Transient predominant right ventricular ischemia caused by coronary vasospasm

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ABSTRACT This study describes the clinical experience with four patients with variant angina caused by spasm of the right coronary artery who were assessed for evidence of right ventricular involvement. The patients were suspected of having predominant right ventricular ischemia on the basis of normal thallium-201 scans, left ventricular ejection fraction, regional wall motion assessed by equilibrium radionuclide angiography (RNA), two-dimensional echocardiographic findings, and left ventricular hemodynamics; all procedures were performed during transient ST segment elevation in the inferior leads. Right ventricular ischemia was documented in four patients by first-pass radionuclide studies and phase analysis of RNA, and in three patients by simultaneous right and left hemodynamic monitoring. The clinical findings from these four patients are compared with those from four other patients with similar electrocardiographic changes, coronary anatomic distribution, and documented right coronary spasm but with evidence of left ventricular involvement as documented by abnormal thallium-201 scintigraphy, RNA, two-dimensional echocardiography, and left hemodynamics during ischemic episodes. Although preliminary, these data indicate the existence of prevalent right ventricular ischemia during variant angina caused by right coronary vasospasm. This condition should be suspected whenever typical anginal symptoms and/or ischemic electrocardiographic changes are accompanied by normal thallium-201 scintigraphic findings and/or normal left ventricular function as assessed by RNA, echocardiography, and left hemodynamic monitoring. Among noninvasive procedures, first-pass radionuclide study and phase analysis of RNA represent suitable techniques for detecting transient right ventricular dysfunction.


CORONARY VASOSPASM is a well-recognized cause of transient ischemia at rest.1 2 Correlation among clinical, electrocardiographic, and angiographic findings has demonstrated that the clinical spectrum of this syndrome is polymorphic and widely variable.2 Radioisotopic,3–5 echocardiographic,6,7 and hemodynamic8,9 studies have characterized the patterns of left ventricular perfusion and wall motion abnormalities during resting angina. At present, few data on right ventricular involvement during vasospastic angina are available and are strictly confined to hemodynamic monitoring performed in patients with frequent ischemic attacks at rest.8

In the past few years, great interest has arisen about right ventricular involvement in the course of acute inferior myocardial infarction, and several authors have demonstrated the existence of predominant right ventricular infarction.10–20

We describe the clinical features of four patients with Prinzmetal’s angina characterized by ST segment elevation in the inferior leads caused by spasm of the right coronary artery (RCA) and evidence of predominant right ventricular impairment.

Methods

Patients. From July 1982 to July 1983, 131 patients with diagnoses of unstable angina at rest were admitted to our coronary care unit. Thirty-three were diagnosed as having a variant form of angina. Fourteen of 33 showed ST segment elevation in the inferior leads during typical attacks at rest. Among them, four men (ages 55 to 61 years) are the subjects of this study. The criteria for selection were (1) episodes of variant angina of short duration (less than 5 min), (2) angiographically documented dominant RCA, (3) no clinical evidence of previous myocardial infarction, and (4) positive results of ergonovine provocative testing and fast resolution of the ischemic episode after nitrate administration. For comparison with the findings in this group of patients with evidence of predominant right ventricular impairment, this report also includes the findings of four additional patients selected on the basis of the above criteria but with evident left ventricular dysfunction.
Exercise tolerance was assessed in all patients by the bicycle stress test. Frequent blood samples for creatine kinase MB analysis and daily basal 12-lead electrocardiograms (ECGs) were obtained to monitor the possibility of acute myocardial infarction during hospitalization. Long-acting nitrates were discontinued 12 hr before each study, and nitroglycerin was allowed in case of anginal attacks. No other drug was used during the study period. All patients gave informed, written consent to participate in the study protocol.

**Hemodynamic and coronary angiographic studies.** All patients underwent left ventricular hemodynamic evaluation. Selective coronary angiograms in multiple views of both right and left coronary systems, and biplane left ventriculograms were performed with the Judkins technique. Left ventricular pressure was then measured via a fluid-filled pigtail catheter and monitored during the ergonovine test until obvious electrocardiographic ischemic changes occurred. Soon after the removal of the pigtail catheter, an RCA catheter was quickly positioned in the right coronary orifice to obtain angiograms during ischemia. Angiograms were interpreted by an experienced cardiologist not involved in this study. Furthermore, in three patients right ventricular pressure was simultaneously monitored during the angiographic studies. Pressure signals as well as two-lead ECGs (III and V2) were recorded on a multichannel photographic recorder (EP12; OTE Biomedica, Italy).

**Noninvasive procedures.** Myocardial perfusion and wall motion were studied in different conditions by thallium-201 scintigraphy, radionuclide angiography (RNA), first-pass RNA, and two-dimensional echocardiography.

The scintigraphic data were collected with a standard large-field gamma camera (Selo KR7, Italy) equipped with a low-energy, high-resolution collimator and interfaced with a dedicated computer system (Medusa 12B; Sepa, Italy).

Thallium-201 was injected intravenously during spontaneous or ergonovine-induced attacks with typical electrocardiographic changes. Scintigrams were obtained within 8 min after thallium-201 injection and 4 hr later according to previously described protocol.3-5 Images were evaluated by three independent observers unaware of clinical data.

RNA was performed in the anteroposterior projection during control conditions and in the left anterior oblique “best septal” projection in control conditions, during spontaneous or ergonovine-induced ischemia, and 3 min after the disappearance of symptoms and normalization of the ECG after intravenous administration of isosorbide mononitrate (12.5 mg) and 3 min later. The gated acquisition was stored in 16 frames of a 64 × 64 matrix.

Regional wall motion was visually assessed by three independent observers. Visual analysis as well as calculation of left ventricular ejection fraction were performed according to previously described methods,21-22

Furthermore, amplitude and phase of the first Fourier harmonic were generated from RNA.23 Phase-distribution histograms were computed separately for the left and right ventricles and were characterized by the standard deviation from the mean of the peak of the histogram (SDP).22

In the absence of conduction disturbances,24 this approach has been demonstrated to allow detection of asynchronous wall motion associated with transient ischemia.22, 25

First-pass RNA was performed by injecting a bolus of technetium-99m pertechnetate (8 to 10 mCi) into a small catheter positioned in superior vena cava. A first bolus was injected during an ischemic episode induced by the ergonovine test; a second bolus was injected 20 min after normalization of the ECG and disappearance of symptoms. First-pass data were collected (list-mode acquisition) and processed with the equipment used for RNA. The right anterior oblique 30 degree projection was chosen to obtain the best right ventricular view. Right ventricular ejection fraction was computed by the generated time-activity curves.26

Two-dimensional echocardiograms (Aloka SSD-800 electronic sector scan) were obtained in control conditions and during ischemia at the time of the radioisotopic procedures. In two cases the subcostal four-chamber view was monitored during the first-pass radionuclide study, while in the other two the parasternal short-axis approach was recorded during thallium-201 scintigraphic examination. Images were recorded on videotape and analyzed by two independent observers unaware of the clinical history.

In all scintigraphic and echocardiographic studies, nine-lead ECGs (standards, V1, V2, and V6) obtained every minute and arterial blood pressure (cuff manometer) measured every 2 min also provided evaluation of the pressure rate products (PRPs) at basal conditions and during ischemia.

**Results**

**Clinical and electrocardiographic observations.** During hospitalization, no patient was diagnosed as having acute myocardial infarction. Results of ECG-monitored exercise tests were positive in two patients, showing ST segment depression in the anterior leads at different PRPs. Continuous Holter monitoring revealed symptomatic and asymptomatic ischemic episodes in all patients. Most of the asymptomatic attacks were nocturnal. In addition to the ST segment elevation in the inferior leads during ischemia, 12-lead ECGs showed reciprocal ST segment depression in the precordial leads in all patients; in one patient ST segment depression was occasionally observed in the anterior leads alone (table 1). The V6 lead, recorded during ischemia, showed no ST-T changes. In all patients, the results of ergonovine testing were positive at low dosages (0.025 to 0.05 mg) and were reproducible.

PRP values obtained during ischemia (spontaneous or ergonovine-induced) were similar to control values (11.7 × 10⁳ and 8.9 × 10³ beats/min × mm Hg, respectively) and markedly lower than those reached at the exercise peak (average PRP, 21.7 × 10⁳ beats/min × mm Hg). No patient showed bradycardia or significant arterial hypotension during angina.

**Hemodynamic and angiographic findings.** Basal left ventricular end-diastolic pressures were within the normal range. When ischemia occurred, left ventricular end-diastolic and systolic pressures did not show obvious changes from control values (table 2).

In three patients with simultaneous right ventricular hemodynamic monitoring, right ventricular end-diastolic pressure increased during ischemia with respect to control (average values, 12.5 and 3.8 mm Hg, respectively) and was higher than the simultaneously measured left ventricular end-diastolic pressure (average value, 10.1 mm Hg). The increase in right ventric-
ular end-diastolic pressure was accompanied by a parallel increase in right ventricular systolic pressure (table 2).

Results of basal left ventricular contrast ventriculography were normal in all patients. No attempt was made to obtain ventriculograms during ischemia. Coronary arteriography showed a 90% RCA stenosis in two patients, a 75% circumflex artery stenosis in one, and normal coronary arteries in the other. Although the RCA supplied the posterior descending artery in all patients, coronary circulation was codominant in one (patient 3, with normal and large circumflex coronary artery), while an evident intercoronary collateral circulation was present in two (patients 1 and 4). A 100% proximal RCA spasm was documented in two patients (patients 3 and 4), and a 90% spasm of the middle RCA, involving right ventricular branches, was detected in the other two (patients 1 and 2) (figure 1). The coronary angiographic findings and the location and extent of coronary spasm in the four patients of our study are described in table 1. For comparison, in the four patients with left ventricular dysfunction during ischemia, left ventricular end-diastolic pressure abnormally increased with the onset of ST-T changes (from 11 to 20.6 mm Hg). Coronary arteries were normal in three patients, while a 90% stenosis of the middle RCA was demonstrated in one. The RCA was dominant in all; RCA spasm was always located distally to the origin of the main right ventricular branches. Right ventricular hemodynamic monitoring was not performed in these patients.

**Thallium-201 scintigraphy.** No myocardial perfusion defects were detected in the scintigrams taken immediately after ischemia as compared with the redistribution images obtained 4 hr later (figure 1). By contrast, the four patients with left ventricular isch-

<table>
<thead>
<tr>
<th>Patient No./age (yr)</th>
<th>Sex</th>
<th>ECG ischemia</th>
<th>Thallium defect</th>
<th>RNA ischemia</th>
<th>Echo ischemia</th>
<th>Coronary arteries</th>
<th>RCA spasm</th>
</tr>
</thead>
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<tr>
<td>1/58</td>
<td>M</td>
<td>ST ↑ II, III, aVF</td>
<td>ND</td>
<td>RVc</td>
<td>N</td>
<td>50</td>
<td>Irr</td>
</tr>
<tr>
<td></td>
<td></td>
<td>ST ↓ V2, V3</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2/58</td>
<td>M</td>
<td>ST ↑ II, III, aVF</td>
<td>ND</td>
<td>N</td>
<td>RV h,e</td>
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<td>75</td>
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<td>ND</td>
<td>RVc</td>
<td>LV hyperk</td>
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<td>0</td>
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<td>RVc</td>
<td>RV h,e</td>
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- e = enlargement; h = hypokinesis; hyperk = hyperkinesis; LV = left ventricle; RV = right ventricle; Mi = middle; P = proximal; CX = circumflex; LAD = left anterior descending; ST ↑ = ST segment elevation; ST ↓ = ST segment depression; ND = not detectable; N = normal; Irr = irregularities.

**TABLE 2**

Hemodynamic and radionuclide angiographic data in the four patients

<table>
<thead>
<tr>
<th>Patient 1</th>
<th>HR (bpm)</th>
<th>RVSP (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>LVSP (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>RVEF (%)</th>
<th>LVEF (%)</th>
<th>RVSDP (degrees)</th>
<th>LVSDP (degrees)</th>
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<td>12</td>
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<td>8</td>
<td>48</td>
<td>57</td>
<td>15.4</td>
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<tr>
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<td>—</td>
<td>—</td>
<td>125</td>
<td>7</td>
<td>32</td>
<td>71</td>
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<tr>
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<td>11</td>
<td>38</td>
<td>68</td>
<td>23.4</td>
<td>17.5</td>
</tr>
</tbody>
</table>

HR = heart rate; RVSP = right ventricular systolic pressure; RVEDP = right ventricular end-diastolic pressure; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; RVEF = right ventricular ejection fraction; LVEF = left ventricular ejection fraction; RVSDP = right ventricular standard deviation from the peak; LVSDP = left ventricular standard deviation from the peak; C = control; I = ischemia.
Emia showed transient defects of perfusion in the inferior wall. In none of these eight patients was the right ventricle visible during ischemia as well as under control conditions.

Radionuclide ventriculography. In all four patients with suspected right ventricular ischemia, visual inspection of angiograms showed normal regional left ventricular wall motion in control conditions, during ischemia, and after the resolution of the ischemic episode after administration of isosorbide dinitrate. Right ventricular enlargement was transiently observed in three patients during ischemia (figure 2).

Baseline left ventricular ejection fraction was within the normal range in all patients, increased or remained constant during ischemia (average values 61% and 66%, respectively) (table 2), and was above the control values after nitrate administration.

Furthermore, during ischemia right ventricular SDP markedly increased as compared with the control values (average values 26.5 and 14 degrees, respectively) while left ventricular SDP showed only minor changes (17.2 vs 14.3 degrees). Phase and amplitude images revealed an area with phase delay and reduction of pixel excursion located in the right ventricular free wall during ischemia (figure 2). After isosorbide dinitrate, both left and right ventricular SDP approached or were below the basal values.

For comparison, in the group of patients with evidence of ventricular dysfunction a transient left ventricular inferoapical akinesis was visualized during ischemia. Left ventricular ejection fraction fell with respect to the control values and returned to normal after administration of isosorbide dinitrate; left ventricular SDP markedly increased during ischemia while right ventricular SDP did not show appreciable changes as compared with control. Phase and amplitude images showed an area with delayed phase and reduction of amplitude in the left ventricle, which correlated well with the location of the dysynergy.

Right ventricular ejection fraction, as evaluated in all patients with suspected right ventricular ischemia by first-pass RNA, fell during transient ischemia (mean values 35% vs 55% at control; table 2). Obvious right ventricular enlargement was also detected in these patients (figure 1). One patient showed ST segment depression in the anterior leads and no changes in the inferior leads at the time of the acquisition during angina.

Two-dimensional echocardiography. Two-dimensional echocardiograms showed transient right ventricular dilation and hypokinesis of the right ventricular inferior wall in two patients and normal or increased left ventricular wall motion in all. In the other two patients, where the parasternal short-axis approach was used, right ventricular wall motion was normal in one and was not visualized in the other.

By contrast, two of the four patients with left ventricular ischemia showed inferior left ventricular akinesis during angina and one showed marked inferolateral and septal hypokinesis; the last patient was not studied echocardiographically. The clinical, electrocardiographic, scintigraphic, echocardiographic, and angiographic findings in the four patients with predominant right ventricular ischemia are shown in table 1. Right ventricular and left ventricular hemodynamics, first-pass, and RNA data are listed in table 2.
Discussion

These four patients could not be differentiated from the group of patients with evident left ventricular impairment during ischemia on the basis of their clinical, electrocardiographic, and angiographic features. All patients showed symptoms typical of Prinzmetal's angina caused by RCA vasospasm and characterized by ST segment elevation in the inferior leads.

Despite these similarities, in the four patients with documented right ventricular ischemia all diagnostic techniques used in this study failed to detect obvious left ventricular involvement. In our experience, in a large group of patients with angina at rest, thallium-201 scans,3-5 RNA,22, 27 and echocardiography6-7 as well as continuous left ventricular hemodynamic monitoring2-9 have proved to be highly sensitive techniques in the detection of transient myocardial ischemia. The negativity of these investigations, which mainly explore left ventricular function, led us to study more extensively the right ventricular function in these patients with suspected right ventricular ischemia by right ventricular hemodynamic measurements and first-pass RNA. We chose the first-pass technique because it provides repeatable, correct measurements of right ventricular ejection fraction, virtually independent from cavitary geometry20 and superimposition of right atrial activity, which may affect the right ventricular ejection fraction measurements derived from RNA.28-30

Our data support the existence of predominant right ventricular functional impairment in patients with transient ischemia at rest. This was reflected by decreased right ventricular ejection fraction and increased right ventricular end-diastolic pressure in the presence of "apparently" normal left ventricular function (table 2). Right ventricular stroke volume was maintained, probably by a compensatory increase in end-diastolic size. However, it is worth noting that left and right ventricular end-diastolic pressures and ejection fractions, which have been extensively used as suitable

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**FIGURE 2.** Radionuclide ventriculograms and phase analysis of RNA acquired at rest (top) and during ischemia (bottom) in a patient with predominant right ventricular ischemia (patient 1). End-diastolic and end-systolic images are shown on the left. Phase histograms and images of the distribution of temporal sequence of count changes in each pixel are represented for left ventricle on the middle and for the right ventricle on the right. During ischemia left ventricular ejection fraction and regional wall motion did not change with respect to the control. By contrast, the phase image reveals a region of right ventricular delayed phase located in the right ventricular free wall (arrow) and a marked increase in standard deviation (SD) from the mean of the peak (from 13 to 32 degrees).
indexes of myocardial function, are also affected by changes in preload, afterload, and ventricular stiffness as well as by changes in contractility. In addition, these parameters are indexes of global function, which in turn depends on left ventricular, right ventricular, and interventricular septal performance. The complex relationship between the two ventricles may be responsible for right ventricular dysfunction secondary to left ventricular ischemia but also for a masked left ventricular impairment in the presence of concomitant right ventricular dysfunction, which prevents adequate left ventricular filling. Although we used all available noninvasive techniques to assess regional left ventricular function, these possibilities cannot be completely ruled out in our patients with evidence of right ventricular dysfunction.

Contrast left ventriculography could allow better detection of segmental left ventricular wall motion abnormalities; however, this technique could not be applied in our protocol because of the very short duration of the ischemic episodes and the concomitant interest in assessing presence and location of the coronary spasm.

**Transient right ventricular ischemia and infarction.** Isner and Roberts showed in a series of postmortem subjects with acute or healed myocardial infarction that right ventricular infarction was frequently associated with posterior left ventricular wall and interventricular septal infarction. Furthermore, a large area of right ventricular necrosis was observed when the RCA narrowing was proximal, involving the conus branch that supplies a large portion of the right ventricle. In our study, none of the four patients with evidence of right ventricular impairment during ischemia showed septal and posterior left ventricular involvement as assessed by the above techniques. Right ventricular dysfunction also appeared to be severe in two patients with coronary spasm involving the middle portion of the RCA.

Differences in coronary anatomic distribution and collaterals as well as different mechanisms inducing flow impairment may have caused the apparent discrepancies between the necropsy findings and our data. Interestingly, in the two patients with incomplete spasm of the middle RCA, the vasoconstriction involved or was close to the origin of the main right ventricular branches while the distal portion of the RCA appeared to be normally filled by the contrast medium. Nevertheless, in the other two patients with proximal RCA spasm, basal coronary angiograms showed an intercoronary collateral circulation in one and a codominant circulation in the other. None of our patients had critical stenosis of the left coronary arterial system, otherwise present in a large extent in the postmortem subjects.

Furthermore, the right ventricular hemodynamic findings observed in three of these patients suggest that the early acute manifestation of transient right ventricular ischemia may be characterized by a different hemodynamic pattern with respect to the typical picture described for acute right ventricular infarction. Cohn et al. have recognized a syndrome of hypotension and low cardiac output in association with right ventricular infarction and attributed this to inadequate left ventricular filling. In our patients, the obvious increase of right ventricular end-diastolic pressure with the onset of the ST segment displacement on the ECG was associated with a slight increase in right ventricular systolic pressure and with normal left ventricular end-diastolic pressure. No arterial hypotension related to ischemia was observed in these patients, both during hemodynamic monitoring and in the coronary care unit. Thus left ventricular filling and cardiac output were probably adequate in these patients during ischemia.

The clinical syndrome we evaluated in this study (i.e., the vaso spas tic occlusion of the RCA) could represent a useful model for understanding the role of each ventricle and the possible sequence of the events in acute infarction involving the right ventricle. However, the very short duration of our observation period and the lack of information on the hemodynamic pattern in the early phase of irreversible right ventricular injury prevent any proper connection between RCA vasospasm and the acute phase of right ventricular infarction.

**Clinical implications.** In the last few years some authors have demonstrated stress-induced right ventricular ischemia in patients with coronary artery disease by scintigraphic evaluation of left and right ventricular ejection fraction. Our preliminary data suggest the possibility of a predominant right ventricular ischemia of vasospastic origin. The location of selective ischemia in the right ventricular wall may explain the negativity of clinical investigations that mainly assessed left ventricular perfusion and wall motion. Moreover, episodes of ischemia occurring in the right ventricle may be manifest with ST segment depression in the anterior leads alone (as detected during spontaneous episodes and the first-pass study in one patient and during the exercise test in two). These electrocardiographic changes in the precordial leads during transient ischemia have already been shown to be a poor predictor of location of the
ischemic process. Thus right ventricular ischemia inducing true or ‘reciprocal’ ST segment depression in the precordial leads represents another potential source of misinterpretation of the electrocardiographic findings. Therefore, a more accurate evaluation of right ventricular function should be performed each time routine diagnostic tools fail to show any ischemic involvement of the left ventricle in the presence of typical anginal symptoms and/or equivocal electrocardiographic changes.

The peculiar geometric and functional characteristics of the right ventricle make its investigation difficult. However, as documented in this study, available noninvasive techniques (such as first-pass RNA and quantitative phase analysis of radionuclide angiograms) can be successfully used for this purpose. Although the actual value of separate left and right ventricular phase analysis in the detection of single-ventricle dysfunction has still to be defined, our findings demonstrate the usefulness of this technique in the assessment of predominant right ventricular dysfunction.

If confirmed, these results would indicate that phase analysis of radionuclide angiograms, as compared with the first-pass technique, could represent an easier way to detect transient right ventricular ischemia by long-term monitoring of right ventricular function.

In addition to the technical approaches used in this study, other experimental and clinical investigations should be performed to examine extensively the functional relationship between the right and left ventricles and to assess primary or secondary right ventricular involvement in different types of angina.

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