Transient Myocardial Damage Secondary to Extravasation of Contrast Material during Left Ventricular Angiocardiography

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RECENT improvements of technics of left heart catheterization have permitted more frequent use of selective left ventricular angiocardiography. Extravasation of contrast material into the myocardium is a rather common complication of intracardiac injection. However, only one previous report of possible myocardial damage from such extravasation has come to our attention. The patient reported here developed myocardial damage after the entry of contrast material into the left ventricular wall. Signs and symptoms of myocardial damage, however, almost disappeared within 24 hours, and there were no serious aftereffects.

Case Report

G.L., a 44-year-old white man, complained of recurrent attacks of dizziness and one episode of syncope during the past year. For the past 8 months, the patient also had discomfort in the lower chest, radiating to the right shoulder and arm, precipitated at times by exertion. Progressive exertional dyspnea developed during the 6 months prior to admission to the Boston City Hospital for cardiac evaluation.

On examination, the patient measured 67 inches in height and weighed 178 pounds. The blood pressure was 160/100 in the right arm sitting, and 180/130 in the right leg supine. There was no cyanosis or clubbing. An A wave was evident in the jugular vein, which was not distended. The lungs were clear to percussion and auscultation. No cardiac heave or thrill was felt. The aortic closure sound was heard quite well.

There was a grade-II/VI short systolic ejection murmur, heard best along the left sternal border, radiating to the neck and the apex.

The electrocardiogram showed normal sinus rhythm at 80, with a PR interval of 0.13 second and a QRS interval of 0.09 second. The tracing was interpreted as within normal limits. The chest x-ray film revealed slight cardiac enlargement with a contour compatible with left ventricular hypertrophy. No intracardiac calcification was seen on fluoroscopic examination. The clinical diagnosis was aortic stenosis of uncertain location and severity.

On September 19, 1963, a diagnostic right and left cardiac catheterization and cineangiograms were performed. In the fasting state and without sedation, a routine right heart catheterization was carried out with a no.-9 Courmand catheter via the left median basilic vein. Percutaneous catheterization of the right femoral vein was then performed, and the left atrium was entered transseptally with a Brockenbrough Teflon catheter, size 8 F, with a distal tip tapering to 0.035 inch ID. The catheter was advanced into the left ventricle. The findings indicated moderate aortic stenosis; subsequent analysis of left ventricular and brachial arterial pressure tracings, recorded during extrasystoles, after administration of Isuprel, and in close proximity to measurements of cardiac output by indicator dilution, indicated valvular aortic stenosis, with a mean systolic pressure gradient of 18 mm. Hg across the aortic valve, and an estimated aortic valve area of 1.5 cm.² Lastly, in the left anterior oblique position, 35 ml. of 85 per cent diatrizoate methylglucamine (Cardiografin) were injected into the left ventricle under pressure (Taveras injector set at 525 psi) in less than 3 seconds. A test dose of the contrast material had been given intravenously in advance without untoward effect. The catheter was freely movable in the left ventricular cavity, although the upstroke limb of the left ventricular pressure tracing was subsequently noted to be slightly distorted (fig. 1B) compared with the recording obtained when the catheter first entered the left ventricle (fig. 1A). The patient was breathing 100 per cent oxygen by mask while the injection was being made. The

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cineangiogram was monitored by mirror (Picker 9-inch image intensifier), and it was noted that contrast material entered the posterior wall of the left ventricle and persisted. Immediately following the injection, the patient developed nausea, vomited, and complained of upper anterior chest pain radiating to the neck. Idioventricular rhythm at a rate of 91 beats per minute predominated for about 5 minutes after the injection; the left ventricular pressure obtained through the catheter was slightly lowered (fig. 1C). About 8 minutes after the injection, normal sinus rhythm at a rate of 68 per minute was restored spontaneously. The ST segments in lead II were now found to be elevated, and left ventricular pressure had returned to the pre-injection level (fig. 1D). The patient was carefully monitored following this event, but felt well except for discomfort in the upper chest, which gradually subsided within 1 hour. The ST segment elevation became less remarkable, and the blood pressure remained normal.

Analysis of the cineangiograms revealed that some contrast material entered left ventricular muscle posteriorly, through the tip of the catheter, while opaque medium filled the left ventricular cavity, presumably through the side holes (fig. 2A). The extravasated opaque medium then rapidly spread upwards and laterally to the border of the cardiac silhouette (fig. 2B and C), where the contrast substance remained for 7 minutes after the injection, while no opaque medium was seen elsewhere (fig. 2D). Ten minutes after the injection, however, contrast medium was no longer demonstrable on the posteroanterior film. In addition to the extravasation of the contrast material, the cineangiogram showed a definitely thickened left ventricular wall. The left ventricular outflow tract was not remarkable, and there was no poststenotic dilatation of the aorta.

The serum glutamic oxaloacetic transaminase levels were 73 units at 12 hours after catheterization, 80 units at 24 hours after catheterization, and 22 units 7 days after catheterization (normal values for this laboratory range from 8 to 40 units). The electrocardiogram taken 24 hours after the catheterization still showed inversion of the T wave in leads I, II, aV_L, V_5, and V_6 suggestive of lateral wall ischemia (fig. 3B). A chest x-ray film taken at the same time demonstrated no residual contrast material and no other interval change. The recovery was quite uneventful. The electrocardiogram taken a week later (fig. 3C) had almost returned to the normal control (fig. 3A). The patient has been observed for a year since the procedure, and has remained well.

Discussion

Hilbish and Herdt

Figure 1

Serial left ventricular pressure tracings (above), and lead II of the electrocardiogram (below). A. Immediately after the first entry of the catheter into the ventricle. The upstroke limb is smooth. B. Immediately before the injection of the contrast material. The upstroke limb is distorted as compared with A. C. Immediately after the injection. Idioventricular rhythm predominates and lasts for 5 minutes. The left ventricular pressure is slightly lower than before injection. D. Eight minutes after the injection, sinus rhythm has become restored, but the S-T segment is elevated. Note that left ventricular pressure has returned to the pre-injection level, but the tracing remains abnormal in contour.
Four single frames of the 35-mm. cineangiogram, using Eastman Double-X panchromatic film, taken at 30 frames per second, in the left anterior oblique position. A, upper left. 0.6 second after the onset of injection the contrast medium is seen to penetrate the inferior and posterior wall of the left ventricle, but also fills the left ventricular cavity. Note the thick left ventricular wall. B, upper right. 2.7 seconds after the onset of injection the contrast medium in the myocardium is seen to extend further upwards and outwards, outlining a portion of the left ventricle. The cavity of the left ventricle and the ascending aorta are also filled. C, lower left. 5.8 seconds after the onset of injection, the contrast medium in the left ventricular wall has migrated further upwards and outwards, approaching epicardium and clearing the inner layer of the wall. D, lower right. Cinefluorogram 7 minutes after the injection shows residual contrast medium limited to the subepicardium of the superior and posterior left ventricular wall.

However, that adverse clinical reactions are rare, with the exception of the cases in which injected contrast material enters the pericardial sac resulting in cardiac tamponade.1,3,6 Usually, transitory cardiac arrhythmias are the only clinical manifestation of intramural extravasation. Cardiac arrhythmias are also seen rather frequently during intracardiac injection without any evidence of extravasation of the contrast material. In the early 1950’s, periph-
eral venous injection of contrast material was at times followed by ST and T-wave changes in the electrocardiogram.\(^7\)\(^-\)\(^9\) In one previous instance\(^2\) substernal pain and elevation of the ST segments in the electrocardiogram were reported following left ventricular angiography performed by means of direct transthoracic puncture of the left ventricle.

In the present case, the development of upper anterior chest pain, the electrocardiographic changes, and the increase in serum transaminase must be interpreted as indicative of transient myocardial damage produced by the entry of contrast material into the posterolateral wall of the left ventricle, although elevation of serum glutamic oxaloacetic transaminase may occasionally be seen after cardiac catheterization and angiography without any complication.\(^10\) Recently, Keates and Wagner\(^6\) reported a case of perforation of the heart during selective right ventricular angiography, without any serious late complication. They noted a distorted right ventricular pressure tracing prior to injection, and concluded from this observation that the tip of the catheter must have been embedded in the myocardium and that the side holes of the catheter were not free.

Our case resembles this one, although the thick left ventricular wall prevented the dispersion of opaque medium into the pericardial sac. It is quite possible that a Brockenbrough catheter with tapered tip may become embedded in the myocardium during an injection, as has been described previously for other catheters. The present case represents the only instance of extravasation encountered in a total of 171 cineangiograms, including 24 selective left ventricular cineangiograms, carried out in our laboratory during the past 4 years. With the increasing frequency of selective left ventricular angiography, the complication reported here may be encountered more often. Careful positioning of the catheter tip, monitoring of the pressure tracing, and use of the loop catheter via retrograde approach may prevent this complication. When doubt arises as to the proper position of the catheter, the manual injection of a small amount of contrast material with direct observation may be helpful.

**Summary**

In a patient with moderate valvular aortic stenosis transient myocardial injury resulted

### Figure 3

Twelve-lead electrocardiogram (10 mm. of paper = 1 mV.) taken (A), before catheterization, at the rate of 83 per minute; (B), 24 hours after catheterization, at the rate of 100 per minute; (C), a week later at the rate of 86 per minute.
from extravasation of contrast material into the posterolateral wall of the left ventricle during selective left ventricular cineangiocardiography via the transfemoral approach. The patient developed anterior upper chest pain, runs of idioventricular rhythm followed by spontaneous return to sinus rhythm with elevated ST segments, and increase in serum transaminase levels. Lack of previous reports of transient myocardial damage secondary to extravasation of contrast material, increased possibility of such accidents with increased use of selective left ventricular angiocardiography, and possible preventive methods were emphasized.

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**Emergency**

With whatever romantic notions we enter upon the practice of medicine, we shall probably find when we are actually engaged in it that but a small part of our time is taken up by the desperate or even the moderately urgent emergency. Nevertheless, it may be said without undue straining of the phrase that in a certain sense the whole of a doctor's life is passed in a medium from which the pressure of emergency is never remote. He may be described, perhaps not too extravagantly, as living to some degree like a soldier in an unfriendly country, where his whole behaviour must be alert and circumspect, and his reaction to events under careful control.—*The Collected Papers of Wilfred Trotter*, F.R.S. London, Oxford University Press, 1946, p. 1.
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