Uremic Pericarditis

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A 31 year old man who developed end-stage renal disease as a result of medullary cystic kidney disease was admitted to our department due to recurrent pleuritic chest pain. He was put on hemodialysis several weeks prior to this admission; however, due to poor compliance his dialysis sessions were short and insufficient. On admission he had subfebrile temperature (37.7°C); his pain was worse with deep inspiration and in the recumbent position. His physical examination was normal and no pericardial rub sounds were detected, nor was there evidence of right heart failure. However, a pulsus paradoxus of 16 mmHg was recorded. An erythrocyte sedimentation rate of 50 mm/minute was also noted.

Chest X-ray demonstrated an enlarged heart silhouette, and a complementary echocardiography showed a large pericardial effusion with signs of hemodynamic impairment manifesting as a tamponade with systolic right atrial collapse [Figure 1]. A computed tomography scan of the chest was performed and was consistent with the echo findings of an enlarged and thick pericardium [Figure 2].

Pericardial drainage was performed ("pericardial window") initially draining 950 ml of serosanguineus fluid. Pathological examination of the pericardium revealed fibrinous pericarditis.

COMMENT

Renal failure is a common cause of pericardial disease, including pericarditis and pericardial effusions. The clinical and laboratory manifestations of acute pericarditis, pericardial effusion, cardiac tamponade, and constrictive pericarditis in patients with chronic renal failure are similar to those observed in non-uremic patients with similar pericardial involvement, except that chest pain occurs less frequently in those with end-stage renal disease [1]. In renal failure patients, two forms of pericarditis have been described: The first is uremic pericarditis, observed in 6–10% of patients with advanced renal failure (acute or chronic) before dialysis is instituted or shortly thereafter [2]. Although the pathogenesis of uremic pericarditis is poorly understood, a
correlation with the degree of azotemia has been reported. Excluding the case of systemic immune disorders (such as systemic lupus erythematosus or scleroderma), there is no relationship with the underlying cause of renal failure. The second type is dialysis-associated pericarditis, which occurs in approximately 13% of patients on maintenance hemodialysis and may occasionally be seen with chronic peritoneal dialysis [3]. Two factors may contribute to this problem: inadequate dialysis and fluid overload [1-4].

Imaging techniques such as computed tomography and cardiovascular magnetic resonance are not necessary if two-dimensional Doppler echocardiography is available and informative. However, pericardial effusion is often associated with cardiac tamponade, distension of the venae cavae and hepatic veins, and deformity and compression of the cardiac chambers. CT and cardiovascular magnetic resonance imaging are indicated when hemorrhagic effusion or pericardial thickening is suspected or when findings at echocardiography are inconclusive [2-5].

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**References**

**Capsule**

*Listeria monocytogenes* can manipulate host transcription

*Listeria monocytogenes* can manipulate host transcription, and a recent study describes an underlying molecular mechanism. Lebretou et al. screened the *L. monocytogenes* genome searching for genes missing in non-pathogenic strains and identified a secreted molecule — *Listeria* nuclear-targeted protein A (LntA). The authors discovered that LntA interacted with the chromatin repressor BAHD1. To explore the functional relevance of this interaction, they infected cells in vitro with *L. monocytogenes* lacking or over-expressing LntA. Bacterial infection led to increased interferon-gamma expression, but, in the absence of LntA, BAHD1 repressed the expression of downstream interferon-stimulated genes (ISGs). In contrast, in cells infected with LntA-over-expressing bacteria, LntA kept BAHD1 from binding ISG promoters, increasing ISG expression. The results in vivo, however, disclosed a more complex picture. Heterozygous mice with one copy of BAHD1 were less susceptible to *L. monocytogenes* infection than wild-type mice, indicating that the LntA-BAHD1 interaction might modulate the host response to this bacterium. Surprisingly, wild-type mice infected with *L. monocytogenes* over-expressing LntA were also less susceptible to infection, indicating that LntA expression may be under tight regulation if the pathogen is to avoid immune attack.

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**Capsule**

Drug resistance in cystic fibrosis lungs

People with cystic fibrosis (CF) suffer from life-threatening antibiotic-resistant *Pseudomonas aeruginosa* respiratory infections, with consequent chronic inflammation, which generates oxidative stress. *P. aeruginosa* expresses several multidrug efflux systems, including the MexXY-OprM pump, which drives antimicrobial resistance in CF lungs. Expression of MexXY-OprM is unexpected, because ribosome disruption, but not antibiotics such as aminoglycosides, induces its expression, yet CF isolates exhibit high degrees of resistance to aminoglycosides. It turns out that MexXY-up-regulation depends on the gene *PA5471*, which is induced by oxidative stress. Fraud and co-workers demonstrated that exposure to inflammation-induced oxidative stress for several days produced a fourfold elevation in aminoglycoside resistance in *P. aeruginosa*, which was indeed mediated by PA5471. Aminoglycoside resistance did not always follow increased mexXY expression alone, which suggests that additional genes required for translation or protein synthesis may be involved. Thus, chronic inflammation, rather than antibiotics, drives the expression of MexXY-OprM, which leads to drug resistance in CF lungs.


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