

中文題目：以非典型症狀表現的穿孔性主動潰瘍

英文題目：Penetrating aortic ulcer with atypical presentation.

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### **Introduction:**

Penetrating aortic ulcer (PAU) can be life-threatening, causing acute tearing chest or abdominal pain, cold sweating, conscious obtundation, or shock. Very rarely do we see PAU presenting with stable hemodynamics and mild symptoms. In this poster, I would like to give a brief description of an 86-year-old woman diagnosed as PAU with a relatively benign clinical course.

### **Case presentation:**

An 86-year-old female with a history of hypertension under appropriate control suffered from a 2-week duration of poor appetite, yellow skin, and shortness of breath. Associated symptoms include mild left flank soreness sensation and fatigue. No chest pain, abdominal discomfort, cough, nausea, vomiting, or cold sweating was reported. Initial vital signs were within normal range. Laboratory results were remarkable for thrombocytopenia, mild hyperbilirubinemia, and renal function impairment. Chest X ray showed blunting of left costo-phrenic angle and mild trachea deviation to the right (Figure 1), with bloody pleural fluid being aspirated on thoracentesis. Contrast enhanced computed tomography (CT) of the chest reveals contrast leakage from an outpouching aortic arch with crescent hyper-density along the ascending aorta, aortic arch, and thoracic descending aorta, compatible with penetrating aortic ulcer (PAU) with intra-mural hematoma formation (Figure 2). The patient's family refused surgical repair and continued the original anti-hypertensive medications. Interestingly, the patient survives one year after being diagnosed with PAU, and regular follow-up doesn't show progression of the PAU and intramural hematoma.

### **Discussion:**

A penetrating aortic ulcer (PAU) is an atherosclerotic lesion in severe atherosclerotic patients, thought to be formed from aortic plaque rupture. The clinical manifestation of PAU ranges from asymptomatic to anterior tearing chest pain and syncope, mimicking that of an acute aortic dissection. Although many factors, including maximal aortic diameter at the ulcer site, length and width of intimal defect, and presence of pleural effusion, have been researched to assess clinical outcome of PAU, there is still a paucity of predictors of the natural history and

behavior of PAU. Patients may require surgery when symptomatic, large (>2cm), or complicated PAU occurs. Moreover, a more standardized approach to treatment has yet to be established. The hyperbilirubinemia depicted in our case may result from resorption of intramural hematoma.

**Conclusion:**

With advances in imaging techniques, more insight into PAU is obtained, with a more patient based management strategy. In addition, we should keep in mind that it is possible for PAU to have a sub-acute and insidious clinical course, leading to delayed diagnosis and treatment.

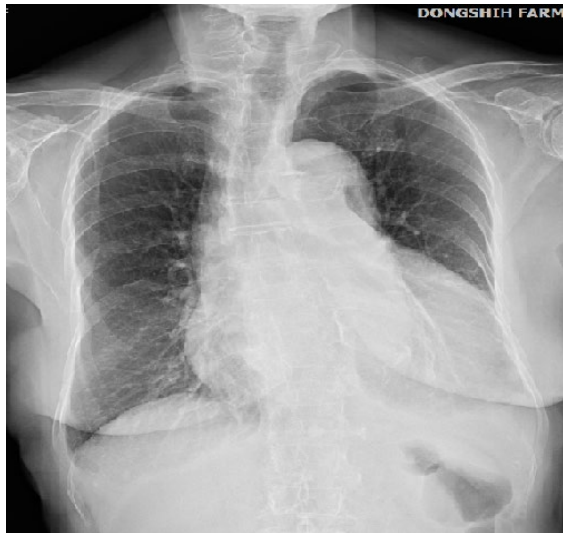


Figure 1. Chest X ray showed left blunting of costo-phrenic angle and right deviation of the trachea.

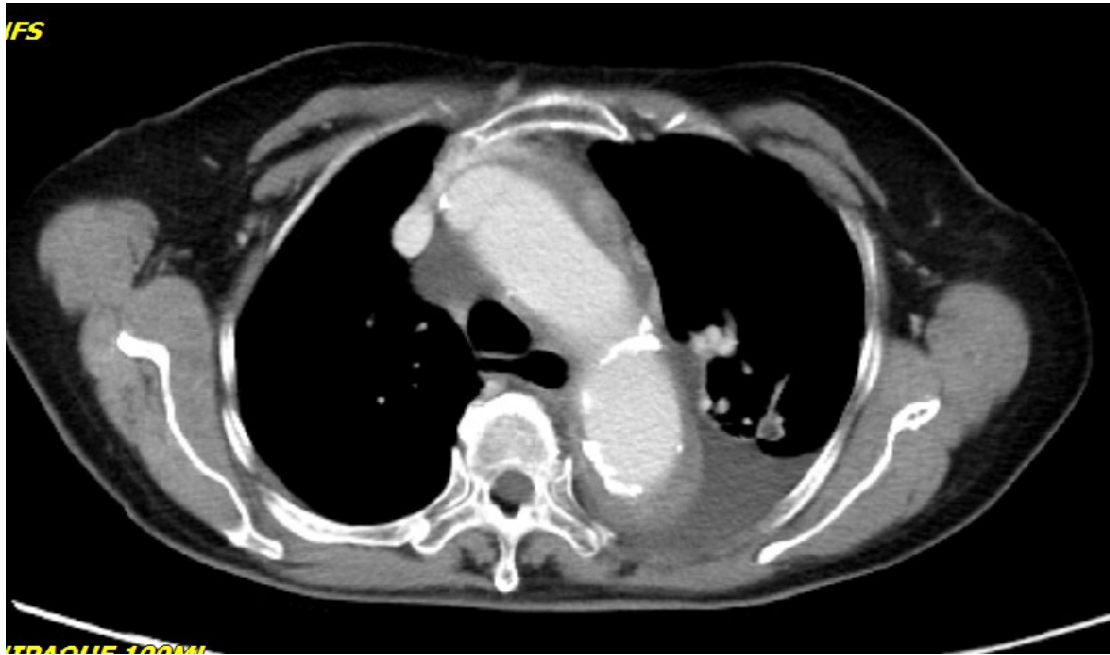


Figure 2. Contrast enhanced computed tomography (CT) of the chest reveals contrast leakage from an outpouching aortic arch with crescent hyper-density along the ascending aorta, aortic arch, and thoracic descending aorta, compatible with penetrating aortic ulcer (PAU) with intra-mural hematoma formation.