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 fever starting ten days after acute pulmonary embolism and severe inflammatory syndrome, but without evidence of infectious disease.

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 agents, 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 pericardiotomy [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
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.

Fig. 1. Two-dimensional echocardiography: Apical four chambers view (13th day). RA: right atrium; RV: right ventricle; LA: left atrium; LV: left ventricle; PE: pericardial effusion.

Fig. 2. Two-dimensional echocardiography: Apical four chambers view (23rd day).

Fig. 3. Temperature according to day(s) after the diagnosis of pulmonary embolism.
Conflict of interest statement

None.

References


