SVC Syndrome: Can We Predict SVC Syndrome on CT?

Mi-Jin Kang

Department of Radiology, Sanggye Paik Hospital
Inje University, Republic of Korea
Email: S2621 [AT] paik.ac.kr

_____

ABSTRACT---

OBJECTIVE: To systematically investigate imaging findings of superior vena cava syndrome(SVCS) on CT to predict SVCS in malignancy patients.

SUBJECTS AND METHODS: From Janunary 2010 to August 2011, chest CTs with narrowing or obstruction of superior vena cava(SVC) were searched from hospital imaging database. After that patients were classified into two groups; SVCS group and non-SVCS group, based on clinical manifestation of SVCS. The long diameter of tumor, presence or absence of intratumoral nerosis, short diameter of SVC, presence or absence of architecture distortion of SVC, subcutaneous edema, and collateral vessels were evaluated.
*   Differences between SVCS group and non-SVCS group were analyzed using the Wilconcox test and x2 test (p<0.05).*
RESULTS: A total of 29 patient were included in this study. Among them, 11 patients were SVCS group, and 18 patients were non-SVCS group. The mean diameter of main mass was 8.2cm and 5.5cm in SVCS group and non-SVCS, respectively (p=0.04). The presence of collateral vessels was significantly frequent in non-SVCS group (n=4 of 18, 22%) than SVCS group (n= 1of 11, 9%)   (p<0.01). The prevalence of chest wall edema was much frequent in SVCS group, but there was no statistically significant difference (p=0.09). The central necrosis of the main mass were found in 5 of 11 in SVCS group (45%), and in 8 of 18(44%) in non-SVCS group (p=0.72). There was no statistical significant difference in SVC diameter or architecture distortion between two groups.

CONCLUSION: Knowledge of the predictive imaging findings of SVSC in malignant patients leads to more proper management of patients.

Keywords---SVC syndrome, cancer
_____

1. INTRODUCTION

SVC syndrome due to malignant disease was considered an oncologic emergency. Patients with untreated malignant SVCS survive for only about 30 days. But this condition usually develops over days to weeks, with subtle symptoms at an early stage. Therefore if doctor could detect sign or imaging change of SCV syndrome, we can treat it more properly.

2. SUBJECTS AND METHODS

Patients

From Janunary 2010 to August 2011, I searched my hospital's imaging database for chest CT with narrowing or obstruction of superior vena cava. With review of pathologic reports, only malignant diseases were included.

Among them the patients were divided into 2 groups: 1) A; With SVCS: patients with clinical suspicion of SVCS or underwent radiation therapy due to SVCS, 2) B; Without SVCS: patients with no clinical suspicion of SVCS. Demographic data, underlying malignancy of each patients were also recorded.

All CT examinations were performed using multi-detector CT scanners: 320-detector rows (Acquillion One; Toshiba Medical Systems Corporations, Otawara, Japan) and 64--detector rows (Acquillion 64; Toshiba Medical Systems). Contrast-enhanced chest CT was obtained after injection of 80 mL of iodinated contrast agent at a rate of 2.3 mL/s. Image acquisition was obtained from lung base to apex, 40 seconds after injection of contrast media. Parameters of CT scans were 120 kVp, 40-100 mAs, 3 mm thickness, and 3 mm collimation.

CTs were interpreted by one radiologist with 8 years of chest radiology experience (M.J. K). During interpretation the radiologist did not know the clinical date of the patients. In terms of main mass, long diameter of tumor, presence or absence of intratumoral nerosis were evaluated. In terms of superior vena cava, short diameter at narrowest point,

presence or absence of architecture distortion were evaluated. And as ancillary findings such as subcutaneous edema, collateral vessels were evaluated.

Differences between SVCS group and non-SVCS group with regard to clinical data, imaing findings were analyzed using the Wilconcox test and x2 test. All statistical analyses was performed using statistical software (SAS, version 5.0, SAS QI). For all statistical analyses, $p<0.05$ was considered to denote statistical significance.

3. RESULTS

From the searching of imging database, 30 cases mentioning the narrowing or obstruction of superior vena cava were detected. According to the electric medical record (EMR), 1 case of tuberculosis was excluded. Thus, remained 29 cases were included in this study. In term of pathologic diagnosis, small cell lung cancer (n=8) and adenocarcinoma (n=8) were most common, and followed by squamous cell carcinoma(n=6), non-small cell lung cancer (n=2), large cell carcinoma (n=1), fibrosarcoma (n=1), and undifferentiated carcinoma (n=2). One case was expired before pathologic comfirm.

Among 29 patients, 11 patients (M: F=9:2, 50-89 years old) were classified into SVCS group, with clinical suspicions of SVCS or underwent emergent radiation therapy due to SVCS. The remaining 18 patients were classified into non-SVCS group (M: F=14:4, 51-85 years old).

In term of mean long diameter of main mass was 8.2cm and 5.5cm in SVCS group and non-SVCS, respectively. There for long diameter of main was significant large in SVC group (p=0.04). The central necrosis of the main mass were found in 5 of 11 in SVCS group (45%), and in 8 of 18(44%) in non-SVCS group (p=0.72).

In terms of SVC, the short diameter was measured from 0-4.7mm in SVCS group and 0-9mm in non-SVCS group. There was no statistically significant difference between two groups (p=0.62). The loss of SVC architecture was observed, 2 of 11 (18%) in SVCS group, and 2 of 18 (11%) in non-SVCS group (p=0.26).

In the evaluation of ancillary finding, chest wall edema was observed, 5 of 11 (45%) in SVCS group, and 3 of 18 (16%) in non-SVCS group. The prevalence of chest wall edema was much frequent in SVCS group, but there was no statistically significant difference (p=0.09). The presence of collateral vessels was significantly frequent in non-SVCS group (n=4 of 18, 22%) than SVCS group (n= 1of 11, 9%) (p<0.01)

4. DISCUSSION

SVCS is obstruction of blood flow through the superior vena cava due to various cause. William Hunter first described the syndrome in 1757 in a patient with syphilitic aortic aneurysm. [1] Nowadays, more than 80% of cases of SVCS are caused by malignant tumors. [2-4] Bronchogenic carcinomas account for 75-80% of all these cases. Other malignant diagnoses include lymphoma, thymoa, metastatic cancer, at al.[5-8]In terms of lung cancer, approximately 4% of patients manifested in SVCS [9]. And 10-20% of patients with small cell lung cancer suffered with SVCS. And 1.9% patients with lymphoma showed SVCS.[10]

SVC syndrome due to malignant disease was considered an oncologic emergency, but only rarely does it present as a truly life-threatening emergency. This condition usually develops over days to weeks, with subtle symptoms at an early stage. Therefore if doctor has caution to detect SCV syndrome, we can treat it properly.

In this report, I studied predictive factor of SVC syndrome on CT, including long diameter of tumor, intratumoral nerosis, short diameter of SVC, architecture distortion of SVC, subcutaneous edema, and collateral vessels. And I found out that main tumor was larger in SVC group and that collateral vessel was much frequent in non-SVC group. I also found that subcutaneous edema also much frequent in SVC group, but statistically not significant.

This study has several limitations. First, this investigation was limited to a relatively small number of patients, so further study with a larger population may be needed in order to confirm our data. Second, due to small data I did not match the number of two groups. So further study with balanced two group may be needed.

5. ACKNOWLEDGMENT

6. REFERENCES

1. National Cancer Institute. Cardiopulmonary syndromes. 2005.

2. Schraufnagel DE, Hill R, Leech JA, et al. Superior vena caval obstruction. Is it a medical emergency?. *Am J Med*. 1981 Jun. 70(6):1169-74.

3. Klassen KP, Andrews NC, Curtis GM. Diagnosis and treatment of superior-vena-cava obstruction. *AMA Arch Surg*. 1951 Sep. 63(3):311-25.

4. Flounders J. Superor vena cava syndrome. *Oncol Nurs Forum*. 2003. 30(4):E84-8.

5. Ahmann FR. A reassessment of the clinical implications of the superior vena caval syndrome. *J Clin Oncol*. 1984 Aug. 2(8):961-9..

6. Sakura M, Tsujii T, Yamauchi A, et al. Superior vena cava syndrome caused by supraclavicular lymph node metastasis of renal cell carcinoma. *Int J Clin Oncol*. 2007 Oct. 12(5):382-4.

7. Nieto AF, Doty DB. Superior vena cava obstruction: clinical syndrome, etiology, and treatment. *Curr Probl Cancer*. 1986 Sep. 10(9):441-84.

8. Lochridge SK, Knibbe WP, Doty DB. Obstruction of the superior vena cava. *Surgery*. 1979 Jan. 85(1):14-24.

9. Akoglu H, Yilmaz R, Peynircioglu B, et al. A rare complication of hemodialysis catheters: superior vena cava syndrome. *Hemodial Int*. 2007 Oct. 11(4):385-91..

10. Houman M, Ksontini I, Ben Ghorbel I, et al. Association of right heart thrombosis, endomyocardial fibrosis, and pulmonary artery aneurysm in Behçet's disease. *Eur J Intern Med*. 2002 Oct. 13(7):455..

Table 1. Comparison between SVCS group and non-SVCS group

	SVCS group	non-SVCS group
Age (years old)	50-89 (m=68.8)	51-85 (m=65.4)
M:F	9:02	14:04
LD* of mass(cm)	3.7-14.7 (m=8.2)	3.0-11 (m=5.5)
necrosis of mass	5 of 11 (45%)	8 of 18 (44%)
SD** of SVC(mm)	0-4.7 (m=2.9)	0-9mm (m=3.4)
AD*** of SVC	2 of 11 (18%)	2 of 18 (11%)
collaterals	1 of 11 (9%)	4 of 18 (22%)

*long diameter, ** short diameter, *** architecture distortion

Fig. 1 71-years-old man with SVC syndrome due to Pancoast tumor and tumor thrombi in SVC. Note infiltration in right subcutaneous fat
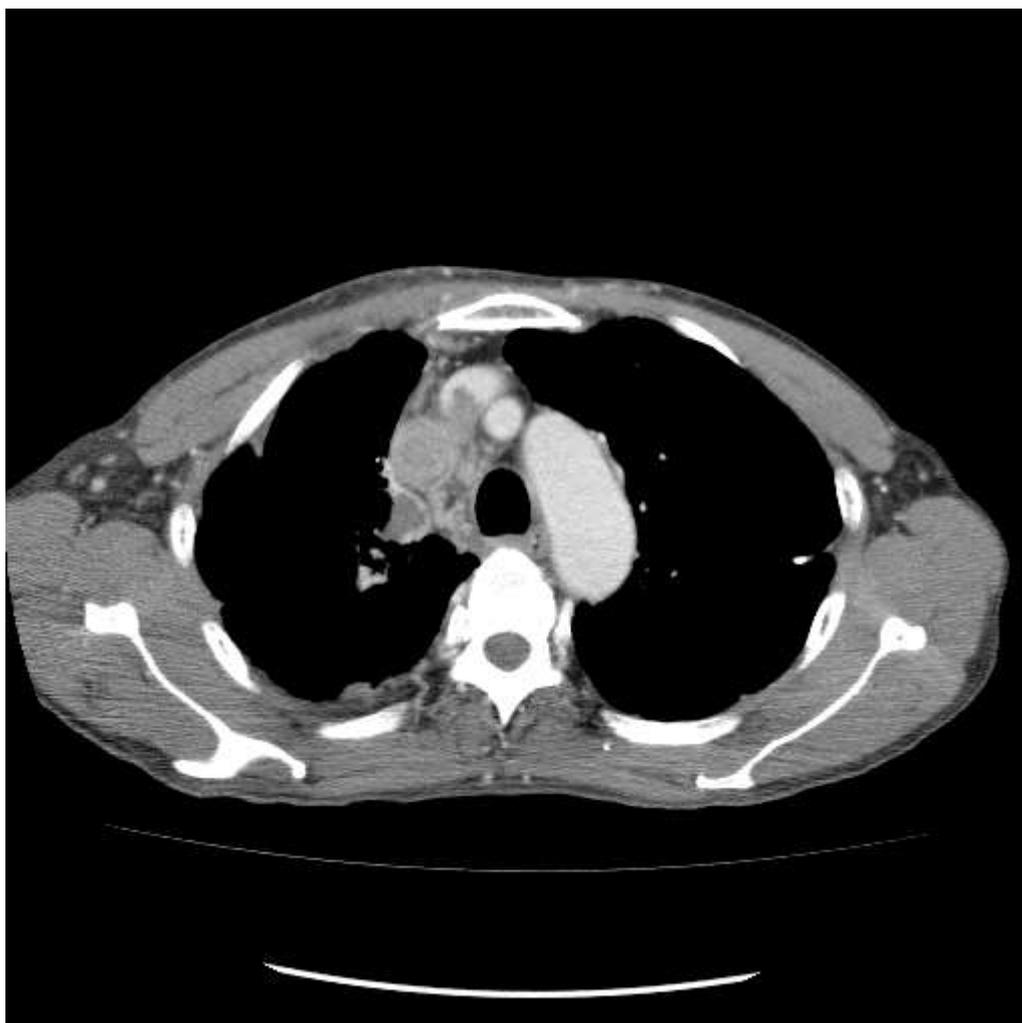
Fig. 2 59-years-old woman with adenocarcinoma. Malignant lymph adenopathy obstruction SVC but with no SVC syndrome. Note collateral vessel in left lateral chest wall