Uraemic Pericarditis

by Dr. Wan

Introduction:

� in the past, pericarditis in uraemia has been regarded as final events before death

� first noted 150 years ago in a necropsy of 100 patients who died or renal failure, Richard Bright found 37 with pericarditis and effusion

� prevalence has dropped from 51% in 1954 (Wacker and Merrill) to 10% in 1980

� dialysis has dramatically improved the prognosis of uraemic pericarditis

� a considerable patient in chronic dialysis suffer single or recurrent episodes

� major cause are intercurrent viral infections or underdialysis (e.g. insufficient fistula flow, duration or efficacy of dialysis)

Diagnosis

� often presented with precordial pain which may resemble angina

� pain usually varies with respiration and position

� cardiac arrhythmia, AF may be seen

� symptoms of congestion like dyspnoea, cough, orthopnoea

� clinical signs: fever (80%), systolic and diastolic pericardial friction rub (up to 90% ) best heard while patient sitting up and leaning forward, enlarged precordial dullness

� with development of large effusion, rub may be gone

� increased and distend jugular veins, pulsus paradoxus, hypotension, narrowed pulse pressure, Kussmaul�s sign, RUQ pain with hepatomegaly, ascites, lethargy, confusion and even coma

� low voltage in ECG not sensitive (may also show arrhythmia and non-specific ST changes, electrical alternan), increase WCC and ESR

� CXR showed globular shape heart with increase CTR

� Echocardiogram is the best, simplest and relatively easy way to diagnose pericardial effusion (easily done by bedside echo)

� CT scan and MRI can show effusion, thickened pericardium, calcifications0 and fibrinous adhesions

� clinically evident effusion occurs in 15-55% of patients with pericarditis and of those with effusions, 10-35% has cardiac tamponade

Pathology

� fibrinous pericarditis with areas of adhesions between the visceral and parietal layers distributed in patches or in a more confluent pattern

� though adherent, layers easily separated revealing numerous friable fibrinohaemorrhagic strands, the bread and butter web like appearance

� microscopically, the pericardium is haemorrhagic, erythrocytes are widely seen, mononuclear infiltrate and neovascularization

� the effusion is almost always haemorrhagic with Hct of 3-12%, does not clot, leukocytes count 400-7500/mm3

Aetiology and Pathogenesis

� for patient with acute renal failure, onset of pericarditis coincide with the severity of the azotemia, presumably due to the accumulation of the uraemic toxins (nitrogenous compounds and low molecular toxins like methylguanidine)

� the functional abnormal platelets and the repetitive impact of the myocardium against the inflamed surface contribute to the haemorrhagic effusion

� the causative factors for the pericarditis is dialyzable

� the occurrence of fever, increase WCC and ESR raised the suspicion of an infective cause

� late dialysis related pericarditis was thought to be due to an entirely different aetiology and pathogenesis but the author though that the uraemic state and/or the fluid volume and the enhanced catabolism may be the cause

Pericarditis in Renal transplantation

� the incidence in a study (1497 patients with 27 year follow up) was 2.4%

� causes attributed to uraemia, CMV infection, non-specific bacterial infection, tuberculosis and minoxidil

Treatment

� acute fibrinous pericarditis with or without small pericardial effusion can be treated conservatively

� dialysis is one of the best ways to treat patient with significant amount of effusion

� use of NSAID (e.g. indomethacin) is controversial, most authors do not recommend routine use of this drug but reserve for patient with significant fever when infective cause is ruled out

� morphine can be used for treatment of the chest pain

� aspirin and related drug should be avoided because they would worsen the bleeding tendency

� if a patient is on maintenance dialysis and develop pericarditis, one should seriously consider the adequacy of the fistula flow, a intensification of the dialysis program should be made (e.g. 5 hrs per day)

� patient with pericarditis are at high risk of cardiac tamponade when heparin is used, heparin free dialysis with high flow is recommended (Glaser et al. 1979; Sanders et al. 1985; Schwab et al. 1987)

� regional anticoagulation has been advocated but tight control is difficult and heparin rebound may occur 2-10 hrs after dialysis and bleeding frequency is considerably greater. (Swartz and Port 1979)

� minimal heparinization by using half dose is also used by some centres

� pericarditis usually resolves in 2-3 weeks time

� for large effusions with significant haemodynamic compromisation (e.g. hypotension and shock), emergency pericardiocentesis should be done (either by subxiphoid approach by needle aspiration or by LA under surgeon�s direct vision) Note the risk of coronary puncture and cardiac arrhytmogenesis with blind needle approach.

� preferred approach for resistant pericardial effusion and sub-acute effusion is by surgical pericardostomy under LA or pericardial window, pericardiectomy under GA with or without decortication of fibrotic tissue (in constrictive pericarditis)

Conclusion

� renal failure patients has a considerable risk of developing pericarditis and pericardial effusion � tamponade, so a high index of suspicion is need for its recognition and treatment

� despite the diagnosis, the morbidity and the mortality is still high, a study with 9 year follow up, 56% mortality among ESRD with pericarditis compared with 32% without

� development in the future include better way of preventing pericarditis and a more effective means of guaranteeing the survival of such patients

References:

1. Pericarditis. Rufino C. Pabico. Oxford Textbook of Clinical Nephrology.

2. Pericarditis in chronic uraemia and its sequel. Ann Intern Med 75:173-183, 1971

3. Uraemic cardiomyopathy and pericarditis. Adv Nephrol 9:33-70, 1980

4. The efficacy of indomethacin in the treatment of uraemic pericarditis Morimoto-S. Feb., 1995

5. Pericarditis following renal transplantation. Transplantation 1991. Jun; 51(6).