One Case of Autopsy Pathological Analysis of Acute Pancreatitis Combined with Hemorrhage in Pericardial Cavity

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Abstract: Senile male, physically fit at usual, he died suddenly without any clinical symptoms. By autopsy dissection, it was found that large amount of bleeding was presented in pericardial cavity, the abdominal cavity and thoracic cavity had a small amount of hemorrhage, partial pancrea tissue had coagulation necrosis accompanied with infiltration of neutrophile granulocyte and degeneration and necrosis of liver cell accompanied with acute or chronic inflammation cell infiltration. Laboratory examination of the patient when he was alive suggested that liver function and coagulation function had obstacles, there was not any timely clinical process, and he died suddenly. Autopsy examination results suggested that acute pancreatitis caused a large quantity of bleeding in pericardial cavity, which led to cardiac tamponade and it cause acute circulation failure, which initiated cardiac arrest and then death. Coronary heart disease may exert certain facilitation effect in the death process. Patients with pancreatitis, especially the senile and pancreatitis patients with coronary artery disease, should be evaluated and prevented ahead of schedule, for those patients who had coma suddenly, it should be thought that it had possibility of combining with hemorrhage in the interior of pericardial cavity, the patient's doctor should try his or her best to reduce death rate.

Keywords: Acute pancreatitis; Hemorrhage in pericardial cavity

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1. Introduction

Acute pancreatitis could cause death due to its severe comorbidity, the most common at early stage was multiple organ function failure, and the most common at later stage was infection.[1] Of these, the symptom with high death rate was hemorrhage in abdominal cavity, those with almost 100% death rate was hemorrhage in pericardial cavity, the occurrence rate of both was relatively low, especially the latter. Those with autopsy report were even rarely seen, at present, 1 case of autopsy analysis on severe symptomatic acute pancreatitis leading to hemorrhage of pericardial cavity was reported.

2. Clinical Data

The deceased is male, 58 years old. He underwent time-ch oosing surgery due to "ring-like hemorrhoid", he could not answer to call when making preoperative preparation, ECG monitoring suggested cardiac arrest, blood pressure could not be measured, SPO2 could not be measured, the patient then died due to ineffective rescue.[2] When the patient was alive, he only had clinical symptoms of hemorrhoid; he had no extenuation, fatigue, no chilly, no fever, no nausea and vomit, no low fever and night sweat. He had regular physical examination annually, he was physically fit, he denied" hepatitis, tuberculosis and typhoid fever" and other infectious disease history and contact history, no "coronary heart disease, diabetics" disease history. He denied trauma and surgical history.

3. Autopsy

3.1 Systematic Anatomy Results

The surface of the corpse showed that the development was normal; skin had no cyanosis and ecchymosis. Bilateral thoracic cavity had 300ml and 840ml light red fluid separately; there was few blood coagulation patches. Bilateral lung surface section was grey and grey white in color with soft nature. There was 900ml blood in pericardial cavity, it was flowed out in eject (Figure 1.2). Cardiac apex surface had an irregular wound in the diameter of 0.5cm, which had not been passed through left ventricle;[3] heart surface partial region was coarse. The anterior descending branch of left coronary artery, the main branch of left coronary artery, the main branch of right coronary artery and aortic arch had local atherosclerosis plaque formation, lumen cavity I-III grade narrowing. 30ml light red fluid was visible in abdominal cavity. Regions surrounding pancrea had no visible hematocele and effusion, the surface section of pancrea was light yellow in color, no hemorrhage and necrosis were observed, the nature was moderate. Liver surface section was dark red in color, the nature was moderate. Spleen surface section was dark red

in color, the nature was moderate. Brain tissue had edema; various brain chambers had no bleeding.

3.2 Pathological Examination Results

3.2.1 Pancreas (Fig 5.6.7)

Macroscopic view: The volume was slightly greater, the mass was slightly increased, and its nature was slightly harder.

Microscopic view: Pancrea tissue structure was in existence, part of the region was autocytolysis, coagulation necrosis with infiltration of large quantity of neutrophile granulocytes were observed in the local, no hemorrhage was observed.

3.2.2 Liver (Fig 3.4)

Macroscopic view: Volume was increased slightly, the surface section was dark red in color, the local was red alternated with yellow, and no hardening change was observed.

Microscopic view: Liver cell was degenerated and necrosis with dilation and congestion of blood sinus, hemorrhage as well as infiltration of large quantity of neutrophile granulocyte and monocyte as well as inflammation cells.

3.2.3 Heart

Macroscopic view: The size of heart was normal, the crevasse of heart apex surface could not be passed through heart chambers, and the surrounding region had suspicious scar formation. The section of heart muscle was grey red in color with moderate nature. The thickness of right ventricle was 0.8cm; the tunica externa had fat-hyperplasia. Anterior descending branch of left coronary artery, main branch of left coronary artery, main branch of right coronary artery and aortic artery had visible formation of atheromatous plaque; its lumen narrowing was 70%, 50%, 10% and 1% separately, no tear and interlayer formation were observed in main aortic arch.

Microscopic view: Anterior descending branch of left coronary artery, main branch of left coronary artery, main branch of right coronary artery and aortic artery had intima thickening, cavity surface fibroplasia, hyaloids formation and fibrous cap, the lower part of fibrous cap had amorphous athero-material and cholesterol crystallization deposition with calcification. Pericardium visceral vessel was dilated with congestion as well as infiltration of relatively more inflammation cells, which were mainly composed of lymphocytes and monocytes. Visceral layer of pericardium had significant congestion, scattered small lesion hemorrhage with infiltration of inflammation cells, which were mainly composed of lymphocytes and monocytes. Tunica adventitia fat of right ventricle had hyperplasia and it was thickened and extended toward myocardial matrix, which reached 1/3-2/3 of the external layer of myocardial wall, myocardial structure was in existence, myocardial infarction was not observed, there were small-lesion shaped hemorrhage in local region.

3.2.4 Bilateral lung

Macroscopic view: the surface was smooth, which showed grey color, its nature was soft; the local nature was slightly solidified. An old scar was observed in the region below lung pleura of left lower lobe of the lung, its diameter was 1cm, and grey-white moderate-natured mass in the size of 3cm × 2cm × 2cm could be observed in region adjacent to bronchus of left lower lobe, which was fused with surrounding lymph nodes.

Microscopic view: vessel of lung interstitial tissue and alveolar wall blood capillary were dilated with congestion, power-stained edema fluid or bleeding were visible in alveolar cavity of local regions of left and right upper lung. A large quantity of large nuclear and dark stained tumor cells that were arranged in micro papilla were observed in mass adjacent to bronchus, immunohistochemical examination showed that TTF-1 (+) and there was formation of tumor embolus in vessels.

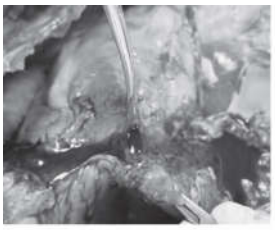
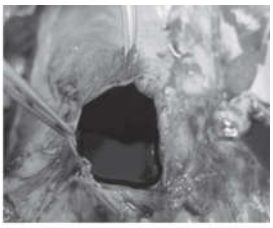


Figure 1



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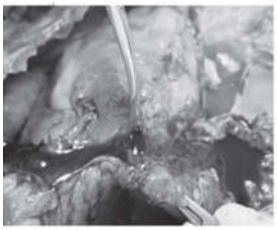

Figure 3

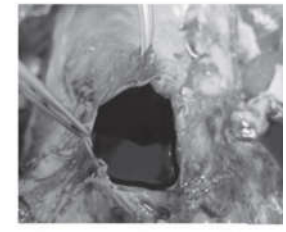

Figure 4


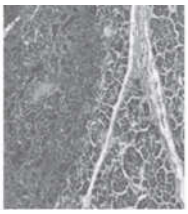
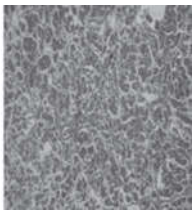

Figure 5 **Figure 6** **Figure 7**

Fig. 1 and Fig. 2 showed that the interior of pericardium cavity had large quantity of hematocele, which was about 900ml.

Fig. 3 and Fig. 4 showed degeneration and necrosis of liver cells accompanied with dilation, congestion, hemorrhage and infiltration of large quantity of neutrophile granulocytes of blood sinus, portal area was accompanied with infiltration of lymph cells and monocyte as well as other inflammation cells.

Fig. 5, Fig. 6 and Fig. 7 showed the existence of pancrea tissue structure, partial region has autolysis, coagulation necrosis with infiltration of large quantity of neutrophile granulocyte could be observed in the local, and hemorrhage was not observed.

3.3 Laboratory Examination

At 1 day prior to his death, blood draw test results showed: ① Liver function: alanine aminotransferase (ALT) 1,472 IU/L (reference value: 0-40 IU/L), asparagus aminotransferase (AST) 635IU/L (reference value: 0-40IU/L), γ-glytamyl transferase (GGT) 120U/L (reference value:0-50U/L), Lactic dehydrogenase (LDH) 2012 U/L (reference value: 104-245 U/L), HBDH 1287Iu/L (Reference value: 72-190 IU/L). ② Coagulation function: Prothrombin time (PT) 23.1S (reference value: 9-17S), international standardized ratio (PT-INR) 2.05 (reference value:0.8-1.31S), Active partial thrombin time (APTT) 46.5 (20-40 sec), fibrinogen (FBG) 1.6g/L(reference value:2.0-4.0g/L). ③ Blood routine: White blood cell (WBC) 14.5×109/L (Reference value: 4-10×109/L), Large scale platelet ratio (P-LCR) 44.40% (Reference value:13-43%）.

3.4 Autopsy Conclusion

Combined with autopsy dissection, pathological examination, laboratory examination and clinical data analysis, it was regarded that the deceased had acute pancreatitis with large quantity blood effusion in pericardial cavity, which lead to cardiac tamponade and caused acute circulation failure, then cardiac arrest and death. Coronary heart disease may exert certain facilitation effect in death process.

4. Discussion

The deceased of this case was 58 years old, the onset of di sease was acute, and he had no abdominal pain, jaundice, high fever and other symptoms when he was alive, he had sudden cardiac arrest, conscious loss, large vessel beat cease, the illness status was dangerous and he died rapidly. Autopsy dissection had found that pericardial cavity of the deceased had 900ml blood effusion, which far exceeding the 150ml diagnosis criteria of acute pericardial effusion, acute pericardial hematocele of effusion could lead to ventricle diastole suffocating, decreased cardiac output, which limiting blood returning to heart and heart beat and causing acute circulation failure as well as cardiac arrest and this is the most direct reason of death. By dissection, it was found that the deceased had relatively serious foundation disease, i.e. coronary artery artherosclerosis cardiac disease, the narrowing of anterior descending branch lumen of left coronary artery reached III grade, the narrowing of main branch lumen of left and right coronary artery also had II grade and I grade; it had artery arch artherosclerosis (atheromatous plaque), because of this, relatively more time should be spent at first sampling to find whether there was formation and break of aneurysm and interlayers, the heart itself had no break, all these were negative findings. At the same time, during sampling of lung tissue, it was found that there were swelling lymph nodes in left lung hilum, after multiple sampling and microscopic testifying, it was left lung micro-papilla type adenocarcinoma with acinous adenocarcinoma accompanied with metastasis of lung hilum and mediastinum lymph nodes, however, there was no metastasis and infiltration of pericardium. Macroscopic and microscopic observation on other organs fond that coagulation necrosis with infiltration of large quantity of neutrophile granulocytes were observed in pancrea tissue, liver cell degeneration and necrosis accompanied with dilation, congestion and hemorrhage of blood sinus as well as infiltration of large quantity of neutrophile granulocytes, portal area was accompanied with infiltration of lymph cells, monocytes as well as inflammation cells, which suggesting it had acute pancreatitis (necrosis type, no hemorrhage), acute liver necrosis. At the same time, 1 day prior to death, the laboratory examination results suggesting impairment of coagulation dysfunction, liver function impairment, it was possible that after the necrosis of pancrea tissue, large quantity of pancrea enzyme was released, which induced pericarditis and effusion by blood flow or lymph path, concurrently, it initiated the release of IL6, IL8, NO,TN-

Fœand other factors, which affected the liver function and coagulation function of organizm and thereby aggravated hemorrhage of pericardial cavity and thoracic cavity.

Severe acute pancreatitis (SAP) belongs to the special type of acute pancreatitis, which is an acute abdominal symptom with dangerous status, more comorbidity and death rate, which accounting for 10%-20% of whole acute pancreatitis. With the development of SAP surgical therapy, the curative rate was improved, however, total death rate was still as high as about 17%. The main clinical manifestations were persistent abdominal pain, jaundice, shock, high fever, breathe abnormality, consciousness change, abdominal tract hemorrhage, ascites, skin and mucosa bleeding and etc., there were few patients, especially the senile or weak patients without abdominal pain or only slight abdominal pain , which were easily to be missed in diagnosis. And senile severe pancreatitis patients themselves' organ storage function was low, often combined with one or several chronic disease, once pancrea had necrosis or infection, the illness status was usually aggravated rapidly.

Acute pancreatitis combined with fake cyst and abdominal cavity hemorrhages were relatively frequently observed in clinical practice, however, cases combined with pericardial hemorrhage was rarely encountered. Han Aijun et al[4] had found that hematocele of cardiac cavity was not coagulated but accompanied with hemolytic phenomenon; however, specific bleeding amount and pathogenesis mechanism were not reported. The bleeding amount was large in this case, it was rarely observed.

5. Conclusion

Some scholars had conducted early stage evaluation on disease cause and severity of severe acute pancreatitis, the results found that senile severe acute pancreatitis were mainly composed of gallbladder origin type or idiopathic acute pancreatitis type, furthermore, senile patients often had concurrent hypertension, coronary artery disease, diabetics and brain infarction as well as other diseases, the occurrence rate and death risk of comorbidities of whole body were higher than those with non-senile acute pancreatitis[5-8]. It is suggested that for the elderly, especially pancreatitis patients with coronary artery disease history, evaluation and prevention should be undertaken ahead of schedule, for patients who had sudden syncope, doctors should think the possibility of comorbidity with hemorrhage in pericardium cavity and attempts should be made to reduce death rate as far as possible.

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