Discussion

Myocardial stunning is defined as a prolonged myocardial dysfunction with gradual return of contractile activity after a brief episode of severe ischemia. This phenomenon was first described as a distinct clinical entity by Braunwald and Kloner in 1982. It has been described in patients with coronary artery disease following successful thrombolytic therapy, after percutaneous transluminal coronary angioplasty, demand induced ischemia, and coronary vasospasm. It also has been described as an uncommon feature of conditions like toxic shock syndrome, Legionnaires' disease, and following electroconvulsive therapy.

The exact mechanism of myocardial stunning is not clear. Jennings et al. proposed that myocardial ischemia leads to reduction in both creatine phosphate and adenosine triphosphate. With reperfusion, there is immediate restoration of creatine phosphate levels to normal or slightly above normal, while adenosine triphosphate takes several days to return to normal. This depletion of the total adenine nucleotide pool leads to prolonged depression of myocardial contractility. Other possible mechanisms include alterations in sarcoplasmic calcium adenosine triphosphatase and calcium metabolism, upregulation of the gene for heat shock protein, and generation of oxygen-free radicals.

Our patient, who was a healthy young female with no risk factors for coronary artery disease, developed prolonged myocardial depression following a brief episode of severe hypoxemia. The global nature of ventricular hypokinesis, the absence of cardiac enzymes, and the complete restoration of electrical and mechanical function of the heart strongly support the diagnosis of myocardial stunning in this patient. The chest pain was most likely musculoskeletal in origin even though pericarditis could not be excluded with certainty. Coronary spasm due to stress-related catecholamine surge is unlikely because of its rare occurrence and the absence of ST segment elevations.

A careful search of the English-language medical literature did not reveal any previous reports of myocardial stunning following respiratory arrest. This case illustrates that myocardial stunning may occur as a sequela following successful resuscitation from respiratory arrest. This condition can occur in young subjects without any preexisting coronary artery disease, and because it is reversible, it does not necessarily indicate a grim prognosis. Hence, we recommend that such patients should be closely observed for any signs of cardiac decompensation.

References


Biopsy Evidence of Atrial Myocarditis in an Athlete Developing Transient Sinoatrial Disease*

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Atrial myocarditis causing transient sinoatrial disease (incessant atrial tachycardia alternating with sinoatrial pauses of up to 6 s in duration) in an athlete is reported. Diagnosis was undertaken by endomyocardial biopsy; biventricular and right atrial specimens were ob-

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tained. After a 6-month rest period, the atrial arrhythmias disappeared, and the athlete was able to resume his professional sporting activities.

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**FAT=focal atrial tachycardia**

**Key words:** atrial arrhythmias; atrial biopsy; myocarditis

In sportsmen with apparently normal hearts, myocarditis has been suggested as a cause of both ventricular and atrial arrhythmias, but a direct *in vivo* demonstration of this relationship has rarely been given through endomyocardial biopsy only in athletes with ventricular arrhythmias.

Indeed, the histologic substrate of lone atrial arrhythmias is difficult to study because of the anatomic structure of the atrial wall. Therefore, only indirect histologic data obtained by ventricular endomyocardial biopsy are available.

The authors report the identification of atrial myocarditis by means of atrial endomyocardial biopsy in an athlete developing lone sinoatrial disease.

**Case Report**

A 20-year-old professional soccer player had been well and training regularly until October 1992 when he experienced a severe traumatic injury to the right leg. When he resumed playing in May 1993, his team doctor noticed a very rapid and irregular pulse. An ECG taken at rest showed incessant runs of focal atrial tachycardia (FAT), at a mean rate of 145 beats per minute, alternating with pauses of 1.4 to 1.5 s. A previous ECG taken 1 year before showed no abnormalities. The athlete was referred to our center for further evaluation.

He did not recall any cardiovascular symptoms or flu-like episodes during the preceding few months. The family medical history disclosed no cardiac diseases, and there was no history of cocaine or other drug abuse.

At the time of admission, the patient was afebrile. Erythrocyte sedimentation rate (8 mm/h), C-reactive protein (4 mg/L), serum electrolyte, and thyroid hormone values were within normal limits.

On physical examination, the patient appeared healthy; cardiac examination revealed tachycardia and an S₃ gallop, with normal BP. A standard echocardiographic Doppler examination revealed normal cardiac dimensions and intracardiac flows. On ECG stress testing on a cycle ergometer, he achieved a maximum workload of 240 W with a maximal heart rate of 181 beats per minute and a BP of 175/65 mm Hg with no symptoms. Overdrive suppression of FAT by normal sinus rhythm occurred at a heart rate of 110 beats per minute and persisted until the 16th minute of recovery when ECG monitoring was interrupted. A 24-h Holter recording documented incessant FAT with posttachycardia pauses lasting up to 6 s (Fig 1).

He was advised to stop his sport activity as a temporary measure. Because the patient’s livelihood was at stake, he underwent elective invasive investigation including electrophysiologic study, cardiac catheterization with coronary angiography, ventriculography, and endomyocardial biopsy.

Intracavity as well as pulmonary pressures were within normal limits, and the cardiac index was 3.5 L/m² of body surface. Right and left ventriculography revealed normal dimensions and kinesia of both ventricles. At the time of the electrophysiologic study, FAT was easily interrupted by continuous atrial pacing at a cycle length of 500 ms, and no tachyarrhythmias could be induced despite an aggressive stimulation protocol. Corrected sinoatrial recovery time was normal.

Endomyocardial biopsy was performed in the septo-apical region of both ventricles and in the right side of the interatrial septum. Three specimens from each ventricular chamber and two from the right atrium were obtained and processed for histologic examination following standard techniques and stains.

Histologic study revealed the presence, in the atrial samples (Fig 2), of clusters of lymphocytes associated with interstitial edema and focal necrosis of adjacent myocytes. These changes were in accordance with the histologic “Dallas criteria” for myocarditis, mostly localized in the atrial wall. Biventricular specimens showed only in some sections rare lymphocytic infiltrates with very occasional fraying of the adjacent myocytes, suggesting less pronounced inflammatory involvement of the ventricular myocardium.

Following histologic results, serologic findings for the commonest viruses (including Echo, Coxsackie B, influenza, parainfluenza) were negative.

The athlete rested for a 6-month period. Serial examinations documented a progressive reduction of FAT periods and the duration of posttachycardia pauses on Holter monitoring. At the end of this period, he resumed training, following a moderate aerobic program.

After 3 months of regular training, a complete noninvasive cardiologic examination revealed the disappearance of all rhythm disturbances except for very rare isolated atrial premature beats on Holter monitoring (Fig 3B). The athlete declined a control cardiac biopsy.
DISCUSSION

Myocarditis has been suggested as a cause of both
tirventricular and atrial arrhythmias in sedentary people and
sportsmen with apparently normal hearts. However, a direct
in vivo demonstration of this relationship has been confined
to idiopathic ventricular arrhythmias in athletes and in un-
trained people because of the anatomic structure of the
atrial wall. Only indirect histologic studies obtained by means
of ventricular endomyocardial biopsies are available in sub-
jects with lone atrial tachyarrhythmias.

To our knowledge, the present case is the first to confirm
that atrial myocarditis may be a cause of complex supraven-
tricular arrhythmias in athletes with apparently normal
hearts.

From a histologic standpoint, it can be speculated that
inflammatory cells can be increased in the interstitium of the
atrial wall as a consequence of the hemodynamic alteration
that accompanies a hyperkinetic atrial arrhythmia. In this
case, the presence of necrotic myocardyocytes, surrounded
by clusters of lymphocytes, makes it unlikely that the histo-
logic changes were the result of the arrhythmia itself and
suggests that an inflammatory process may have been
present. Biventricular specimens showed a less pronounced
involvement by the inflammatory process and explained the
absence of ventricular hypocontractility or arrhythmias or
both. Furthermore, the presence of lymphocytic myocardi-
tis suggests a viral cause but the serologic tests for such were
negative. In an extensive virologic study of patients with
clinical and histologic evidence of myocarditis, a positive
serologic test result was found in only 50% of cases.

From the clinical point of view, our report demonstrates
that a complete resolution of atrial myocarditis can occur
spontaneously, but it may take a long period of time.
Therefore, sport physicians must be aware of this possibility
when evaluating sportsmen with a recent onset of such
arrhythmias, especially if they had recently suffered flu-like
episodes. An early diagnosis of myocarditis and an adequate
period of rest are essential in order to facilitate the recovery;
strenuous physical activity can negatively influence the
immunologic processes that accompany a myocardial inflam-
mation.

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Necrosis of the Bronchus*

Role of Radiation

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The effects of radiation on the lung parenchyma and
pleura are well described in the literature. Necrosis of
the larynx is a known complication of radiation ther-
apy. Necrosis of a part of the tracheobronchial tree
following radiation therapy for bronchogenic carci-
noma is likely to occur; however, there is little mention
in the English-language literature about such an ef-
fect. This report describes four cases with total necro-
sis of a specific bronchus following radiation therapy
for squamous cell carcinoma of the lung. All patients
received 5,000 to 6,400 rad (50 to 64 Gy) of external-
beam radiation. Two patients presented with massive
hemoptysis and two with pneumonia. In all four cases,
the patients were found to have, by bronchoscopy,
necrosis of the bronchus with the involved lobe of the
lung replaced by a large cavity lined by tumor tissue.
Diagnosis was made 5 to 7 months after radiation
therapy was completed. Three of the patients died of
exsanguination within weeks following diagnosis of the
complication. We suspect that such necrosis occurs as

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