murmurs.

CNS: Intact.

BLOOD INVESTIGATIONS:

Hb:14.5gms/dl

Total Count:

15,020/ml

N:84.4%, L:11.2%

RBC:4.75 million/ml

MCV:87.8fl

MCH:30.5pg

MCHC:34.8g/dl

RDW-CV:12.8%

PLATELET:4.95lakhs/ml

PERIPHERAL BLOOD SMEAR:

Red blood cells are normocytic normochromic. White blood cells are increased in number with an increase in neutrophils. Platelets are increased in number.

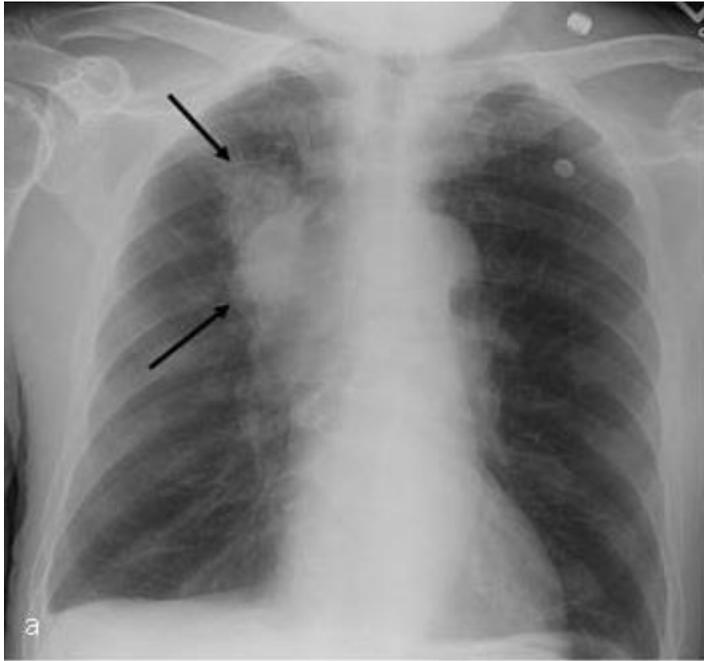
Impression: Neutrophilic leukocytosis with thrombocytosis

URINE MICROSCOPY:

Plenty of pus cells

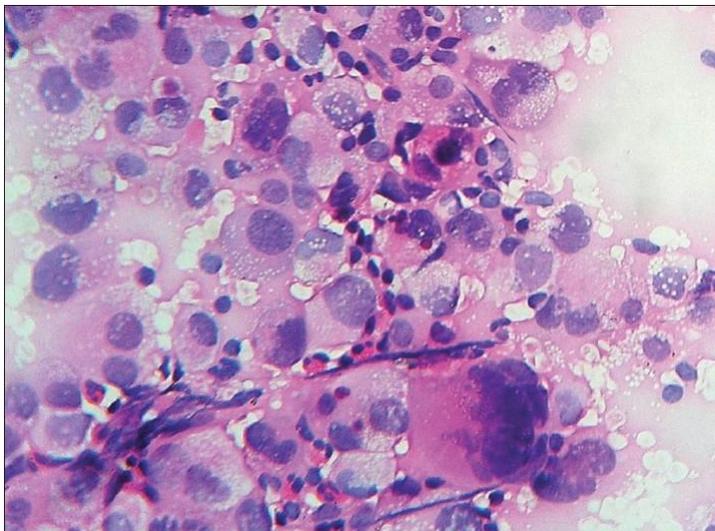
GRAM STAIN OF SPUTUM –Gram-positive cocci in pairs and chains

CHEST XRAY



Chest x-ray showing right-sided homogenous opacity suggestive of infiltrating mediastinal mass over the right side of the chest.

KOH- preparation of sputum: Budding yeast seen.



FNAC of right supraclavicular lymph node

FINAL DIAGNOSIS:

Bronchogenic Carcinoma with Metastasis presenting with SVC Syndrome

Type 2 Diabetes Mellitus (Newly detected)

Hypertension COPD

DISCUSSION:

Superior Venacaval Syndrome (SVC) is the obstruction of superior vena cava, the most common etiology being malignancy. It is the obstruction of superior vena cava by primary or metastatic cancer (mostly intrathoracic); through invasion, external compression, or thrombosis.

In an article by Straka C et al, it is stated that The Superior vena cava is thin-walled and is opened by relatively low venous pressure, hence making it particularly susceptible to compression by adjacent masses.

The severity and rapidity of onset of symptoms depend on the rate at which the SVC is occluded and if collateral venous drainage to develop in time. In the event of SVC obstruction, venous pressure in collateral vessels increases and, over time, a collateral blood-flow network develops. Different vessels may enlarge in response such as the azygos, the hemiazygos, intercostal, mediastinal, paravertebral, thoracoepigastric, internal mammary, anterior chest wall veins, and thoracoacromioclavicular veins. The azygos vein may connect the SVC and the inferior vena cava (IVC) directly, so SVC obstructions that occur below the insertion of the azygos vein result in more severe symptoms of SVC Syndrome[1].

Lung cancer, lymphoma, thymic neoplasms, germ cell tumors, mesothelioma, and other solid tumors that cause mediastinal involvement are the commonest[2].

Symptoms can range from interstitial edema of the head and neck which are asymptomatic to life-threatening complications such as airway narrowing from the upper airway or laryngeal edema and cerebral herniation and ischemia secondary to cerebral edema as a result of venous outflow obstruction.

Facial plethora, cyanosis, and arm edema are often observed. A physical examination can present as venous collaterals on the chest and neck. Stridor headaches or confusion is also present[3].

Management of SVC syndrome depends on the presentation and underlying malignancy. Patients presenting with stridor from laryngeal edema and stupor from cerebral edema need emergent management like with radiation and or stent placement. Upfront radiation before histologic diagnosis in stable patients can obscure a pathologic diagnosis and should not be pursued. Steroids have a role in steroid-responsive tumors such as lymphoma and thymoma. It can control edema in case of airway obstruction. They have not been systematically studied for its use in stenting[1].

Chemotherapy is the treatment of choice for patients with cancers such as Small-cell Lung Carcinoma (SCLC), lymphoma, and germ cell tumors.

For SVC Syndrome caused by an infectious etiology, antibiotics should be the first line of treatment. In cases of thrombus-driven SVC Syndrome, Anticoagulants are indicated[1].

Stent placement and radiotherapy may be required with less chemo responsive tumors such as Non-Small Cell Carcinoma(NSCLC)

Surgical repair via bypass grafting with the femoral vein, spiral saphenous vein, Dacron graft, or polytetrafluoroethylene graft can put the SVC obstruction in check. However, this is mostly used for cases where recanalization via endovascular repair is impossible or has failed.

Recently, Stereotactic body radiation therapy (SBRT) has become a well-accepted modality for administering radiation at a variety of anatomical sites. Potential benefits of SBRT include a novel bio-the

the logic rationale for tumor cell kills as well as a decreased number of treatments, resulting in minimized damage to healthy tissue and also decreased treatment cost. It is not an ideal treatment method for a patient who needs

rapid symptom palliation[1]. However Endovascular therapy now acts as a first-line treatment for SVC syndrome[3].

ACKNOWLEDGEMENTS: None

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Image Courtesy: In Seminars in interventional radiology 2017 Dec (Vol. 34, No. 4, p. 398).

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