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Received: March 16, 1981

Accepted: September 29, 1981

Acta haemat. 1982;68:339-340

Spontaneous Anticoagulant-Induced Hemopericardium with Tamponade

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Hemopericardium secondary to traumatic laceration of the pericardium or an inflammatory process has been reported with increased frequency. In addition, bleeding into the pericardial sac during anticoagulation therapy in association with pericardial inflammation is a well-documented complication. We report here a case of hemopericardium during anticoagulant therapy with no previous pericardial injury.

A 48-year-old white male was admitted to the Department of Internal Medicine with pain in both shoulders, dyspnea on effort, and abdominal discomfort, of 2 weeks' duration. For the past 6 months he had been receiving dicoumarin (Warfarin Sodium USP) for treatment of deep vein thrombosis of the left calf. There was no history of recent trauma or fever, and he had not taken any other drugs prior to hospitalization.

On admission the patient was tachypneic, the blood pressure was 110/80 mm Hg, the pulse rate 104/min, and the temperature was 37 °C. A 'paradoxical' pulse of 20 mm Hg was recorded. There was mild jaundice in both conjunctivae, and the jugular veins were markedly congested to the level of the earlobes. Fine basilar crepitations were present, the apex beat could not be defined, and the heart sounds were barely audible. There was mild hepatomegaly, no splenomegaly or lymphadenopathy. Slight bilateral ankle pitting edema was present. The sedimentation rate was 35/50 mm (Westergren). Urinalysis revealed increased urobilinogen. Hemoglobin 13.7 g/dl, hematocrit 43%, white blood cell count 8,900/wl with a normal peripheral blood differential count. Thrombocytes were 122,000/ml, prothrombin time was 42 s (control 11s), partial thromboplastin time was 102 s (control 47 s). All the biochemical blood tests were within normal limits, with the exception of mildly disturbed liver functions. There was no evidence of either 'autoimmune disease' or any bacterial or viral infections in samples and cultures from the patient's blood, urine, saliva and feces. An electrocardiographic recording showed sinus tachycardia, low voltage QRS complexes, electrical alternans and diffuse ST-T segment changes. The chest roentgenogram showed an enlarged cardiac shadow, pulmonary congestion and a small left pleural effusion. The final diagnosis of pericardial effusion was confirmed by an echocardiographic examination. Following the correction of the bleeding tendency, a pericardiocentesis was performed which yielded 2,500 ml of bloody fluid with an hematocrit of 13%, a glucose level of 95 mg/dl and protein 5.6 g/dl with an electrophoretic pattern similar to plasma. All bacteriologic, viral and pathologic tests of this fluid were negative. 11 days later the patient was discharged in an excellent physical condition. A repeated chest

roentgenogram was normal. Repeated echocardiography showed good myocardial function with no residual pericardial fluid.

Hemorrhagic complications during anticoagulant therapy are well recognized, but the possibility of bleeding into the pericardium is not mentioned [1]. Nontraumatic bleeding into the pericardial sac is an uncommon event which most frequently occurs following acute myocardial infarction [2, 3], and dissection of an aortic aneurysm [2]. Other conditions described in association with hemopericardium and tamponade during anticoagulant

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therapy are postmyocardial infarction syndrome[4] and nonspecific [3–5] or rheumatic [6] pericarditis. A well-documented case of hemopericardium without obvious laceration of the heart or pericardium was described by Fell et al. [6]. Intraumatic and nontraumatic hemopericardium a high index of suspicion is required in order to reach the diagnosis. As seen from our case, the diagnosis of hemopericardium should be entertained in all patients receiving anticoagulants, who develop circulatory collapse for no obvious reason

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