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The Role of Multislice CT in Evaluation of Superior Vena Cava Syndrome

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The Role of Multislice CT in Evaluation of Superior Vena Cava Syndrome

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Abstract- The superior vena cava (SVC) syndrome is a clinical entity caused by obstruction of the superior vena cava by infiltration, compression or thrombosis. Cancer is the most common underlying cause of superior vena cava obstruction. The incidence of catheter-induced superior vena cava obstruction is rapidly increasing. Fibrosing mediastinitis and Behçet disease are rare causes of SVC syndrome. Clinical presentation of SVC syndrome may include cough, dyspnea, dysphagia, and swelling or discoloration of the neck, face and upper extremities. *Aim of this study* is to evaluate the role of Multislice CT in study of superior vena cava obstruction syndromes and assessment of collateral circulation in different causes of superior vena caval obstruction.

I. Introduction

The superior vena cava (SVC) syndrome is a clinical entity caused by obstruction of the superior vena cava by infiltration, compression or thrombosis. Although clinical symptoms of the disorder were first described in 1757 in a patient with a syphilitic aneurysm of the ascending aorta, vascular causes are now rare and approximately 90% of cases are associated with a cancerous tumor that is compressing the superior vena cava, such as bronchogenic carcinoma including small cell and non-small cell lung carcinoma, Burkitt's lymphoma, lymphoblastic lymphomas, acute lymphoblastic leukemia (rare), and other acute leukemias *(Krimsky et al, 2002)*. Tuberculosis has also been known to cause superior vena cava syndrome (SVCS). SVCS can be caused by invasion or compression by a pathological process or by thrombosis in the vein itself, although this latter is less common (approximately 35% due to the use of intravascular devices) *(1).*

Malignant superior vena cava obstruction In most published studies, cancer is the most common underlying cause of superior vena cava obstruction e.g. lung cancer and lymphoma *(Sakura et al, 2007)*. Most primary malignant tumors of the superior vena cava, such as leiomyosarcoma or angiosarcoma, are un-common. The presence of arterial enhancement of the thrombus is highly suggestive of tumor thrombus *(2).*

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Benign Causes of Superior Vena Cava Obstruction *Iatrogenic superior vena cava obstruction*— the incidence of catheter-induced superior vena cava obstruction is rapidly increasing e.g. large central venous catheters, such as dialysis catheters, Hickman catheters, and parenteral nutrition catheters *(3).* *Fibrosing mediastinitis* is a rare histologically benign disorder caused by proliferation of collagen tissue and fibrosis in the mediastinum *(4).* *Behçet disease* is a rare systemic disease in which superior vena cava stenosis or occlusion may result *(5).*

Clinical presentation of SVC syndrome: SVC syndrome is caused by gradual compression of the SVC, leading to edema and retrograde flow, but it can also be caused more abruptly in thrombotic cases. Symptoms may include cough, dyspnea, dysphagia, and swelling or discoloration of the neck, face and upper extremities. Often, collateral venous circulation causes distension of the superficial veins in the chest wall. Although SVC syndrome is usually a clinical diagnosis, plain radiography, computed tomography (CT) and venography are used for confirmation. (6).

CT Findings: CT can diagnose SVC affection in SVC syndrome and can detect subclinical superior vena cava obstruction in patients who are relatively asymptomatic *(Yu JB et al, 2008).* Regardless of its cause, the CT diagnosis of superior vena cava obstruction includes lack of opacification of the superior vena cava, an intraluminal filling defect or severe narrowing of the superior vena cava, and visualization of collateral vascular channels *(Eren et al, 2006).* MDCT, with its multiplanar and 3D imaging capabilities, allows thorough anatomic delineation of the various collateral pathways diverting the blood from the site of obstruction. Although axial images allow evaluation of potential causes of superior vena cava obstruction, such as a mediastinal mass, MDCT provides information about the level and degree of superior vena cava obstruction, the length of the affected segment, and the presence or absence of intraluminal clot distal to the obstruction, thereby allowing the interventional radiologist to choose the optimal treatment option. *(Plekker et al, 2008).*

II. Aim of the Work

To evaluate the role of Multislice CT in study of superior vena cava obstruction syndromes and

assessment of collateral circulation in different causes of superior vena caval obstruction.

III. Patients and Methods

a) Patients

This study involved 20 patients; 13 males and 7 females, age range 15-69 (average of 39.3years). Cases were referred from the chest department to the radiology department in Kasr Al-Aini for MSCT of the chest.

Twelve patients presented clinically with mediastinal syndrome; of whom six cases were biopsy proved central Bronchogenic carcinoma, five were known cases of Behçet disease and one case with unknown etiology.

Two cases had right upper limb DVT. Two cases had lymphoma and one case was surveyed for chest metastases. One case had fever of unknown origin after chemotherapy. One case with anti-phospholipid antibody syndrome where echo detected calcified right atrial thrombus. One case with bilateral pleural effusion. (See table 1).

Table 1 : Showing patients' presentation

Referred case	Number of patients
Mediastinal syndrome	12
Upper limb DVT	2
Chest assessment in case of lymphoma	2
Chest assessment for metastases	1
Fever of unknown origin after chemotherapy	1
Antiphospholipid antibody syndrome	1
Bilateral pleural effusion	1

b) Methods

All patients were subjected to:

1. Thorough clinical examination with history taking, general and chest examination.
2. Routine laboratory tests mostly complete blood picture, other tests were considered according to case e.g. culture and sensitivity.
3. MSCT of the chest: Toshiba Aquilion MSCT 64 channels was used. All cases were given pump IV administration of 40 ml of omnipaque 350 mg/ml at a rate of 3 ml/sec.

IV. Results

See the summary of MSCT results in tables 2-4. Table 2 shows summary of the MSCT findings in our 20 cases, while table 3 gives summary of the SVC different MSCT appearances and table 4 shows collateral circulation in MSCT.

Table 2 : Summary of MSCT Findings

Case presentation	MSCT finding
5 Behçet cases	-Partial SVC obstruction -Eminent **mediastinal** collaterals
6 central infiltrating Bronchogenic carcinoma	Partially Infiltrated SVC with few collaterals noted in 5 cases and eminent **chest wall** collaterals in one case (the case was a follow up case of Bronchogenic carcinoma after radiotherapy).
Case of mediastinal syndrome of unknown etiology	Diffuse affection of the mediastinum with soft tissue density lesion causing effacement of SVC and marked attenuation of the right pulmonary artery ; a picture suggesting fibrosing mediastinitis* Eminent **chest wall** collaterals seen.
2 cases with left upper limb DVT	DVT seen extending into left brachiocephalic vein. Normal contralateral brachiocephalic vein and SVC.
2 cases with lymphoma	Huge anterior mediastinal mass .Displaced and compressed SVC. No collaterals
One case of chest assessment for metastases	Lung, pleura and mediastinal metastases .SVC distortion and partial infiltration . No collaterals
One case of fever of unknown origin after chemotherapy	Partial SVC obstruction with a heterogenous thrombus**with air density, enlarged SVC, no collaterals
One case of Antiphospholipid antibody syndrome	Partial SVC obstruction at lowest portion , no collaterals
One case of bilateral pleural effusion	Partial SVC thrombus in relation to CV-Line catheter, no collaterals

*case was proved fibrosing mediastinitis.

** Case was proved to have MRSA infection.

Table 3 : Summary of MSCT SVC findings

MSCT SVC findings	No. of cases
Partial obstruction	9 cases
Infiltration	7 cases
Normal	4 cases

Table 4 : MSCT detection of collaterals

No of cases	HRCT finding
5 Behçet cases	Eminent collaterals
1 case of fibrosing mediastinitis	Eminent collaterals
1 case of infected thrombus	No collaterals
2 cases with partial thrombus	No collaterals
5 cases of malignant mediastinal infiltration by Bronchogenic carcinoma	Few collaterals mostly chest
1 case of follow up Bronchogenic carcinoma with SVC infiltration	Eminent chest wall collaterals
1 case of 2ry malignant mediastinal infiltration.	No collaterals

V. Discussion

Multi–detector row computed tomography (CT) with 2D and 3D reconstructed images provides a unique perspective on thoracic anatomy and disease. Multi–detector row CT allows shorter acquisition times, greater coverage, and superior image resolution *(7).*

In vascular imaging, this would provide image quality that equals or surpasses that of conventional angiography. Its use has expanded to aid in diagnosis and surgical planning, as it is reliable in depicting clot, thoracic vasculature and may also be used to evaluate thoracic venous anomalies and to plan therapy *(8).*

Multi–detector row CT has been used for venous angiography. Superior vena cava obstruction, often related to tumor, is frequently seen at multi–detector row CT. This technique may be used to establish the extent of tumor involvement and document the extent of collateral vessel formation *(9).*

MDCT, with its multi-planar imaging capabilities, allows thorough anatomic delineation of the various collateral pathways diverting the blood from the site of obstruction. Although axial images allow evaluation of potential causes of superior vena cava obstruction, such as a mediastinal mass, if intervention is warranted, MDCT provides valuable information about the level and egree of superior vena cava obstruction, the length of the affected segment, and the presence or absence of intra-luminal clot distal to the obstruction, thereby allowing the interventional radiologist to choose the optimal treatment option *(10).*

Superior vena cava syndrome usually presents more gradually with an increase in symptoms over time as malignancies increase in size or invasiveness *(11).* The severity of the syndrome depends on the rapidity of onset of the obstruction and its location *(12).* (see table 5).

Table 5 : Proposed grading system for superior vena cava syndrome: (Wilson, 2007).The blue one

Grade	Category	Incidence %	Definition
0	Asymptomatic	10	Radiographic superior vena cava obstruction in the absence of symptoms
1	Mild	25	Edema in head or neck (vascular distention), cyanosis, plethora
2	Moderate	50	Edema in head or neck with functional impairment (mild dysphagia, cough, mild or moderate impairment of head, jaw or eyelid movements, visual disturbances caused by ocular edema)
3	Severe	10	Mild or moderate cerebral edema (headache, dizziness) or mild/moderate laryngeal edema or diminished cardiac reserve (syncope after bending)
4	Life-threatening	5	Significant cerebral edema (confusion, obtundation) or significant laryngeal edema (stridor) or significant hemodynamic compromise (syncope, hypotension, renal insufficiency)
5	Fatal	<1	Death

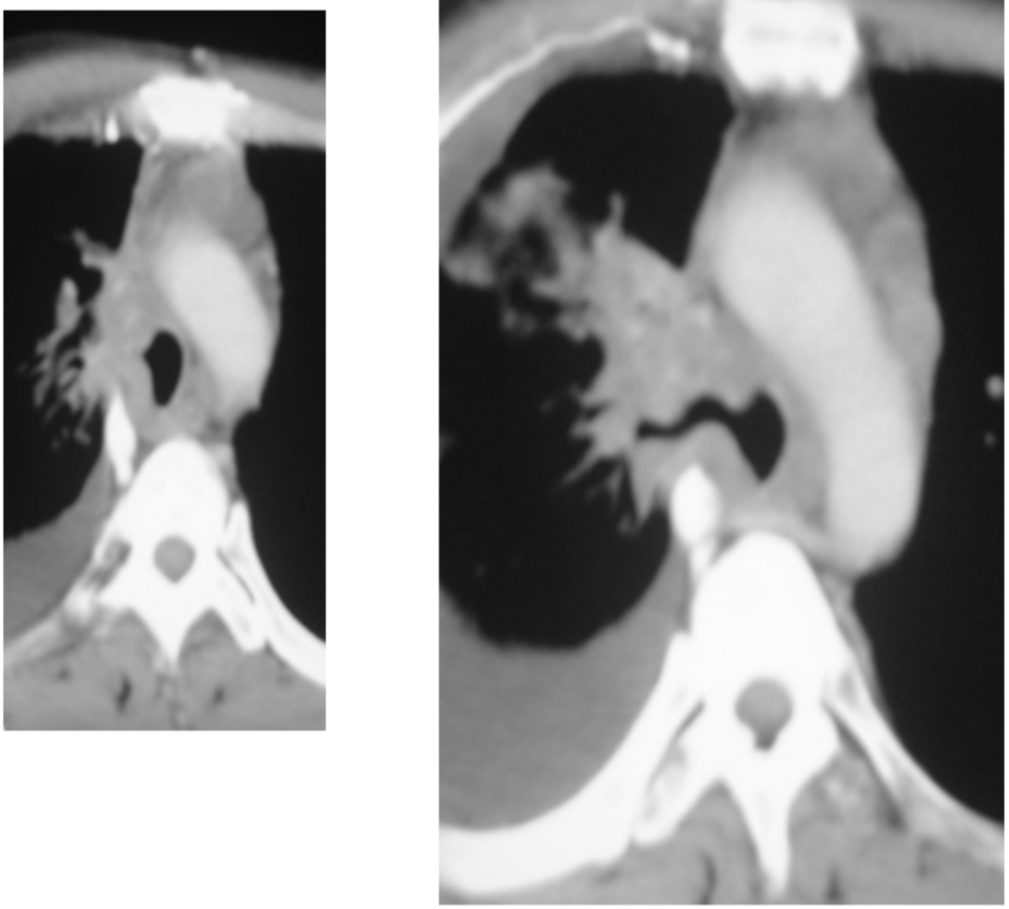
SVC syndrome can lead to the formation of downhill esophageal varices and pleural effusion. Numerous case reports have described pleural effusions in conjunction with the SVC syndrome. These effusions occur in 60% of SVC syndrome cases. The effusions are small, usually occupying less than one-half of the affected hemithorax, and occur approximately equally on either side or bilaterally. Although previously thought to be largely transudates, a large case series found that 18% of the effusions were chylous, with the remainder being exudates. None of the effusions sampled in the series were transudates. Occluded lymphatic flow from increased hydrostatic pressure in the SVC and left brachiocephalic vein probably contributes to the development of chylous pleural fluid. The pathophysiology of the exudative effusions, however, remains unknown. Many factors, including diuresis, small pulmonary emboli, and the underlying inflammatory or malignant condition all likely contribute. Chylous or exudative pleural effusions occur in most patients with SVC syndrome. The effusions are usually small and resolve upon correction of the underlying SVC obstruction. ***(13).***

Five to 10 percent of cases of SVC obstruction are due to benign causes. Most result from invasive monitoring techniques, such as the placement of central venous lines, Swan-Ganz catheters, and interventional techniques, such as the placement of pacemakers and central venous catheters for chemotherapy ***(14).***

The most common malignant causes are non–small-cell lung cancer (fig 1,2). Other types of cancer that can lead to this condition include: Breast cancer, lymphoma, metastatic lung cancer (lung cancer that spreads), testicular cancer, thyroid cancer, thymic tumors. (fig 3) ***(15).***

Case 1

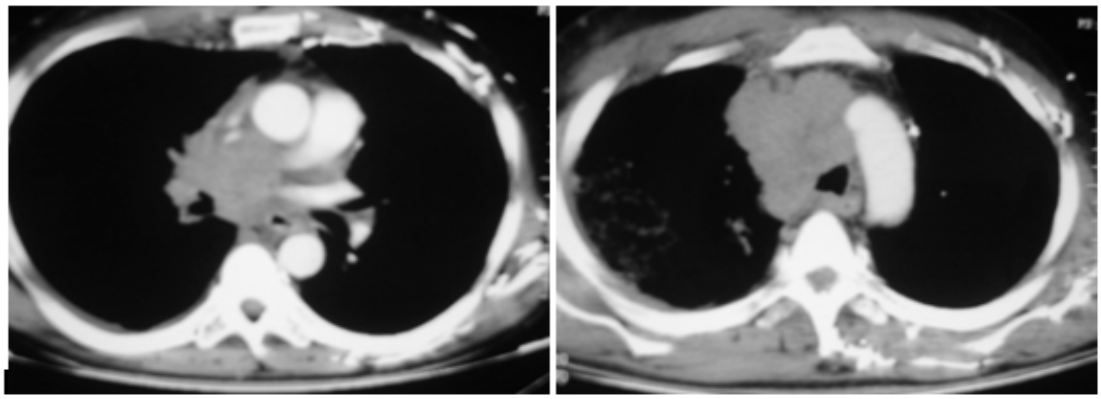
- **Clinical Data**
  48 year-old male patient known central Bronchogenic carcinoma presenting with mediastinal syndrome.
- **MSCT**



CECT chest mediastinal window axial images at different levels showing right central bronchogenic carcinoma with partial infiltration of SVC and chest wall collaterals. Note prominent azygos vein.

Case 2

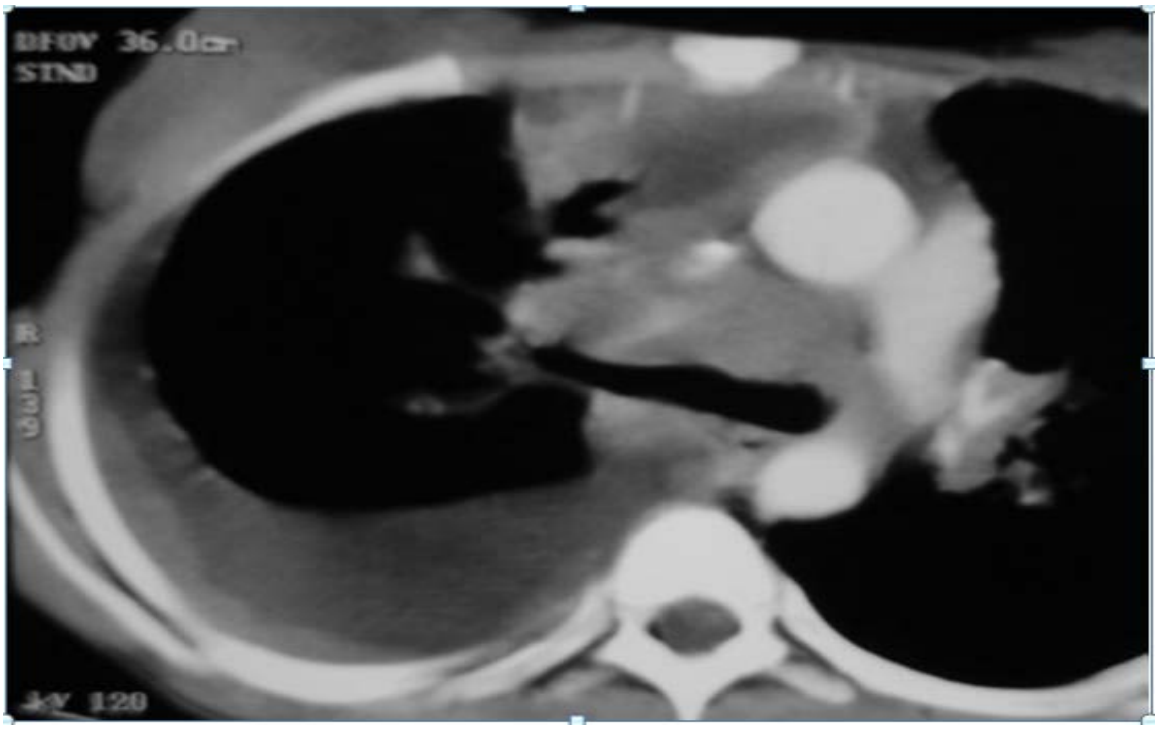
- **Clinical Data**
  39 year-old male patient known central Bronchogenic carcinoma with mediastinal syndrome. Case coming for follow up after radiotherapy.
- **MSCT**



CECT chest mediastinal window axial images at different levels showing right central bronchogenic carcinoma with partial infiltration of SVC and chest wall collaterals. Note non-enhanced azygos vein.

Case 3

- **Clinical Data**
  15 year-old female patient known ovarian carcinoma surveyed for metastases.
- **MSCT**



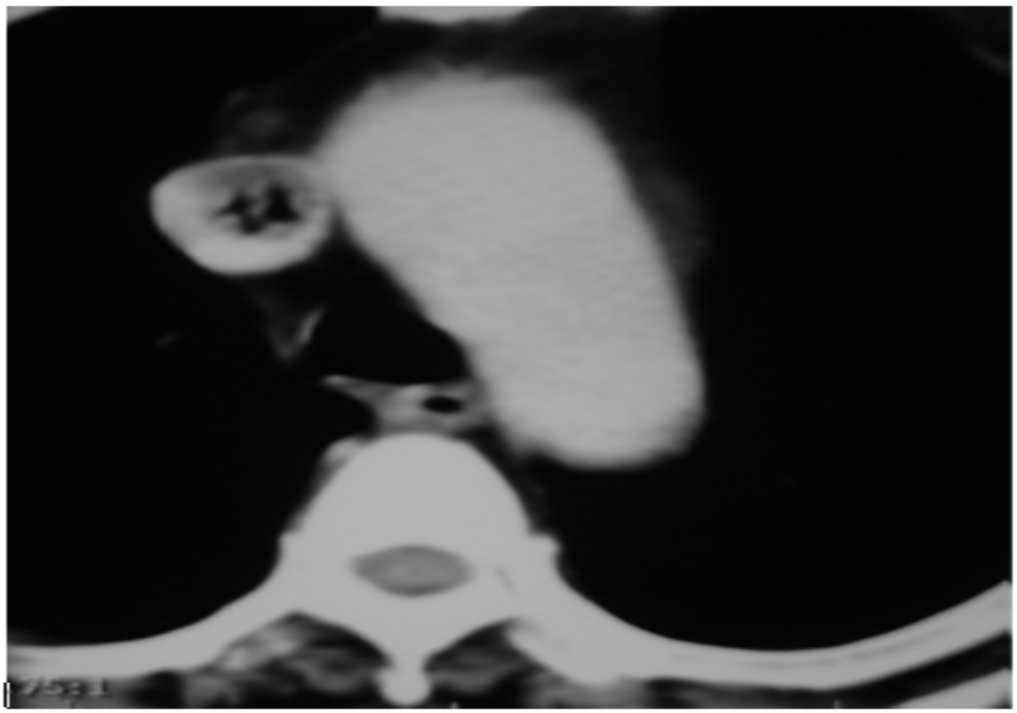
CECT **chest** mediastinal window axial image showing right moderate pleural effusion with thickened enhanced parietal pleura and mediastinal infiltration causing distortion and infiltration of SVC. No collaterals detected.

Non malignant conditions causing superior vena cava syndrome (SVCS) include mediastinal fibrosis; vascular diseases such as aortic aneurysm, vasculitis, and arteriovenous fistulas; infections such as histoplasmosis, tuberculosis, syphilis, and actinomycosis; benign mediastinal tumors such as teratoma, cystic hygroma, thymoma, and dermoid cyst; cardiac causes, such as pericarditis and atrial myxoma; and thrombosis related to the presence of central vein catheters. These account for approximately 22% of the causes of superior vena cava syndrome (SVCS) ***(16).***

- Iatrogenic superior vena cava obstruction (fig 4)—Iatrogenic causes of SVCS include venous thrombosis as a consequence of central venous catheters or pacemaker catheters and also fibrosis, caused by radiation therapy of the mediastinum.
- Thrombi in the SVC were detected by trans-esophageal echocardiography in 30% of patients who had single lumen silicone rubber hemo-dialysis catheters. Polyvinyl chloride, polyethylene and teflon catheters are associated with increased thrombogenisity, as compared to silicone rubber. Furthermore, the SVC stenosis may be induced by persistent trauma of the endothelium by the catheter tip and the higher blood flow during dialysis hours *(17).*
- Fibrosing mediastinitis (fig 5)—it is a rare benign disorder caused by proliferation of acellular collagen and fibrous tissue within the mediastinum. Although many cases are idiopathic, many (and perhaps most) cases in the United States are thought to be caused by an abnormal immunologic response to Histoplasma capsulatum infection. Affected patients are typically young and present with signs and symptoms of obstruction or compression of the superior vena cava, pulmonary veins or arteries, central airways, or esophagus. There may be two types of fibrosing mediastinitis: focal and diffuse. The focal type usually manifests on computed tomographic (CT) or magnetic resonance (MR) images as a localized, calcified mass in the para-tracheal or sub-carinal regions of the mediastinum or in the pulmonary hila. The diffuse type manifests on CT or MR images as a diffusely infiltrating, often non-calcified mass that affects multiple mediastinal compartments. CT and MR imaging play a vital role in the diagnosis and management of fibrosing mediastinitis *(18).*
- Behçet disease (fig 6,7) —Behçet disease is a multisystem disease of unknown etiology. The syndrome carries the name of the Turkish dermatologist Hulusi Behçet, who, in 1937, described a syndrome of recurrent aphthous ulcers, genital ulcerations, and uveitis leading to blindness. Although the cause of the disease is still unknown, it has become recognized as a multisystemic inflammatory disease *(19).*
- Enlargement of the thyroid gland (goiter) *(20).*

Case 4

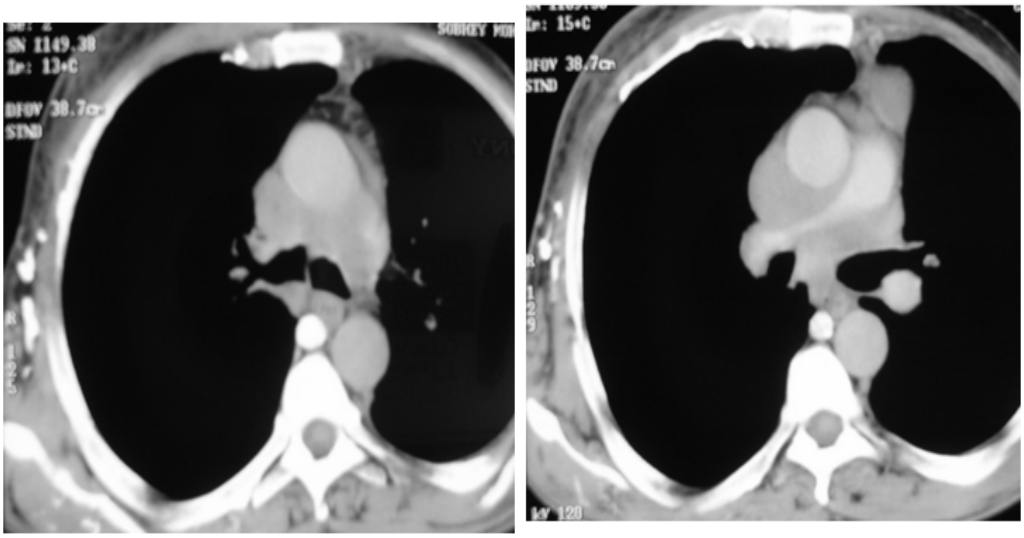
- Clinical Data
  60 year-old female patient investigated for fever of unknown origin.SVC heterogenous thrombus by CECT proved to be MRSA infected.
- MSCT



CECT chest axial image mediastinal window shows Partial SVC obstruction with a heterogeneous thrombus with air density, enlarged SVC, no collaterals.

Case 5

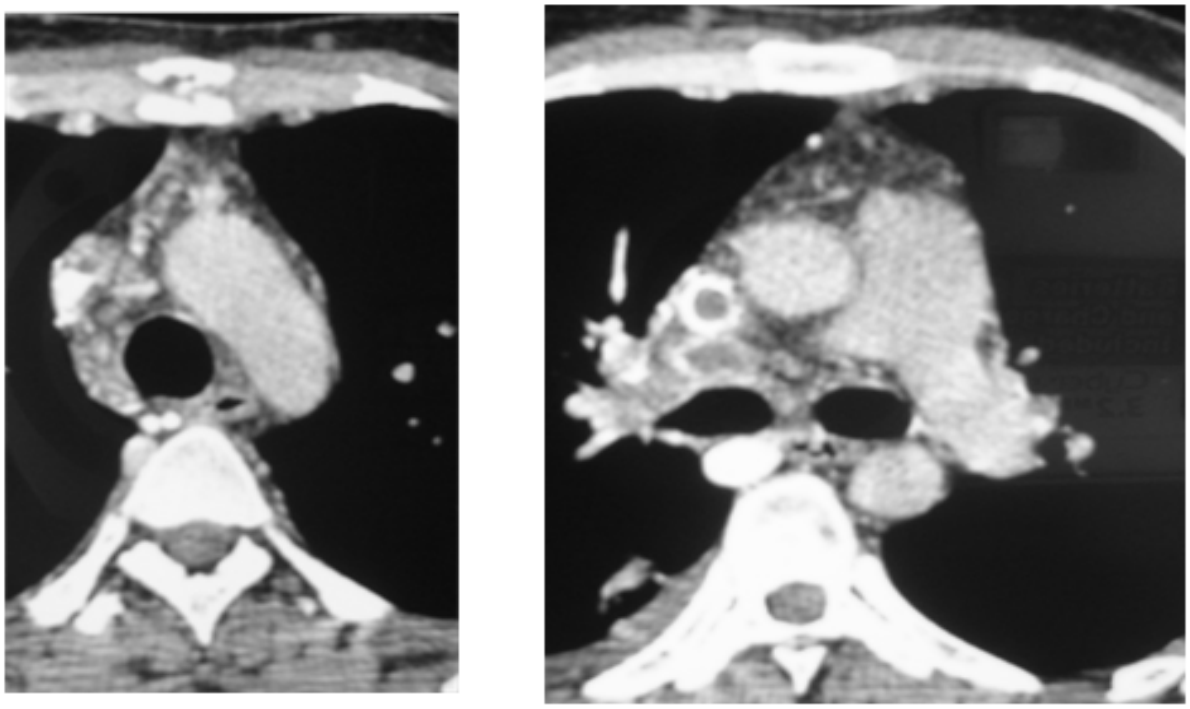
- **Clinical Data**
  65 year-old female patient with mediastinal syndrome proved to be fibrosing mediastinitis.
- **MSCT**



CECT chest mediastinal window axial images at different levels in a case of fibrosing mediastinitis showing effacement of SVC and chest wall collaterals. Note prominent azygos vein. Marked attenuation of the right pulmonary artery.

Case 6

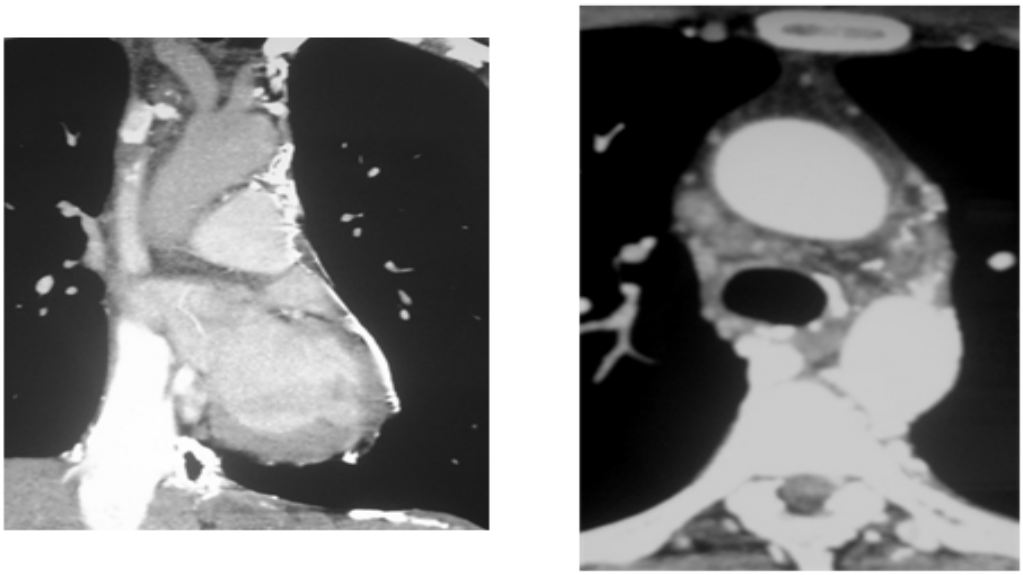
- **Clinical Data**
  28 year-old male patient known Behçet presenting with mediastinal syndrome.
- **MSCT**



CECT chest mediastinal window axial images at different levels showing partial thrombus of SVC with eminent mediastinal collaterals. Note prominent azygos vein.

Case 7

- **Clinical Data**
  31 year-old male patient known Behçet presenting with mediastinal syndrome.
- **MSCT**



CECT chest mediastinal window sagittal and axial images partial thrombus of SVC with eminent mediastinal collaterals. Note prominent azygos vein.

The aim of this study was to provide evidence that MSCT is useful in patients with superior vena caval obstruction, detecting the level and the cause of obstruction.

Multi-slice CT scans were reviewed in 20 patients referred from chest department to radiology department in Kasr Al-Aini.

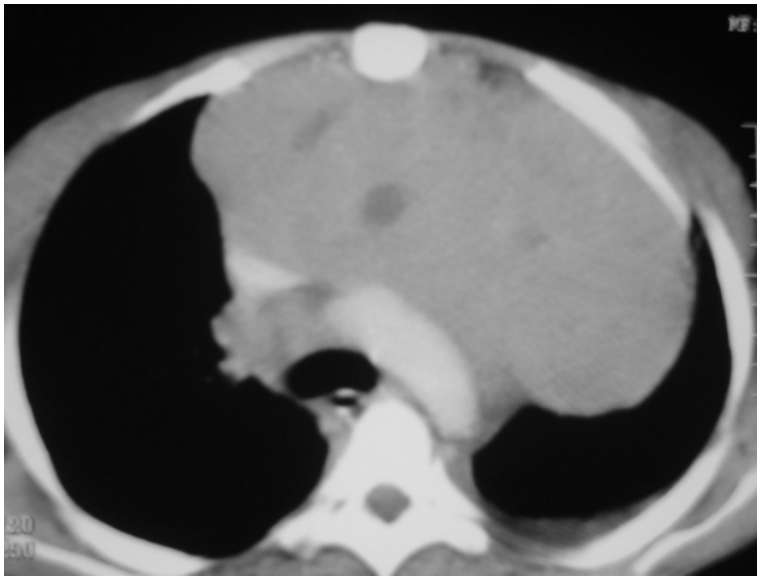
Twelve patients presented clinically in our study with mediastinal syndrome, Two cases had right upper limb DVT. Two cases had lymphoma and one case was surveyed for chest metastases. One case had fever of unknown origin after chemotherapy. One case with anti-phospholipid antibody syndrome where echo detected calcified right atrial thrombus. One case with bilateral pleural effusion

The twelve patients presented clinically in our study with mediastinal syndrome, six cases were biopsy proved central Bronchogenic carcinoma, five were known cases of Behçet disease and one case with unknown etiology. These findings are more or less consistent with the study made by Bagheri et al, 2009 which stated that among 45 patients with SVCS, their diagnostic pathological reports showed 26 (57.8%) were compatible with bronchogenic carcinoma (small cell lung cancer [SCLC] in 19 cases and non-SCLC in 7) which was the most common etiology of superior vena cava syndrome. Lymphoma was reported in 14 cases (31.1%), (12 of non-Hodjkin's lymphoma and 2 of Hodjkin's lymphoma), and germ cell tumor and malignant thymoma were observed in 3 (6.7%) and 2 (4.4%) of patients ***(21).***

Among the twenty cases reviewed in our study, there were 2 cases diagnosed to have lymphoma (fig 8); MSCT revealed huge anterior mediastinal mass, displaced and compressed SVC with no collaterals. These findings are not consistent with the study made by ***Kantarci et al, 2008*** which presented multi-detector row CT (16-detector scanner) features of a case of SVC syndrome caused by compression of lymphadeno-pathies at Hodgkin lymphoma of an 8-year-old girl. Her chest radiograph showed mediastinal enlargement and computed tomography of the chest showed massive mediastinal lymphadenopathy, axillary adenopathy, and bilateral pleural effusions. Multi Slice Computed tomography showed no contrast within the SVC and contrast within ***enlarged collateral venous channels*** of the left chest and no channels on the right because of the right subclavian venous occlusion ***(22).***

Case 8

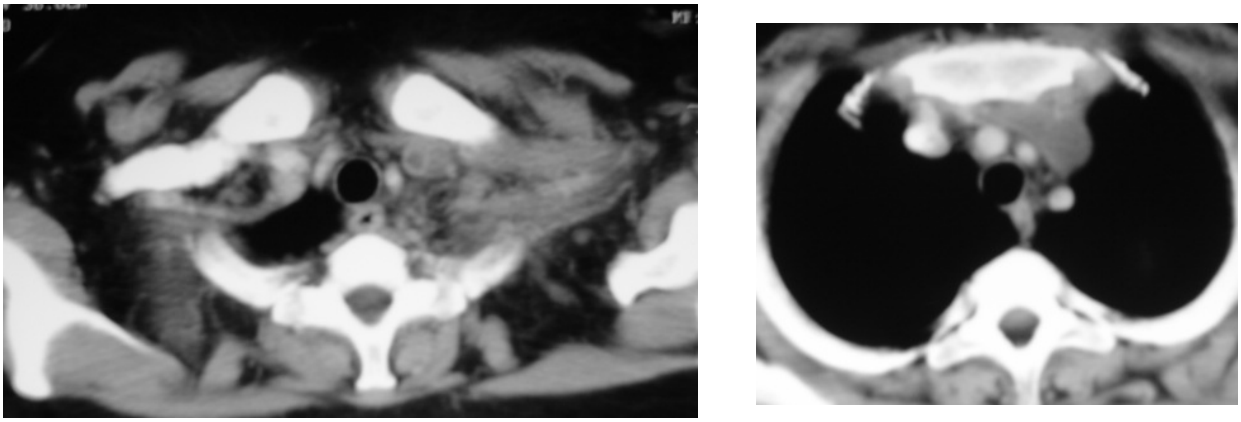
- Clinical Data
  17 year-old female patient known Hogkin's lymphoma.
- MSCT



CECT chest axial image mediastinal window showing huge anterior mediastinal mass compressing and posteriorly displacing SVC.

Case 9

- Clinical Data
  37 year-old female patient with left upper limb DVT.
- MSCT



CECT chest axial images mediastinal window showing the swollen non enhanced left brachiophephalic vein with normal right brachiocephalic vein and SVC.

Osman Temizöz et al, 2010 reported a 50-year-old female patient with BD who was admitted to hospital with a 4-week history of persistent dyspnea and chest pain. Physical examination revealed swelling of the neck and upper extremities. In addition, there were visible venous collateral channels, particularly on the right lateral side of her upper trunk. A clinical diagnosis of SVC syndrome was made, and MDCT of the chest and abdomen revealed occlusion of the SVC. The dilated right lateral chest wall veins were seen as collateral channels crossing the right diaphragm The azygos and hemiazygos veins were also dilated. These findings are more or less consistent with our study having 5 cases of Behcet's disease; their CT scans revealed eminent mediastinal collaterals with prominent azygos vein *(23).*

In our study, 5 Behçet cases revealed partial SVC obstruction and eminent mediastinal collaterals, 6 cases with central infiltrating Bronchogenic carcinoma revealed partially infiltrated SVC with few collaterals noted in 5 cases and eminent chest wall collaterals in

one case (the case was a follow up case of Bronchogenic carcinoma after radiotherapy), 1 Case of mediastinal syndrome of unknown etiology showed diffuse affection of the mediastinum with soft tissue density lesion causing effacement of SVC and marked attenuation of the right pulmonary artery ; a picture suggesting fibrosing mediastinitis with eminent chest wall collaterals, 2 cases with left upper limb DVT seen extending into left brachiocephalic vein and normal contra-lateral brachiocephalic vein and SVC, 2 cases with lymphoma showed huge anterior mediastinal mass, displaced and compressed SVC with no collaterals, one case of chest assessment for metastasis revealed lung, pleura and mediastinal metastases with SVC distortion and partial infiltration and no collaterals, one case of fever of unknown origin after chemotherapy showed partial SVC obstruction with a heterogenous thrombus with air density, enlarged SVC, no collaterals, one case of Antiphospholipid antibody syndrome revealed partial SVC obstruction at lowest portion , no collaterals, one case of bilateral pleural effusion revealed partial SVC thrombus in relation to CV-Line catheter, no collaterals.

To conclude, the rate of obstruction and its location greatly affects the severity of symptoms of SVCS, this depends on the development of the collateral circulation. Meaning that when the onset of obstruction is slow (as in benign causes of obstruction e.g. Behcet's disease and with central venous catheters), the collateral circulation will have time to distend and accommodate the increased blood flow. On the other hand, diseases causing rapid onset of obstruction (malignant tumors), produce more severe symptoms because the collateral veins will not have time to distend. *Longmore et al, 2007* suggested that the general recruitment of venous collaterals over time may lead to remission of the syndrome although the SVC remains obstructed *(24).*

Wilson, 2007 stated that not all cases of SVC obstruction must present with SVCS (e.g. facial edema, venous distension in the neck, upper limb edema) and that there is 10% of cases may have radiographic SVC obstruction in absence of symptoms. This is consistent with our study which includes twelve of the twenty patients presented clinically with mediastinal syndrome, two cases had right upper limb DVT, two cases had lymphoma and one case was surveyed for chest metastasis. One case had fever of unknown origin after chemotherapy, one case with anti-phospholipid antibody syndrome and one case with bilateral pleural effusion *(25).*

VI. Summary and Conclusion

Superior vena cave syndrome (SCVS) is a constellation of signs and symptoms resulting from obstruction of the SVC or its major tributaries by intra-luminal occlusion or by extrinsic compression and/or invasion from malignant and benign diseases.

SVC obstruction leads to increased venous pressure and edema of the neck, arms, upper chest, and head causing increased intracranial pressure. Patient may present with headache, syncope or pre-syncope, nausea and vomiting, hoarseness, dysphagia, cough, dyspnea and chest pain. Severity of symptoms depend on the time course of obstruction. As obstruction develops, venous collaterals develop to find alternate pathways for venous return to the right atrium. In the post-antibiotic era malignancy remains the commonest etiology. Lung cancer is the commonest malignancy. SVCS is most common with small cell lung cancer as it grows rapidly in central airways. CT chest is the investigation of choice which provides information on location, possible etiology, extent of collaterals and guide biopsy attempts.

The advent of multi-detector CT has revolutionized imaging of the mediastinal vascular structures. In comparison to single-detector helical CT scanners, multi-detector scanners not only provide faster speed, greater coverage, and improved spatial resolution, but also have the unique ability to create images of thick and thin collimation from the same data set.

One of the greatest benefits of this new technology is the improved quality of two-dimensional (2D) multi-planar and three-dimensional (3D) reconstruction images.

MSCT can easily prove or exclude the affection of SVC by partial or complete obstruction, the development of collateral circulation as well as detecting the cause of obstruction whether thrombosis, compression or infiltration and its extent.

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