

Letter to the Editor

## Myocarditis with ST-Elevation Myocardial Infarction presentation in young man. A case series of 11 patients

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### Abstract

Acute myocarditis may mimic an infarction. Aim is to describe a case series of peculiar myocarditis. From 1997 to 2003, 11 male patients (age 17–39 years) were admitted with diagnosis of acute myocardial infarction, localized ST segment elevation and minimal enzyme release. Ten patients had fever in the 3 days prior to admission. Eight patients underwent coronary angiography showing normal coronary arteries. All remained asymptomatic at long term follow-up. In conclusion, myocarditis with ST elevation myocardial infarction presentation is an acute benign syndrome especially frequent in young males.

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Myocarditis is a poorly understood disease because it progresses through stages with distinctly different mechanisms and manifestations. The disease is considered to be present when the heart is involved in an inflammatory process, often caused by an infectious agent [1]. Acute myocarditis may mimic ST-Elevation Myocardial Infarction (STEMI), and its identification may have important therapeutic and prognostic implications [2]. In our experience in the last 6 years in a primary care coronary care unit of Southern Italy, we observed 11 patients with striking similarities in clinical presentation, laboratory findings, in-hospital outcome and long-term follow-up, possibly outlining a “young male benign myocarditis” syndrome which may be worthwhile recognizing.

From 1997 to 2003, 11 patients (all males, age 17–39 years) were admitted to our Coronary Care Unit with presumptive diagnosis of STEMI. The main clinical, laboratory, electrocardiographic, and follow-up findings are reported in Table 1. All patients shared the following features: young (>16 and <40 years) males; low coronary risk profile (smoking was present in six patients); prolonged (>1 h, range 1–9 h) typical chest pain on admission;

persistent ST elevation >1 mm in at least two contiguous leads; early and sustained cardiac enzyme release.

The differential diagnosis between myocarditis and acute myocardial infarction can be very challenging, and sometimes impossible [2]. In our series, we identified a constellation of features which make the diagnosis of myocarditis likely. A young male with low coronary risk profile and a recent history of fever can have a high likelihood of myocarditis on clinical grounds. The ECG shows mild, usually localized (inferior or anterior) ST segment elevation, slowly normalizing in the subsequent days. The cardiac enzymes are abnormal, but with a distinct non-coronary pattern: elevated on admission (even when the time from onset of chest pain is very short), with relatively low peak value and with late normalization (requiring 4 to 7 days). There was no correlation between initial enzyme levels and the initial and late left ventricular ejection fraction [3], and all patients showed normal echocardiographic findings at late (>3 months) follow-up examination. An intriguing finding is that all our patients were young males in the 17 to 39 years range. Due to the small sample size, this can be simply a game of chance. Alternatively, it can represent the clinical counterpart of a well-known experimental finding that some viral strains require adult males to induce myocarditis. Coxsackievirus B3 infection causes significant cardiac inflammation in male,

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Table 1  
Study patients

Progressive number, initials, age (years)	Month admission	Fever before admission	CK-MB admission/peak (ng/ml) (Tropon. I)	Coronary risk factors (smoking)	ECG on admission	ECG upon discharge	Follow up (months)
1. S.W., 19	March	+	71/73 (NA)	0	↑ST D1, aVL V4–V6	Diphasic T waves V4–V6	87
2. T.G., 24	November	+	39/83 (NA)	0	↑ST D1 aVL, V6	Negative T waves D1, D2, aVL, V5, V6	68
3. C.E., 32	December	–	76/77 (NA)	0	↑ST D1, D2 V4–V6	Negative T waves V4–V6	66
4. A.A., 38	February	+	67/120 (NA)	1 (smoking)	↑ST D2, D3 aVF, V5–V6	Diphasic T waves D3, aVF	65
5. C.M., 30	April	+	91/130 (NA)	1 (smoking)	↑ST D2, D3 aVF	Negative T waves D3, V6	51
6. B.M., 17	January	+	60/60 (NA)	0	↑ST D1, D2 aVF, V4–V6	Negative T waves D2, D3 aVF, V4–V6	42
7. C.R., 32	August	+	24/43 (17)	1 (smoking)	↑ST D2, D3 aVF, V4–V6	Normal	23
8. C.G., 39	October	+	17/30 (16)	1 (smoking)	↑ST D1, aVL	Negative T waves D1, aVL, V6	9
9. S.S., 28	December	+	23/23 (10.5)1	1 (smoking)	↑ST D2, D3 aVF, V5–V6	Normal	7
10. C.R., 21	April	+	34/34 (5.4)	1 (smoking)	↑ST D2, D3 aVF, V6	Normal	3
11. C.R., 28	April	+	17/17 (3.6)	0	↑ST D1, aVL	Flat T waves D1, aVL	3

NA = not available; + = present; – = absent.

but not female, B1.Tg.Ealpha mice, probably because high levels of testosterone are required to express cell surface antigens triggering antigen-specific immune responses [4,5]. In our series, some clues can be considered consistent with the viral etiology: a fever with flu-like symptoms was present in the few days before admission in 10 patients, and in 3 out of the 4 tested patients, a direct evidence of enterovirus infection was found. Nevertheless, all these evidences are indirect, weak and circumstantial, and therefore, the possible viral etiology of the “young male benign myocarditis” remains an educated guess.

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