Constrictive Pericarditis after Myocardial Infarction

Sequela of Anticoagulant-Induced Hemopericardium

AMIN H. KARIM, M.D., M.R.C.P.*
JOSEPH SALOMON, M.D., F.A.C.P.
Baltimore, Maryland

Hemopericardium developed following adequate (and not excessive) anticoagulation in a patient with acute transmural anterior myocardial infarction and an early pericardial friction rub. Drainage of a bloody effusion by pericardiocentesis did not prevent progression to constrictive pericarditis within one year. The clinical features of this case and of the only two other similar occurrences reported in the medical literature are described and lead to a discussion of therapeutic and preventive measures.

Hemopericardium and subsequent constrictive pericarditis are a rare sequela of anticoagulation for acute myocardial infarction. Only two reports of that nature have appeared in the medical literature [1,2]. We have encountered a similar case that allows us to describe characteristic clinical features and to suggest preventive and therapeutic measures.

CASE REPORT

A 52-year-old Caucasian man was admitted to Sinai Hospital of Baltimore in January 1976 with an extensive anterior myocardial infarction, and was noted to have a pericardial friction rub on the second hospital day. He underwent prophylactic anticoagulation, having also had varicose veins in the lower extremities. Twenty-four thousand units of heparin sulfate were administered daily and were followed two days later by warfarin sodium in doses that maintained the prothrombin time at about twice the control value; the longest prothrombin time was 22 seconds. A large pericardial effusion was found on the seventh hospital day, although the activated partial thromboplastin time and the prothrombin time were within the therapeutic range. Three hundred fifty milliliters of sterile nonclotting sanguineous fluid were drained by pericardiocentesis. The hospital course was otherwise uncomplicated and the patient was released free from symptoms; his medications on discharge were digoxin, furosemide, and quinidine sulfate. He had no clinical or laboratory evidence of post-myocardial infarction syndrome, cardiac tamponade, or congestive heart failure, either in the hospital or following discharge.

The patient was rehospitalized one year later, in January 1977, because of the insidious development of ascites and pedal edema. His blood pressure was 105/70 mm Hg and there was no "paradoxic pulse." His heart rate was regular at 82 beats per minute. The jugular venous pressure was elevated at 15 cm of water and was also notable for a very steep Y descent (**Figure 1**). The first and second heart sounds were distant and were followed by an early third heart sound. Electrocardiography revealed evidence of an old anterior myocardial infarction. Echocardiography showed a thickened posterior pericardium and no pericardial effusion. Cardiac catheterization revealed the following intracardiac pressures: right atrium 17 mm Hg; right ventricle 32/16 mm Hg; pulmonary artery 32/19 mm Hg; pulmonary capillary wedge 20 mm Hg; left ventricle 85/19 mm Hg. The aortic pressure was low,

From the Department of Medicine and the Division of Cardiology, Sinai Hospital of Baltimore, Belvedere at Greenspring, Baltimore, Maryland, and The Johns Hopkins School of Medicine, Baltimore, Maryland. Manuscript accepted July 19, 1984.
* Current address and address for reprint requests: Department of Cardiology, Baylor College of Medicine, The Methodist Hospital, 6535 Fannin, Suite 905, Houston, Texas 77030.

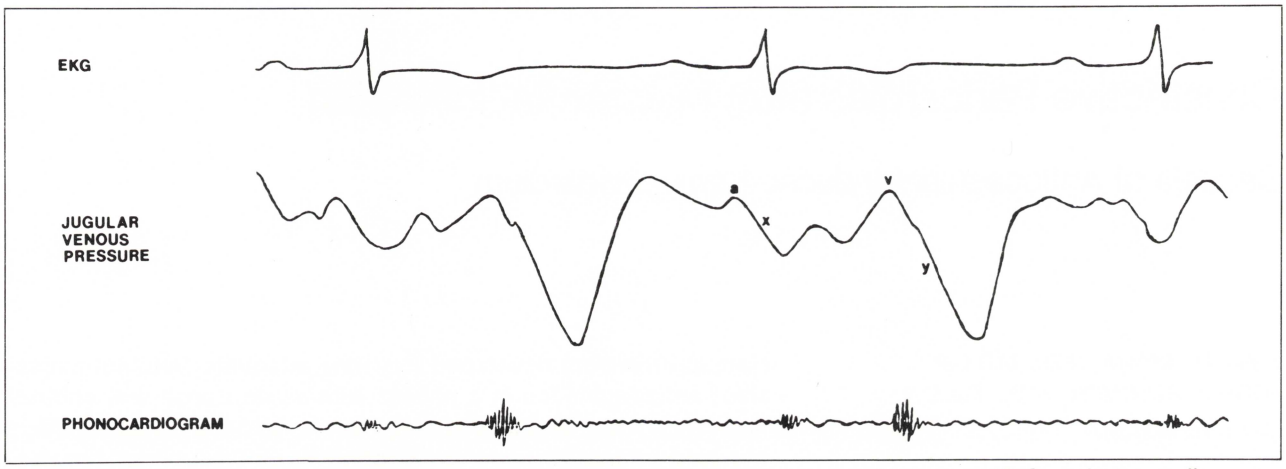

Figure 1. *Jugular venous pressure tracing prior to pericardiectomy shows a steep Y descent. EKG = electrocardiogram.*

85/60 mm Hg. The equalization of pressures in the cardiac chambers during diastole, a "dip and plateau" pattern in the right ventricle, and the steep jugular Y descent (Figure 1) were suggestive of constrictive pericarditis.

The patient underwent rather complete pericardiectomy. The pericardium was almost 1 cm in thickness and consisted primarily of fibrous tissue. The pressure in the right atrium and in the pulmonary artery returned to normal intraoperatively. The postoperative course was uneventful and the patient has had no manifestations of cardiac compression or failure to date.

COMMENTS

Although intrapericardial bleeding is not a very infrequent complication of anticoagulation following acute myocardial infarction [3,4], progression to chronic constriction is rare. Laszlo [1] was the first to report constrictive pericarditis six months following myocardial infarction in a patient who underwent excessive anticoagulation with bishydroxycoumarin and in whom hemopericardium developed during the acute phase of the ischemic event; this patient had a pericardial friction rub early in the course of the infarction. Beaufils et al [2] described a patient who had manifestations of Dressler's syndrome five days following an acute myocardial infarction; he received an oral anticoagulant one month later, was found to have hemopericardium after three additional months, and presented with pericardial constriction five months later. The three patients, ours as well as those described by Laszlo [1] and Beaufils et al [2], have striking similarities. All had evidence of pericardial involvement early in the course of the myocardial infarction and had hemopericardium following prophylactic anticoagulation before finally presenting with constrictive pericarditis six to 12 months later. The intrapericardial bleeding was probably facilitated by the pericardial reaction, since our patient and the one described by Beaufils et al [2] had no excessive prolongation of the prothrombin time.

The rarity of this complication is especially striking considering the high prevalence of myocardial infarction in Western civilization, the not infrequent involvement of the pericardium (in the form of an early friction rub [5] or a late Dressler's syndrome [6]), and the relatively common practice of post-infarction anticoagulation. Although progression to pericardial constriction is rare, the few reported cases underscore the risk of prophylactic anticoagulation in patients with acute myocardial infarction who have evidence of pericardial involvement, a practice that should be discouraged. Treatment with anti-inflammatory agents, even in the absence of clinical stigmata of pericarditis, should be considered as a potential therapeutic means of preventing evolution of late constrictive pericarditis. Awareness of these early and late sequelae of anticoagulation in the setting of acute myocardial infarction is important, to avoid confusion with congestive heart failure and to assure proper therapeutic considerations.

REFERENCES

1. Laszlo MH: Constrictive pericarditis as a sequel to hemopericardium; report of a case following anti-coagulant therapy. Ann Intern Med 1957; 46: 403–413.
2. Beaufils P, Bardet J, Jemkine J, et al: Syndrome de Dressler: pericardite constrictive post-infarctus operee avec success. Arch Mal Coeur 1975; 68: 651–656.
3. Wright ES, Maple CD, Beck DF: Myocardial infarction: the clinical manifestations and treatment with anti-coagulants—a study of 1,031 cases. New York: Grune and Stratton, 1954.
4. Anderson NW, Christensen NA, Edwards JE: Hemopericardium complicating myocardial infarction in the absence of cardiac rupture. Arch Intern Med 1952; 90: 634–645.
5. Sternberg M: Pericarditis epistenocardia. Wien Med Wochenschr 1910; 60: 14–23.
6. Dressler W: A post-myocardial infarction syndrome. JAMA 1956; 160: 1379–1383.

Reprinted from the September issue of **The American Journal of Medicine,** A Yorke Medical Journal. Published by Technical Publishing, a Division of Dun-Donnelley Publishing Corporation, a Company of the Dun & Bradstreet Corporation, 875 Third Avenue, New York, N.Y., 10022.

Constrictive Pericarditis after Myocardial Infarction

Sequela of Anticoagulant-Induced Hemopericardium

AMIN H. KARIM, M.D., M.R.C.P.*
JOSEPH SALOMON, M.D., F.A.C.P.
Baltimore, Maryland

Hemopericardium developed following adequate (and not excessive) anticoagulation in a patient with acute transmural anterior myocardial infarction and an early pericardial friction rub. Drainage of a bloody effusion by pericardiocentesis did not prevent progression to constrictive pericarditis within one year. The clinical features of this case and of the only two other similar occurrences reported in the medical literature are described and lead to a discussion of therapeutic and preventive measures.

Hemopericardium and subsequent constrictive pericarditis are a rare sequela of anticoagulation for acute myocardial infarction. Only two reports of that nature have appeared in the medical literature [1,2]. We have encountered a similar case that allows us to describe characteristic clinical features and to suggest preventive and therapeutic measures.

CASE REPORT

A 52-year-old Caucasian man was admitted to Sinai Hospital of Baltimore in January 1976 with an extensive anterior myocardial infarction, and was noted to have a pericardial friction rub on the second hospital day. He underwent prophylactic anticoagulation, having also had varicose veins in the lower extremities. Twenty-four thousand units of heparin sulfate were administered daily and were followed two days later by warfarin sodium in doses that maintained the prothrombin time at about twice the control value; the longest prothrombin time was 22 seconds. A large pericardial effusion was found on the seventh hospital day, although the activated partial thromboplastin time and the prothrombin time were within the therapeutic range. Three hundred fifty milliliters of sterile nonclotting sanguineous fluid were drained by pericardiocentesis. The hospital course was otherwise uncomplicated and the patient was released free from symptoms; his medications on discharge were digoxin, furosemide, and quinidine sulfate. He had no clinical or laboratory evidence of post-myocardial infarction syndrome, cardiac tamponade, or congestive heart failure, either in the hospital or following discharge.

The patient was rehospitalized one year later, in January 1977, because of the insidious development of ascites and pedal edema. His blood pressure was 105/70 mm Hg and there was no "paradoxic pulse." His heart rate was regular at 82 beats per minute. The jugular venous pressure was elevated at 15 cm of water and was also notable for a very steep Y descent (**Figure 1**). The first and second heart sounds were distant and were followed by an early third heart sound. Electrocardiography revealed evidence of an old anterior myocardial infarction. Echocardiography showed a thickened posterior pericardium and no pericardial effusion. Cardiac catheterization revealed the following intracardiac pressures: right atrium 17 mm Hg; right ventricle 32/16 mm Hg; pulmonary artery 32/19 mm Hg; pulmonary capillary wedge 20 mm Hg; left ventricle 85/19 mm Hg. The aortic pressure was low,

From the Department of Medicine and the Division of Cardiology, Sinai Hospital of Baltimore, Belvedere at Greenspring, Baltimore, Maryland, and The Johns Hopkins School of Medicine, Baltimore, Maryland. Manuscript accepted July 19, 1984.
* Current address and address for reprint requests: Department of Cardiology, Baylor College of Medicine, The Methodist Hospital, 6535 Fannin, Suite 905, Houston, Texas 77030.

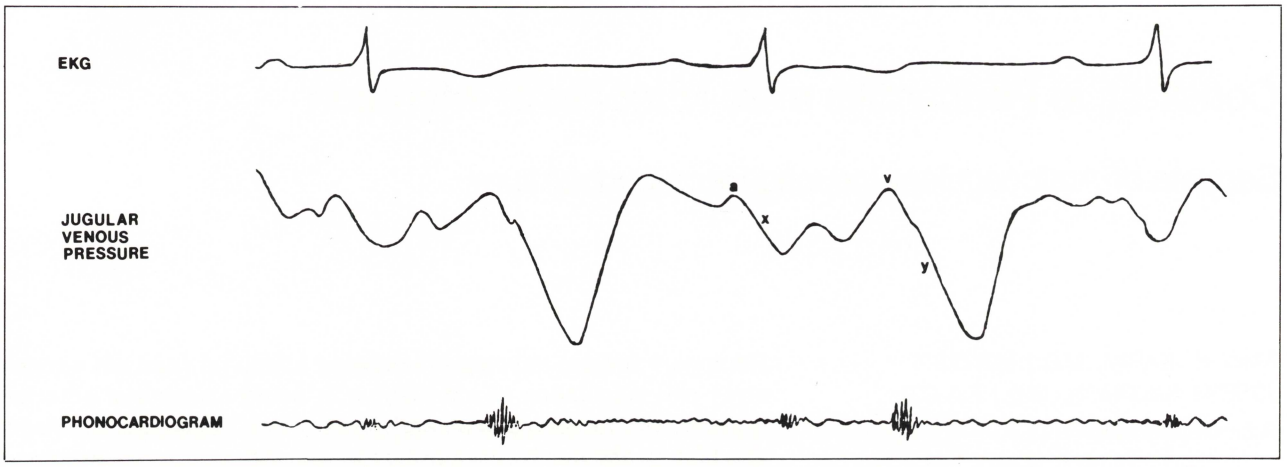

Figure 1. *Jugular venous pressure tracing prior to pericardiectomy shows a steep Y descent. EKG = electrocardiogram.*

85/60 mm Hg. The equalization of pressures in the cardiac chambers during diastole, a "dip and plateau" pattern in the right ventricle, and the steep jugular Y descent (Figure 1) were suggestive of constrictive pericarditis.

The patient underwent rather complete pericardiectomy. The pericardium was almost 1 cm in thickness and consisted primarily of fibrous tissue. The pressure in the right atrium and in the pulmonary artery returned to normal intraoperatively. The postoperative course was uneventful and the patient has had no manifestations of cardiac compression or failure to date.

COMMENTS

Although intrapericardial bleeding is not a very infrequent complication of anticoagulation following acute myocardial infarction [3,4], progression to chronic constriction is rare. Laszlo [1] was the first to report constrictive pericarditis six months following myocardial infarction in a patient who underwent excessive anticoagulation with bishydroxycoumarin and in whom hemopericardium developed during the acute phase of the ischemic event; this patient had a pericardial friction rub early in the course of the infarction. Beaufils et al [2] described a patient who had manifestations of Dressler's syndrome five days following an acute myocardial infarction; he received an oral anticoagulant one month later, was found to have hemopericardium after three additional months, and presented with pericardial constriction five months later. The three patients, ours as well as those described by Laszlo [1] and Beaufils et al [2], have striking similarities. All had evidence of pericardial involvement early in the course of the myocardial infarction and had hemopericardium following prophylactic anticoagulation before finally presenting with constrictive pericarditis six to 12 months later. The intrapericardial bleeding was probably facilitated by the pericardial reaction, since our patient and the one described by Beaufils et al [2] had no excessive prolongation of the prothrombin time.

The rarity of this complication is especially striking considering the high prevalence of myocardial infarction in Western civilization, the not infrequent involvement of the pericardium (in the form of an early friction rub [5] or a late Dressler's syndrome [6]), and the relatively common practice of post-infarction anticoagulation. Although progression to pericardial constriction is rare, the few reported cases underscore the risk of prophylactic anticoagulation in patients with acute myocardial infarction who have evidence of pericardial involvement, a practice that should be discouraged. Treatment with anti-inflammatory agents, even in the absence of clinical stigmata of pericarditis, should be considered as a potential therapeutic means of preventing evolution of late constrictive pericarditis. Awareness of these early and late sequelae of anticoagulation in the setting of acute myocardial infarction is important, to avoid confusion with congestive heart failure and to assure proper therapeutic considerations.

REFERENCES

1. Laszlo MH: Constrictive pericarditis as a sequel to hemopericardium; report of a case following anti-coagulant therapy. Ann Intern Med 1957; 46: 403–413.
2. Beaufils P, Bardet J, Jemkine J, et al: Syndrome de Dressler: pericardite constrictive post-infarctus operee avec success. Arch Mal Coeur 1975; 68: 651–656.
3. Wright ES, Maple CD, Beck DF: Myocardial infarction: the clinical manifestations and treatment with anti-coagulants—a study of 1,031 cases. New York: Grune and Stratton, 1954.
4. Anderson NW, Christensen NA, Edwards JE: Hemopericardium complicating myocardial infarction in the absence of cardiac rupture. Arch Intern Med 1952; 90: 634–645.
5. Sternberg M: Pericarditis epistenocardia. Wien Med Wochenschr 1910; 60: 14–23.
6. Dressler W: A post-myocardial infarction syndrome. JAMA 1956; 160: 1379–1383.

Reprinted from the September issue of **The American Journal of Medicine,** A Yorke Medical Journal. Published by Technical Publishing, a Division of Dun-Donnelley Publishing Corporation, a Company of the Dun & Bradstreet Corporation, 875 Third Avenue, New York, N.Y., 10022.