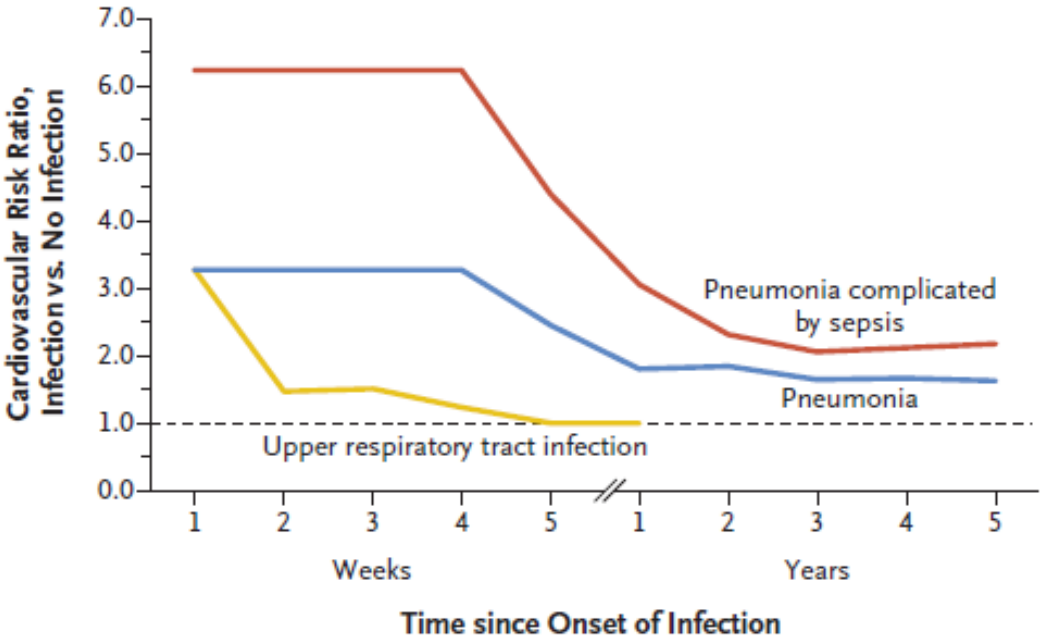

Figure 1. Temporal Pattern of Cardiovascular Risk after the Onset of Acute Infection.

The risk of a cardiovascular event is several times higher after the onset of respiratory infection than in the absence of infection. The risk of a cardiovascular event is proportional to the severity of the infection. The risk returns to baseline over a period of weeks after an upper respiratory tract infection. However, the time required for the risk to return to baseline is prolonged after a severe infection, such as pneumonia. Data are pooled from Smeeth et al.,[2] Kwong et al.,[5] Corrales-Medina et al.,[12] Warren-Gash et al.,[14] and Warren-Gash et al.[15]

一般的には2週間、肺炎では1年間は要注意でしょうか

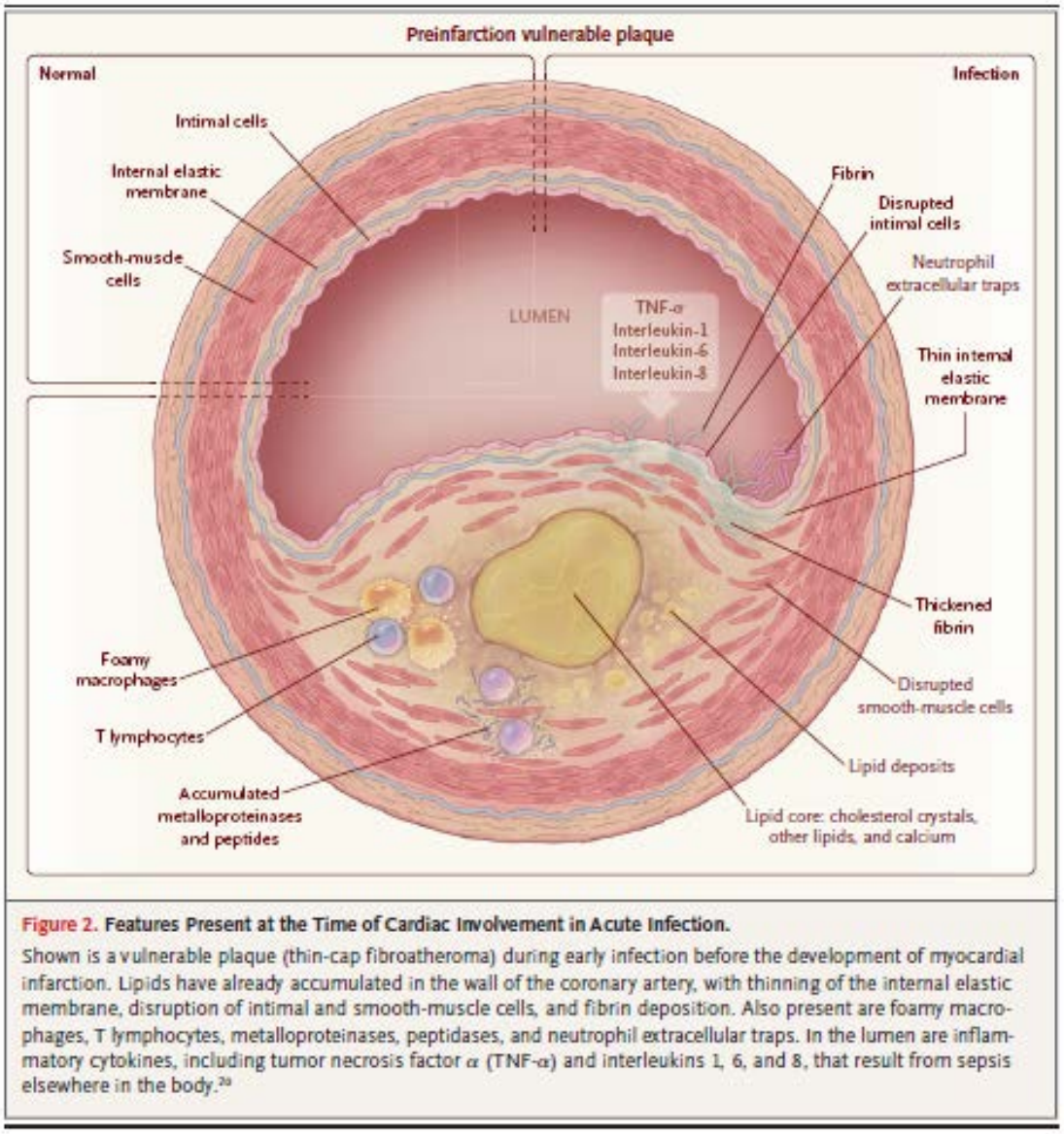

Figure 2. Features Present at the Time of Cardiac Involvement in Acute Infection.

Shown is a vulnerable plaque (thin-cap fibroatheroma) during early infection before the development of myocardial infarction. Lipids have already accumulated in the wall of the coronary artery, with thinning of the internal elastic membrane, disruption of intimal and smooth-muscle cells, and fibrin deposition. Also present are foamy macrophages, T lymphocytes, metalloproteinases, peptidases, and neutrophil extracellular traps. In the lumen are inflammatory cytokines, including tumor necrosis factor α (TNF-α) and interleukins 1, 6, and 8, that result from sepsis elsewhere in the body.[20]

プラークの中の炎症性細胞が感炎症の合併で活性化されます。

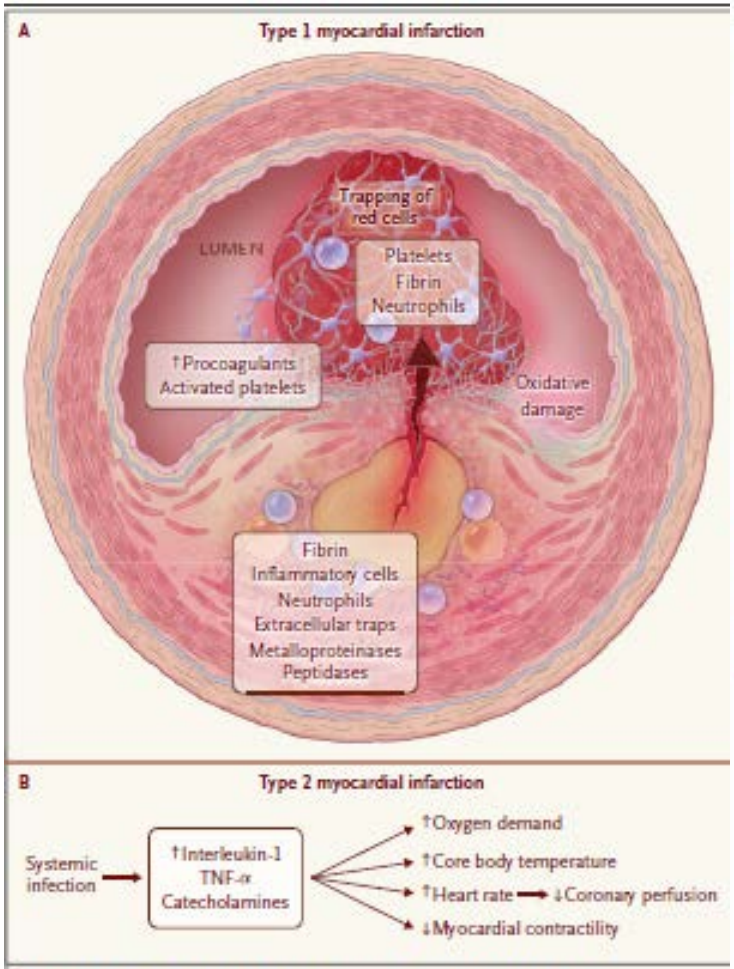

Figure 3. Mechanisms of Cardiac Involvement in Acute Infection.

Panel A shows rupture of an atheromatous plaque, the mechanism of type 1 myocardial infarction. As a result of the inflammation that develops with infection, the thin-cap atheroma ruptures, releasing inflammatory cells and fibrin into the lumen. In the presence of circulating procoagulants and activated platelets, this release causes immediate accumulation of platelets, fibrin, and neutrophils and trapping of red cells, all of which cause acute obstruction of the coronary arteries. Panel B shows the process of demand ischemia, the mechanism of type 2 myocardial infarction. Acute infection causes the release of interleukin-1, TNF-α, and catecholamines, which increase the core body temperature, oxygen demand, and heart rate. Coronary perfusion declines because of decreased filling time. Cytokines also act to suppress cardiac output. These factors, taken together, cause a mismatch of oxygen needs and oxygen supply, resulting in demand ischemia.

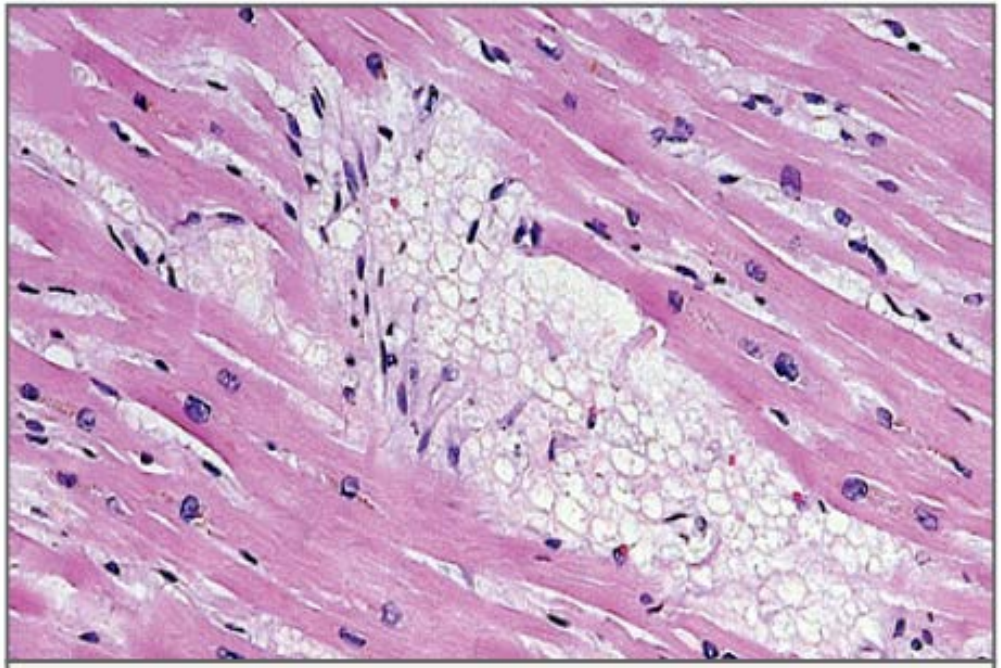

Figure 4. Features Present after Cardiac Involvement in Acute Infection.

Shown is an example of direct myocardial involvement in pneumococcal pneumonia. In the heart of a patient who was treated with antibiotic agents but still died from pneumococcal pneumonia, there are disrupted myocytes and there is a relative absence of neutrophil infiltration.[21] In addition, in experimentally induced infection and without treatment, microcolonies of *Streptococcus pneumoniae* were present.[21]

肺炎球菌の影響で心筋が変性していますが
炎症所見はなく、直接の作用です。