中文題目：血液透析患者併發上腔靜脈症候群-個案報告
英文題目：Superior vena cava (SVC) syndrome in a patient with hemodialysis
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Background：

Superior vena cava (SVC) syndrome is caused by either partial or complete obstruction of blood flow at SVC. Clinically, this obstruction is most commonly resulted from thrombus or tumor compression of the vessel wall, and the patients may develop a series of symptoms, like neck and facial swelling, dyspnea, and cough. We reported a case with SVC syndrome, including the clinical manifestation, diagnostic approach, and therapeutic strategy.

Case Presentation：

A 72-year-old man has a medical history of diabetes, hypertension, old stroke, coronary artery disease, and end stage renal disease under regular hemodialysis. He presented to our emergency department with vomiting, shortness of breath, and progressive facial swelling for three days. Physical examination showed that his consciousness was clear, body temperature was 37.1℃, heart rate was 80 beats per minute, respiratory rate was 18 breaths per minute, and blood pressure was 130/70 mmHg. Additionally, neck jugular engorgement, right facial edema (**Figure 1**), and bilateral clear breath sounds were found. Laboratory data disclosed elevated white blood count ($12*10^3$/uL) with segment predominance (85.3%), and an increased hs-Troponin I level of 13072 pg/mL (reference range, 0~26 pg/mL). Electrocardiogram (ECG) showed no specific ST-T change (**Figure 2**). Chest X-ray film revealed neither cardiomegaly, pleural effusion, nor widened mediastinum (**Figure 3**). He was diagnosed with non-ST elevation myocardial infarction (NSTEMI) according to abovementioned findings, and treated with dual antiplatelet therapy (DAPT) and heparinization. However, orthopnea, facial flushing, cyanotic lips, and right upper arm swelling cannot be explained by NSTEMI. Tracing back his history, he was just shifted from Hickman catheter to arteriovenous shunt for hemodialysis six months ago. Therefore, a chest computed tomography (CT) was arranged due to a suspicion of SVC syndrome, which demonstrated focal stenosis of SVC accompanied by upstream total thrombotic occlusion (**Figure 4**). Hence, SVC thrombus complicated with SVC syndrome was diagnosed. Besides, we surveyed his coagulation, e.g., protein C, protein S, and antithrombin III, and the tests were all within normal range. Cardiac catheterization with balloon angioplasty and SVC stenting was performed subsequently (**Figure 5**). His dyspnea and facial swelling

gradually subsided, and we shifted his medication to anti-coagulant (rivaroxaban) plus single anti-platelet agent (clopidogrel). He was then discharged with outpatient department follow-up, and there has been no recurrence so far.

Conclusion：

Clinical features of SVC syndrome can be subtle or dramatic, which require expeditious attention and intervention. Benign etiologies such as stenosis or occlusion of central veins in hemodialysis patients are common, especially with previous intravascular catheter or device use. Palliative therapeutic with endovascular treatment plus anti-coagulation may provide a useful therapeutic strategy.

Figures

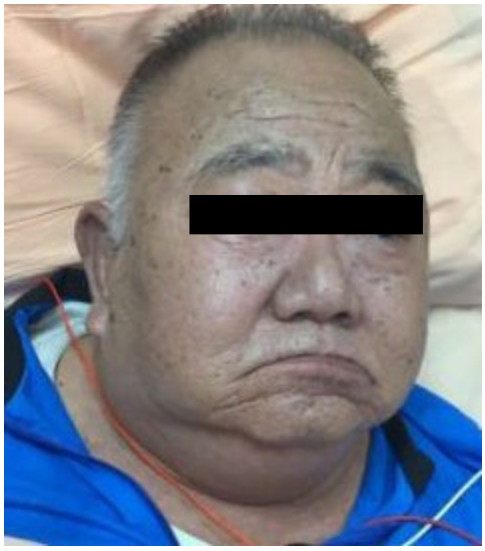

Figure 1： A 72-year-old man suffered from vomiting, dyspnea, and progressive facial swelling. Physical examination showed neck jugular engorgement and right facial edema.

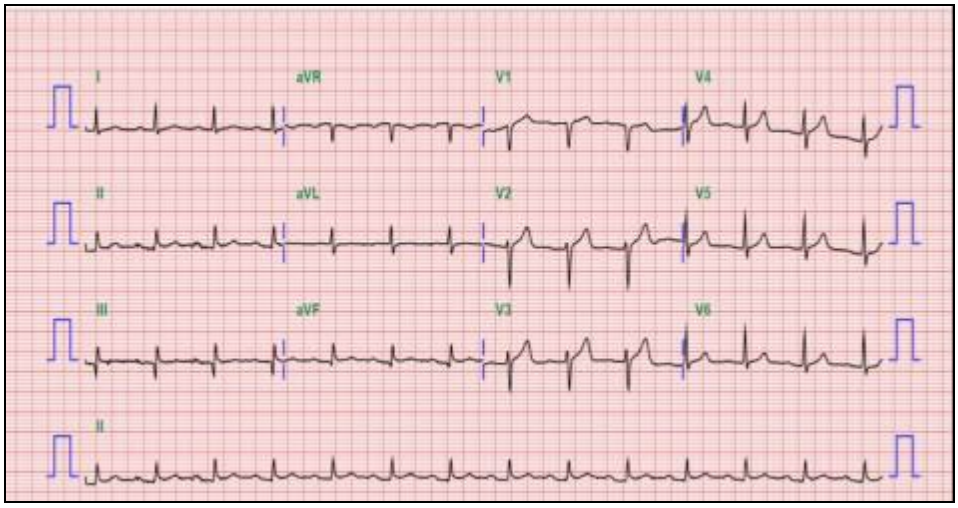

Figure 2：12-lead ECG showed sinus rhythm without specific ST-T change

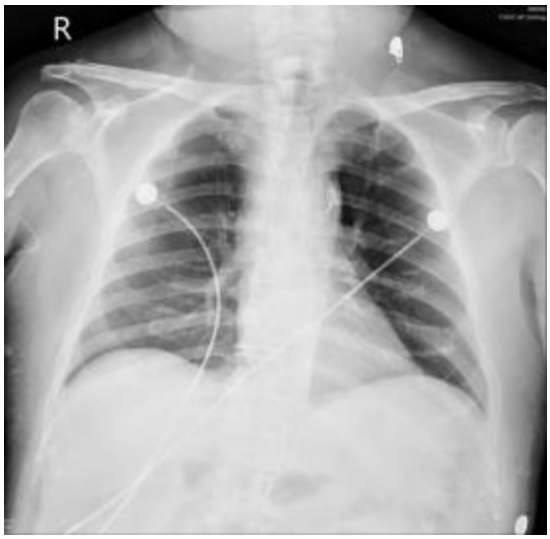

Figure 3：Chest X-ray film revealed no cardiomegaly, pleural effusion, or mediastinum widening

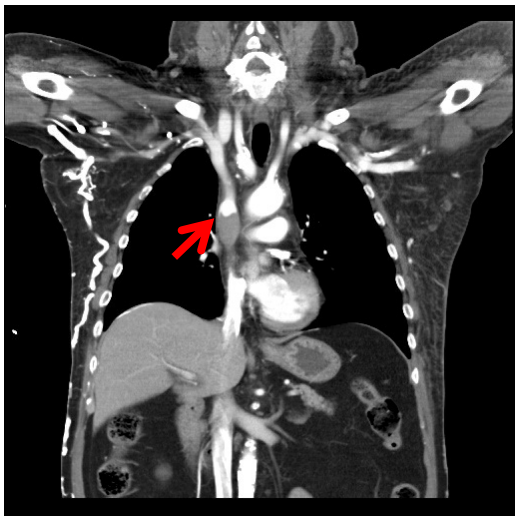

Figure 4：Chest CT showed focal stenosis of SVC associated with upstream total thrombotic occlusion (red arrow)

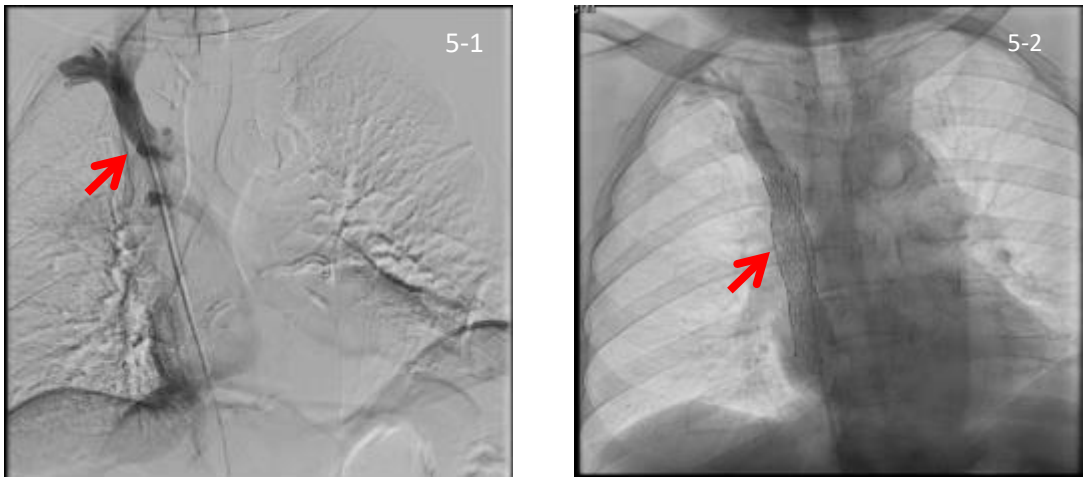

Figure 5：Cardiac catheterization demonstrated SVC total occlusion (5-1, red arrow) and balloon angioplasty with SVC stenting (5-2, red arrow)