**Table 12-14** Causes of Pericarditis

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| **Infectious Agents** |
| Viruses |
| Pyogenic bacteria |
| Tuberculosis |
| Fungi |
| Other parasites |
| **Presumably Immunologically Mediated** |
| Rheumatic fever |
| Systemic lupus erythematosus |
| Scleroderma |
| Postcardiotomy |
| Postmyocardial infarction (Dressler) syndrome |
| Drug hypersensitivity reaction |
| **Miscellaneous** |
| Myocardial infarction |
| Uremia |
| Following cardiac surgery |
| Neoplasia |
| Trauma |
| Radiation |

invasion or direct contiguous extension into the pericardium. Histologically, serous pericarditis elicits a mild inflammatory infiltrate in the epipericardial fat consisting predominantly of lymphocytes; tumor-associated pericarditis may also exhibit neoplastic cells. Organization into fibrous adhesions rarely occurs.

**Fibrinous and serofibrinous pericarditis** are *the most frequent types of pericarditis*; these are composed of serous fluid variably admixed with a fibrinous exudate. Common causes include acute MI (Fig. 12-18*D*), postinfarction (Dressler) syndrome (an autoimmune response appearing days-weeks after an MI), uremia, chest radiation, rheumatic fever, SLE, and trauma. A fibrinous reaction also follows routine cardiac surgery.

## MORPHOLOGY

In fibrinous pericarditis the surface is dry, with a fine granular roughening. In serofibrinous pericarditis a more intense inflammatory process induces the accumulation of larger amounts of yellow to brown turbid fluid, containing leukocytes, erythrocytes, and fibrin. As with all inflammatory exudates, fibrin may be lysed with resolution of the exudate, or can become organized (Chapter 3).

Symptoms of fibrinous pericarditis characteristically include pain (sharp, pleuritic, and position dependent) and fever; congestive failure may also be present. *A loud pericardial friction rub is the most striking clinical finding.* However, the collection of serous fluid can actually prevent rubbing by separating the two layers of the pericardium.

**Purulent or suppurative pericarditis** reflects an active infection caused by microbial invasion of the pericardial space; this can occur through:

- Direct extension from neighboring infections, such as an empyema of the pleural cavity, lobar pneumonia, mediastinal infections, or extension of a ring abscess through the myocardium or aortic root
- Seeding from the blood
- Lymphatic extension
- Direct introduction during cardiotomy

The exudate ranges from a thin cloudy fluid to frank pus up to 400 to 500 mL in volume. The serosal surfaces are reddened, granular, and coated with the exudate (Fig. 12-37). Microscopically there is an acute inflammatory reaction, which sometimes extends into surrounding structures to induce *mediastinopericarditis*. Complete resolution is infrequent, and organization by scarring is the usual outcome. The intense inflammatory response and the subsequent scarring frequently produce *constrictive pericarditis*, a serious consequence (see later). Clinical findings in the active phase resemble those seen in fibrinous pericarditis, although the frank infection leads to more marked systemic symptoms including spiking fevers and rigors.

**Hemorrhagic pericarditis** has an exudate composed of blood mixed with a fibrinous or suppurative effusion; it is most commonly caused by the spread of a malignant neoplasm to the pericardial space. In such cases, cytologic examination of fluid removed through a pericardial tap often reveals neoplastic cells. Hemorrhagic pericarditis can also be found in bacterial infections, in persons with an underlying bleeding diathesis, and in tuberculosis. Hemorrhagic pericarditis often follows cardiac surgery and is occasionally responsible for significant blood loss or even tamponade, requiring re-operation. The clinical significance is similar to that of fibrinous or suppurative pericarditis.

**Caseous pericarditis** is, until proved otherwise, tuberculous in origin; infrequently, fungal infections evoke a similar reaction. Pericardial involvement occurs by direct spread from tuberculous foci within the tracheobronchial nodes. Caseous pericarditis is a common antecedent of disabling, fibrocalcific, chronic constrictive pericarditis.

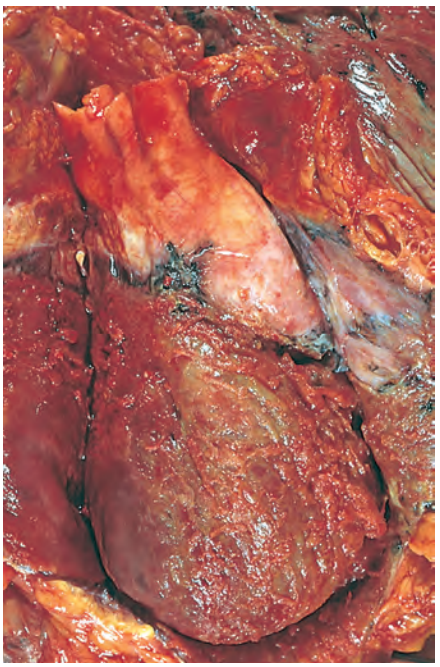

**Figure 12-37** Acute suppurative pericarditis arising from direct extension of an adjacent pneumonia. Extensive purulent exudate is evident.

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