

## CASE REPORT

# Immunological analysis of pleural fluid in post-cardiac injury syndrome

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Postgrad Med J 2002;78:362-363

Post-cardiac injury syndrome (PCIS) is an inflammatory process involving pleura and pericardium secondary to cardiac injury. Even though this clinical entity has been recognised for decades, diagnosis is difficult because of lack of a diagnostic test. Antimyocardial antibody titre in pleural fluid and serum has been proposed to have diagnostic value. However there are inherent difficulties in measuring and interpreting the role of antimyocardial antibody. A case of PCIS with low pleural fluid complement level is reported, which it is believed can be useful to support the diagnosis of PCIS.

The post-cardiac injury syndrome (PCIS) refers to pericarditis secondary to cardiac injury by various mechanisms. Diagnosis depends upon clinical suspicion and exclusion of other clinical conditions that may mimic this syndrome, such as pulmonary embolism, pneumonia, and congestive heart failure. The injury is believed to be immunological. Kim and Sahn reported immunological assessment of pleural fluid, including antimyocardial antibody testing, in a patient with PCIS.<sup>1</sup> We report the results of immunological analysis of pleural fluid in a patient with PCIS. We think that a low complement level in pleural effusion may serve as a potential diagnostic tool.

### CASE REPORT

A 60 year old man was admitted with pleuritic chest pain, fever, and shortness of breath of one days' duration. He had undergone redo coronary artery bypass graft surgery six days previously. His past medical history was significant for PCIS 10 years previously after his first coronary artery bypass graft surgery, which required the use of corticosteroids. Physical examination showed low grade fever (37.4°C), decreased breath sounds at lung bases, pleural and pericardial friction rubs. Chest radiography showed moderate size bilateral pleural effusion. A ventilation-perfusion scan was of low probability for pulmonary embolism. Significant laboratory findings

included a white blood cell count of  $19.9 \times 10^9/l$  with 72% polymorphonuclear leucocytes and 2% band forms, and high erythrocyte sedimentation rate of 71 mm/hour. Echocardiogram was unremarkable. Thoracentesis showed an exudative pleural effusion with low complement levels (table 1). His symptoms improved after treatment with indomethacin.

### DISCUSSION

PCIS is an inflammatory process involving the pleura and pericardium secondary to myocardial infarction (post-myocardial infarction syndrome) or secondary to cardiac surgery/trauma (post-cardiotomy syndrome).<sup>2</sup> The original description of PCIS by Soloff in 1953 was in patients after mitral valve commissurotomy (mitral valve commissurotomy syndrome).<sup>3</sup> Clinical features include fever, pleuritic chest pain, raised erythrocyte sedimentation rate, leucocytosis, and pleural effusion. Pleural effusion is generally exudative in nature, as described in our case also.<sup>1,4,5</sup> Prognosis is usually favourable but occasional complications may occur, which include cardiac tamponade, anaemia, premature bypass graft occlusion, unstable angina, and persistent chest pain.<sup>2</sup> Recognition of PCIS is important because of potential life threatening complications. Moreover, most patients respond to simple treatments like non-steroidal anti-inflammatory agents or corticosteroids. But lack of a diagnostic test for PCIS often leads to expensive and unnecessary diagnostic studies, undesired therapies, and sometimes complications from empiric treatment.<sup>5</sup>

Kim and Sahn suggested that a pleural fluid to serum antimyocardial antibody ratio of more than 1 could serve to support a presumptive diagnosis of PCIS.<sup>1</sup> They quoted studies describing an association between antimyocardial antibody and development of PCIS. Although it is thought that the patients with high titres of antimyocardial antibody may develop PCIS, it is difficult to interpret these studies because of differences in assay techniques. In our patient the antimyocardial antibody titres in pleural fluid and serum were <1:40. The pleural fluid to serum antimyocardial antibody ratio could not be calculated, as we could not quantify titres below 1:40. Moreover, titres below 1:40 may not be significant, and the

**Table 1** Results of pleural fluid analysis

Parameters	Pleural fluid	Serum	Comments
Glucose (mmol/l)	9.5	2.4	
Protein (g/l)	31	52	P/S ratio* = 0.59
Lactate dehydrogenase (μkat/l)	21.41	3.84	
Antimyocardial antibody titre	<1:40	<1:40	
C3 (μmol/l)	3.3	9.8	C3 index† = 0.57
C4 (μmol/l)	0.55	1.50	C4 index† = 0.62
C1q binding	Present	Absent	

Note: pleural fluid cultures were negative.

\*P/S ratio = pleural fluid/serum ratio.

†C3 or C4 index = (pleural fluid complement/serum complement) ÷ (pleural fluid total protein/serum total protein).

### Learning points

- Post-cardiac injury syndrome is an inflammatory process.
- The injury is mediated by immunological mechanisms.
- Low complement level in pleural effusion can be of diagnostic value.

presence of antimyocardial antibodies could just be a non-specific finding.<sup>6,7</sup> Because of these difficulties in interpreting the significance of antimyocardial antibodies we think that low complement level in the pleural fluid by itself may be used as a diagnostic test for PCIS in the appropriate clinical setting. Our patient had reduced C3 and C4 levels in pleural fluid, and had immune complexes as documented by the presence of C1q binding in pleural fluid. Low complement index, obtained after adjusting for total protein concentration, also indicates significant complement consumption in pleural space and suggests an immunological mechanism causing pleural effusion.<sup>1,4</sup> These results are similar to the case report published by Kim and Sahn. These findings suggest that complement is being consumed in the pleural cavity probably secondary to a local immune reaction. To the best of the authors' knowledge, in addition to the report by Kim and Sahn, there is only one another case report in a French language journal by Beynel *et al*, who reported a patient with PCIS presenting with exudative pleural effusion and low pleural fluid complement level.<sup>4</sup> Kahl *et al* and Meri *et al* described patients with PCIS showing diminished serum complement levels during early postoperative period followed by an increase in serum complement levels.<sup>8,9</sup> The increase in the complement level was considered to be an acute phase response. Similar pleural fluid complement pattern is also seen in systemic lupus erythematosus and rheumatoid arthritis.<sup>10</sup> However, pleural fluid glucose level is usually extremely low in connective tissue disorders like rheumatoid arthritis. Pleural fluid glucose level was normal in our patient. Moreover, systemic lupus erythematosus and rheumatoid arthritis can be easily ruled out with simple serological tests if clinically suspected as a cause of low complement level in pleural fluid.

### ACKNOWLEDGEMENT

The authors are greatly indebted to Drs Murali Duggirala, Ganiyu Oshodi, and Anja Bottler for their valuable contribution in the preparation of the manuscript.

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Submitted 25 July 2001

Accepted 27 February 2002

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