Fever after a pulmonary embolism: Dressler-like syndrome

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1. Case presentation

A 96-year-old man was hospitalized because of acute dyspnoea and chest pain. D-dimer levels were elevated and the chest spiral CT-scan showed signs compatible with widespread pulmonary embolism including lobar and segmental branches of the right-sided middle and lower lobes. The echocardiography revealed a moderate pulmonary arterial hypertension (56 mmHg), without evidence of left-ventricular dysfunction. Around ten days after hospitalisation, the patient presented with 38.5°C fever. The cardiopulmonary auscultation was not very helpful, except for some sub-crepitant rales. Laboratory tests showed an inflammatory process with elevated C-reactive protein levels (283 mg/L) and white blood cells (27 G/L) with 15% immature forms. We started an intravenous antibiotic therapy because we suspected a bronchopneumonia. No differential diagnosis of tuberculosis was made because of the rapidly evolving inflammatory signs and the absence of pulmonary symptoms. As the dyspnoea did not resolve and there was no sign of bronchopneumonia on the chest X-ray, the initial diagnosis appeared unlikely. The new differential diagnosis included a Dressler-like syndrome, and a follow-up echocardiography was rapidly conducted. In the absence of ORL syndrome, diarrhoea or history of swimming, no other cause (e.g. viral) of pleuropericarditis was considered. The second echocardiography detected a newly formed moderate (±500 ml) collection of intrapericardial liquid (Fig. 1), without cardiac tamponade (absence of pulsus paradoxus, no Beck’s triad – arterial hypotension, rising central venous pressure, suppressed heart sound – and no new sign on the electrocardiography as compared to what was found on admission). As we assumed a Dressler-like syndrome, we stopped the antibiotic and started a steroid treatment (prednisone 30 mg/day) rather than aspirin because of a history of upper gastrointestinal haemorrhage. The patient favourably responded to this treatment: the fever disappeared within 24-hour (Fig. 2) and the inflammation also quickly resolved. As the pericardial effusion had nearly completely resolved (Fig. 3) on the control echocardiography conducted at 1-week, we stopped steroid treatment and the inflammatory process did not recur. After several weeks of rehabilitation, the patient was able to walk with some help and could go back home with his wife.

2. Discussion

Dressler syndrome or postcardiac injury syndrome was first described by W. Dressler as an almost invariably benign, but sometimes recurrent, pericarditis that occurs two to three weeks after an acute myocardial infarction [3]. Its incidence appears to have decreased in the reperfusion era (i.e. from 5% to 0.5% in the series by Shahar et al. [4]), most likely because of the extensive use of thrombolysis and coronary angioplasty. Bendjelid and Pugin suggested that the anti-inflammatory effect of the therapy used for acute coronary syndromes, in particular, the widespread use of angiotensin converting enzyme (ACE) inhibitors and lipid lowering drugs, could also explain this important decrease [5]. This is consistent with the most commonly accepted theory that Dressler syndrome would result from the apparition of anti-heart antibodies [6,7].

The few cases of pericarditis described following pericardiectomy [8], chest trauma [9], myopericarditis [10], pacemaker implantation [11,12], percutaneous puncture of the left ventricle [13], radiofrequency ablation of idiopathic left-ventricular tachycardia [14], percutaneous mitral balloon valvuloplasty [15] and pulmonary embolism [16,18] are usually called “Dressler-like” syndromes. Clinical and biological signs of Dressler or Dressler-like syndromes are not specific. The clinical symptoms and signs include fever, malaise, chest pain, and sometimes a pericardial friction rub. Laboratory investigations usually show a varying inflammatory syndrome as well as slightly elevated troponins, creatine kinase (CK), aspartate aminotransferase (AST) and alanine

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aminotransferase (ALT), LDH, as markers of cardiac damage [19]. For cases occurring after a pulmonary embolism, the pericarditis is also believed to result from an autoimmune process, but so far the evidence is not as clear as for postmyocardial infarction syndrome, and the pericarditis is also attributed to a right-ventricular myocardial injury following the pulmonary embolism [20,21].

Primary pericarditis is a relative contraindication to therapeutic anticoagulation, which is needed for the treatment of pulmonary embolism. However, no study so far has shown that therapeutic anticoagulation in patients with pericarditis increased the risk of tamponade; in the available small case series, all patients with pericarditis following pulmonary embolism have been prescribed heparin therapy, with or without corticosteroid therapy, and no tamponade has been described [16,17,20]. Dressler-like syndrome has been reported to occur following as much as 4% of pulmonary embolisms [17] and it is important to make the proper diagnosis in order to avoid inadequate management, in particular, antibiotic therapy. Indeed, the best management is often the absence of treatment. If necessary, first line drugs are non-steroidal drugs. In some cases, steroids may be used. The course is usually benign. Steroid prophylaxis does not decrease the risk of developing postcardiac injury.

In conclusion, the prevalence of Dressler and Dressler-like syndromes is probably underestimated because of the usual benign course of this entity [22]. As they can mimic infectious disorders, these syndromes likely result in the overuse of antibiotic treatment. Clinicians should therefore keep in mind the fact that fever with inflammatory syndrome, that occurs one to two weeks after pulmonary embolism, should raise the diagnostic of Dressler-like syndrome and prompt to make an echocardiography.
Conflict of interest statement

None.

References