Rare Case of Recurrent Combined Pleural and Pericardial Effusion After CABG: Difficulties in Resolving Due to Bad Socio/Economic Issues and Alcohol Abuse

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Background

Pleural effusions are present as a co-finding of lung cancer, heart failure, autoimmune disorders, in surgical practice etc. Pleural effusion is complication regularly seen in post-cardiac surgery patients. It mostly appeared 6 days after surgery with incidence around 6% [1]. The primary symptoms are fever, shortness of breath and chest pain, sometimes with lowering of haemoglobin level. Post pericardiotomy syndrome is frequently present, in 10-40% patients after heart surgery. Pleural effusions frequently showed up with pericardial effusions too and those findings prone to repeat. Certain numbers of patients require pleural and/or pericardial puncture, re-admission more than once.

Afterwards, colchicine affords suitable and proven protection while corticosteroids and non-steroidal anti-inflammatory drugs remain unclear even though some studies give us some information. It has been used during surgery received as a single intraoperative dose of 1 mg/kg methylprednisolone [2].

Pericardial effusion is even more often present according to literature [2]. More often pleural effusions were noticed when there is pericardial involvement [3]. In CABG patients when revascularization was done with LITA graft number of pleural effusions are more frequently noticed than in patients where pleural spaces stayed intact. Both kinds of effusions are also very often asymptomatic and there is no connection between ejection fraction and this postoperative event [4].

We present here a case of 71-year-old man admitted to hospital with: post MI NSTEMI (in 30 days) angina, triple vessel disease, NYHA 3-4, HTA, HLP, BMI 23, ef 50%, with alcohol abuse and bad socio-economic issues (what is of extreme importance in this case), elective surgery.

Patient underwent elective coronary artery bypass grafting, in standard fashion, on pump. Aorta coronary bypass is performed. Triple bypass, LITA graft was harvested in a skeletonized manner and vein grafts by using “no touch” technique. Pleura stayed intact in 70% of its size. Nothing special to declare during surgery, without major events during and after surgery. Patient is weaned off the CPB spontaneously without vasopressor or inotropic support. Drainage in both (pleural and pericardial space) was 650ml in the first 24h. No blood products transfused. One day in ICU, normal recovery in ward and discharged on 6th day in optimal condition, without pleural effusion and with minimal amount (5mm) of pericardial effusion on Echo-control. Patients was asymptomatic and discharged with next therapy: Beta blocker, Statin, ACE inhibitor once a day, Aspirin 100mg/die.
Almost 2 months after surgery, patient was admitted to our hospital in stable condition, without dyspnea and signs of decompensation. In the meantime, he had two hospitalizations: one in general hospital and one more 5 days prior to second admission at our institute, when was evacuated 900ml serohemorrhagic liquid from the left pleural space.

In the first postoperative hospitalization at our institute, chest roentgenoscopy revealed significant pleural effusion until the level of the fourth rib left-sided. Echocardiographic check, NYHA II, movable, asymptomatic, pleural puncture was performed and 1700ml serohemorrhagic liquid was evacuated in two consecutive days. Analysis of this liquid is done and there was low level of cholesterol and proteins in punctate. Lymphorrhea is excluded. Patient was discharged with minimal pleural effusion and without pericardial effusion, with therapeutic doses ibuprofen in the therapy see in (Figure 1-3).

One month and 15 days later (second hospitalization) patient arrived again with pleural effusion at his left pleural space. After X/ray and Echocardiography, pigtail was inserted and in 2 days 2200ml, transudate like liquid was pulled out. Punctate analysis showed again the same results. Cytology and histology analyses were negative. Lower legs in light edema. Patient was discharged on the third day.

According to data and patients’ characteristics, we expected exacerbation.

20 days after (third hospitalization) patient admitted in bad condition. This time with again left-sided pleural effusion and the first-time pericardial effusion as a dominant feature. It was massive pericardial effusion. Pericardiocentesis was performed and 1300ml of serous fluid evacuated. Pleural puncture evacuated 1100ml cases serous fluid.

Investigation in direction of lymphorrhea were negative. Cytology without pathological findings. After 9 days, patient was discharged. Control in our hospital every third days. Corticosteroids were administrated in the therapy with gastric protection.

Patient came again 15 days later (fourth hospitalization), in cardiac tamponade. Before that event, controls were every 3 days, because of specific conditions. We expected to admit him again because we did not have a chance to try colchicine as our drug of first choice and we could not keep him in the hospital permanently in expectation of new worsening. Colchicine was our drug of choice but we had to use NSAID according to recommendation [5] because in our country colchicine is not on the health care system drug list covered by national fund. Family could not afford it and alcohol addiction was our second problem with that patient.

Pericardial effusion was about 10-15mm after discharge, then went on 18-20 on the last control and after that for 2 days abruptly on 50mm circular. This time pleural effusion was insignificant. Pericardiocentesis was performed and 1700ml serous liquid evacuated.

Nonsteroidal anti-inflammatory drugs and corticosteroids were initiated, but without results because 11 days after, we admitted him in tamponade again (fifth hospitalization), close to reanimation. Urgent puncture was performed because of the progressive symptoms and size of the pericardial effusion and
approximately 1500 ml serous pericardial fluid was removed. A pigtail catheter was left in the pericardial cavity and during the following 24 hours 1650 ml blood-tinged fluid removed, subsequently. (Figure 4-5). By using pigtail, 5gr of sterile medical chock was diluted in saline and injected into the pericardial cavity to promote adhesions creation. Again, all analysis were repeated and results were in similar range. Colchicine was available and response was good.

Colchicine was administrated in loading dose 2mg/die. We decided to continue with that dose for 1 month, and after that period to reduce on 1mg /die for next 6 months. Patient had daily check with ECHO sonography and x-rays every third day. Effusion was around 10mm, 10 days in a row and patient was stable. On the 15th day, patient was discharged with a minimal effusion.

In the first month after last discharge, he had echocardiography control in every two days, later once a week and there was no effusion with hemodynamic importance.

After six months, he was stable and alive. Later Sars-Cov2 infection become general problem, so we could not see him again as we planned earlier.

Discussion

The term post-cardiac injury syndromes (PCIS) are an umbrella term indicating a group of inflammatory pericardial syndromes including post-myocardial infarction pericarditis, post-pericardiotomy syndrome (PPS) and post-traumatic pericarditis (either iatrogenic or not). Such syndromes are presumed to have an autoimmune pathogenesis triggered by initial damage to pericardial and/or pleural tissues caused by either myocardial necrosis (late post-myocardial infarction pericarditis or Dressler syndrome), surgical trauma (PPS), accidental thoracic trauma (traumatic pericarditis) or iatrogenic trauma with or without bleeding (pericarditis after invasive cardiac interventions).

Post pericardiotomy syndrome (PPS) is a common complication of cardiac operation with the incidence of at almost 20 - 40% of patient within the 6 months after initial operation. According to the last ESC guidelines [5] for treatment PPS, we administrated an aspirin in the beginning (class I level A) with no result. In further hospitalizations, classic NSAIL ibuprofen (400mg 3times a day), indomethacin 75mg per day and corticosteroids (1mg/kg) as additional therapy applied without result. Approximately 5–15% of patients with a recurrent pericarditis may have a systemic autoimmune disease (SLE, Sjogrenov syndrome, rheumatoid arthritis, sarcoidosis, tuberculosis etc) or some neoplastic syndrome. In the next hospitalisation, after pericardiocentesis we excluded autoimmune condition as a cause, in consulting with the immunologist (his blood tests result was in normal range). Performed CT scan chest and abdomen such as specific neoplastic marker and citology of pericardial effusion weren’t reveal any element for neoplastic disease. Colchicine and his anti-inflammatory effects in pericardial effusion was a subject of a many multicentric study in the last decade [6]. First publication about using a colicin in the patient with the recurrent pericardial effusion was in 1987. The main anti-inflammatory mechanism is in decreasing the granulocyte migration into inflammated area and high turnover in GI tract. The need for a loading dose in the beginning of treatment is unclear so we decided for 2g/die with a good response. Because of the low compliance (alcohol abuse, financial status), we believe that treatment of post pericardiotomy...
syndrome lasted much longer and by including colchicine in higher doses we finally resolved recurrent pericardial effusion.

Despite positive correlation of colchicine and reduced pleural and pericardial effusions [7] colchicine has good side effects on postoperative atrial fibrillation if it is administered preoperatively, but still, that is ongoing question [8].

On the global level, there is a lot of health care systems that do not have proper financial resources and this case report might be useful experience. Western countries also might get a clue how different and difficult treating of patients in developing communities might be in certain circumstances.

References