The Journal of Emergency Medicine, Vol. 17, No. 5, pp. 865–872, 1999

0736-4679/99 $–see front matter

ELSEVIER

PII S0736-4679(99)00097-9

Selected Topics: Cardiology Commentary

ELECTROCARDIOGRAPHIC MANIFESTATIONS: ACUTE MYOPERICARDITIS

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□ **Abstract—Acute, or so-called "dry," myopericarditis occurs in the presence of diffuse inflammation of the pericardial sac and superficial epicardium from a multitude of infectious and inflammatory processes. This inflammation results in a current of myocardial injury resulting from the epicardial irritation manifested by a number of electrocardiographic findings. Classically, the electrocardiographic changes have been described as an evolution through several distinct stages involving ST segment elevation with PR segment depression, normalization of the ST segment abnormality with T wave inversion, and eventual normalization of the electrocardiogram over a period of days to several weeks. The following discussion focuses on the electrocardiographic manifestations of acute myopericarditis and includes findings useful in establishing the diagnosis as well as distinguishing the disease from other syndromes, particularly acute myocardial infarction. © 1999 Elsevier Science Inc.**

□ **Keywords—acute myopericarditis; ST segment; ST segment elevation; PR segment depression; electrocardiogram**

INTRODUCTION

Acute pericarditis, a diffuse inflammation of the pericardial sac and superficial myocardium, has a number of underlying causes including infection (primarily viral), immunologic disorders, uremia, trauma, malignancy, cardiac ischemia, acute myocardial infarction (AMI), and a multitude of other etiologies (Table 1) (1–4). Patients commonly present with complaints of sharp, pleuritic, precordial or retrosternal chest pain, mild to severe in intensity, which can radiate to the left upper back, left shoulder, or left arm. The pain is often worsened by recumbency, coughing, swallowing, and inspiration, while it may be relieved by an upright position and leaning forward. Physical examination findings are variable and transient, the most specific of which is a "Velcro-like" 3-phase pericardial friction rub.

The clinical presentation of acute pericarditis can be confused with a variety of other cardiac and noncardiac syndromes, including pneumothorax, pulmonary embolism, aortic dissection, and acute coronary ischemia. In addition, postmyocardial infarction myopericarditis, occurring in immediate association with infarction (as opposed to Dressler's syndrome, which occurs days to weeks after acute myocardial infraction) has been reported in up to 23% of such patients. Acute myopericarditis occurring with AMI is associated with larger infarcts and an overall poorer prognosis (4–7). The difficulty in distinguishing acute myopericarditis from AMI, coupled with an increasing emphasis on rapid revascularization therapies in patients with acute infarction, has led to administration of thrombolytic agents to patients with acute pericarditis (8–13).

It has been reported that up to 90% of patients with acute myopericarditis have abnormalities on electrocardiogram (EKG) (1). Classically, several stages of electrocardiographic change have been described involving

RECEIVED: 8 March 1999; ACCEPTED: 26 March 1999

Table 1. Causes of Acute Myopericarditis

Infectious
Viral: coxsackievirus, echovirus, influenza virus
Bacterial: staphylococcus, pneumococcus, syphilis, tuberculosis, mycoplasma, rickettsia
Fungal
Immunologic/Rheumatologic
Lupus, sarcoidosis, vasculitis, inflammatory bowel disease
Cardiac
Ischemia, myocardial infarction, postinfarct Dressler's syndrome, valvular disease, endocarditis, aortic dissection, trauma, postsurgical S/P instrumentation
Medications
Procainamide, hydralazine, methyldopa, heparin, warfarin
Other
Cancer (metastatic, breast, lung, lymphoma, leukemia)
Uremia
Pregnancy
Radiation therapy
Idiopathic

ST segment elevation, PR segment depression, T wave inversion, and normalization (14–18). Less than half of patients with acute myopericarditis, however, evolve through all stages (2). In fact, atypical progression is common (14,16). Despite this, careful evaluation of the EKG in the emergency department (ED) can help in providing important clues to the diagnosis of acute myopericarditis.

CASE PRESENTATIONS

Case One

A 38-year-old male without past medical history presented to the ED with left chest pain. The intermittent pain had appeared approximately 2 days before arrival in the ED and was worsened upon inspiration, assuming the supine position, and with upper extremity movement; no associated symptoms were noted. The patient had recently experienced an upper respiratory infection that he self-medicated with over-the-counter remedies. These symptoms had resolved 2 weeks before presentation. The physical examination was normal; no chest wall tenderness was found. A 12-lead EKG (Figure 1) revealed normal sinus rhythm with widespread ST segment/T wave changes.

The patient's pain lessened considerably with treatment with a nonsteroidal anti-inflammatory agent and morphine sulfate. He was admitted to the ED-based chest pain center with echocardiographic examination that revealed a small pericardial effusion. The patient was admitted to the cardiology service for 24-h observation and discharged without incident. No etiology was discovered for the pericarditis; an infectious etiology was suspected based upon the patient's recent upper respiratory infection symptoms.

Table 2. Electrocardiographic Findings for the 4 Stages of Acute Myopericarditis

Stage I	Days–2 weeks	Diffuse PR segment depression (I, II, III, avL, avF, V2-6) (With reciprocal PR segment elevation in avR, V1) Diffuse ST segment elevation (I, II, III, avL, avF, V2-6) (With reciprocal ST segment depression in avR, V1)
Stage II	1–3 weeks	ST segment normalization T wave flattening with decreased amplitude
Stage III	3-several weeks	T wave inversion
Stage IV	Several weeks	Normalization Return to baseline EKG

Case Two

A 47-year-old male with a history of hypertension and renal insufficiency presented to the ED via ambulance with left-sided chest pain that worsened upon both inspiration and reclining. The patient had demonstrated noncompliance with antihypertensive therapy and surveillance of his renal impairment. Examination revealed a middle-aged man in considerable distress, clutching his chest; the remainder of the examination was remarkable only for a pericardial friction rub. A 12-lead EKG (Figure 2) demonstrated sinus rhythm with marked ST segment, PR segment, and T wave changes. Laboratory studies revealed marked progression of the renal insufficiency with a serum creatinine of 10.9 mg/dL and blood urea nitrogen of 112 mg/dL as well as a metabolic acidosis. The patient received i.v. morphine sulfate and ketorolac while arrangements were made to initiate hemodialysis. With the above medications, the patient's pain was reduced. Echocardiography revealed a moderate pericardial effusion without evidence of tamponade. He was admitted to the hospital with a diagnosis of uremic myopericarditis.

DISCUSSION

Electrocardiographic abnormalities associated with acute myopericarditis have been attributed to repolarization changes involving both the atrium and ventricle as a result of epicardial inflammation. These repolarization changes consequently affect the morphology of the PR segment, ST segment, and T wave. Depolarization is usually normal, and abnormalities of the P wave and QRS complex are rarely seen. The four-stage evolution

Table 3. Electrocardiographic Findings for Acute Myopericarditis, BER, and AMI

	Acute Pericarditis	BER	AMI
ST morphology	Concave upsloping	Concave upsloping	Flat/convex
PR segment depression	Present	Absent	Absent
Q waves	Absent	Absent	Present
T wave inversion	After ST normalization	Absent	With ST elevation
Distribution	Diffuse*	Precordial	Anatomic
ST/T ratio	>0.25	<0.25	N/A
Evolution	Days–weeks	Years	Hours–days

* May have reciprocal changes in avR, V1.

seen in acute myopericarditis was first described by Spodick and others (19–21). Delineation of the stages is based on ST segment and T wave changes associated with the disease (Table 2). Patients may manifest all or only some of the classic abnormalities seen on the EKG, some having more typical and others more variant patterns (16). Stage I occurs during the first few days of inflammation, lasting up to 2 weeks. Electrocardiographic abnormalities in this stage are seen more often than those associated with subsequent stages and, in fact, are considered "quasi-diagnostic" (16). The most characteristic electrocardiographic finding is ST segment elevation that occurs as a result of ventricular repolarization abnormality not confined to a discrete anatomic location. In addition, the EKG may reveal diffuse PR segment depression as a result of atrial repolarization abnormalities.

Stage II, characterized by ST segment normalization, is extremely variable in duration, lasting from a few days to several weeks after resolution of the ST segment elevation seen in Stage I. In addition, T wave amplitude will decrease, perhaps "flattening." Onset of Stage III occurs in the second or third week of the illness and may be transient or prolonged, lasting from a few days to several weeks. During this time, full T wave inversion occurs with reversal of polarity from baseline. Stage IV is marked by resolution of T wave abnormalities and return to the baseline premyopericarditis EKG.

It is important to note that while this four-stage evolution has been described as an orderly progression that is essentially "pathognomonic" for the disease, its actual occurrence or documentation of occurrence on serial EKGs is not at all common. In many instances, only part of this sequence occurs, and particular stages can be missed completely; the EKG may progress from any of the stages directly to stage IV or resolution. This is particularly true in the ED setting, where the physician must evaluate patients at one point in time, most commonly during Stage I.

ST Segment Elevation

The ST segment elevation (Figures 1–3) occurs as a result of repolarization abnormality attributed to epicardial inflammation of the ventricular myocardium. The elevation is usually less than 5 mm, occurring simultaneously throughout all limb and precordial leads (I, II, III, avF, avL, V2-V6) with the exception of avR and V1 (which often have reciprocal ST depression). Other than in the case of postinfarct myopericarditis where inflammation may be localized to a single anatomic region such as the inferior wall, there is no clear territorial distribution on the EKG (5,7,15). This findings helps in making the distinction between AMI and myopericarditis. The shape of ST segment elevation in acute myopericarditis is characterized by concavity on its initial upslope, though in some instances the elevation may be obliquely flat (1,2). As a result, the J point is often indistinct. During Stage I ST segment elevation, the T wave remains concordant without evidence of flattening or inversion. "Reciprocal" ST segment depression may be seen in leads avR and V1, as demonstrated in Figures 2 and 3.

The presence of ST segment elevation typical of myopericarditis, particularly the morphologic and distribution features, is considered "virtually diagnostic" of the disease (15). These classic features are often not present, though, probably occurring in less than half of all patients. In particular, postinfarct myopericarditis may reveal more variant ST segment changes (5,15). In a review of 44 patients with acute myopericarditis, Bruce and Spodick found that 43% had atypical electrocardiographic findings, including a lack of ST segment changes or atypical distribution or evolution (15).

PR Segment Depression

Although ST segment elevation reflects ventricular repolarization abnormalities, PR segment depression (Figures 1–3) results from atrial repolarization irregularities and,

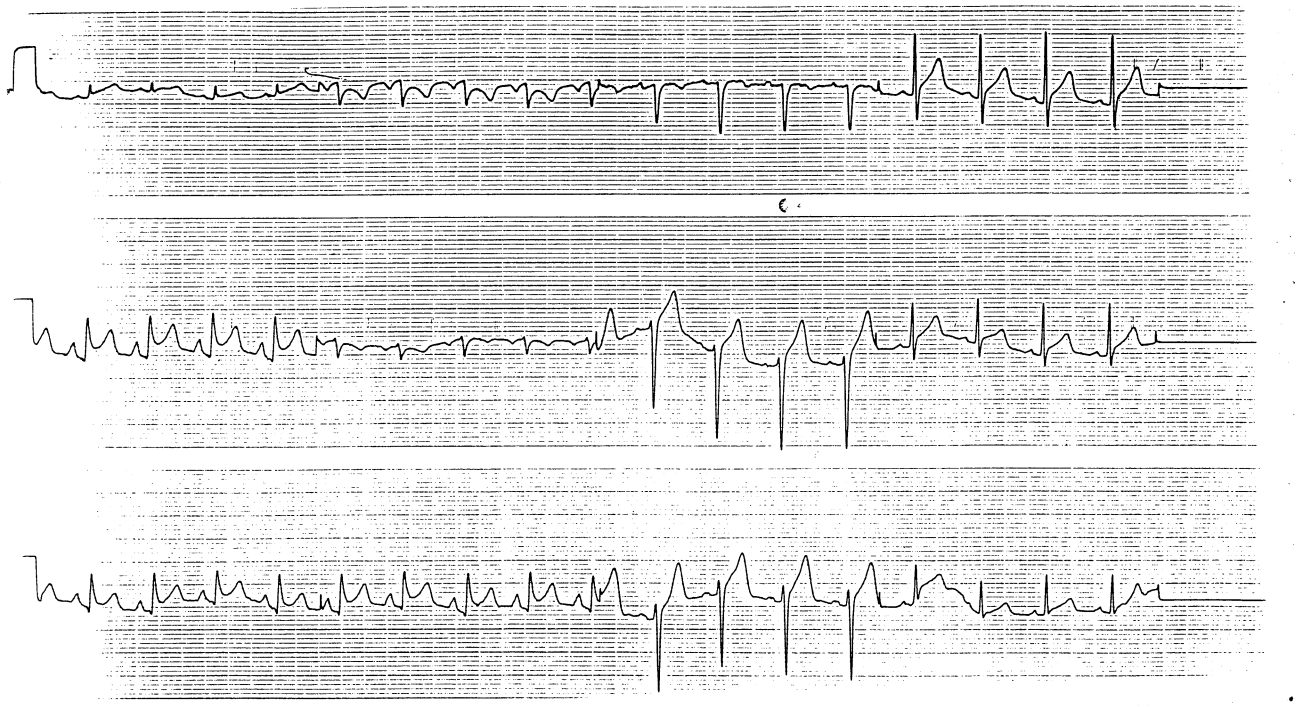

Figure 1. (Case One): Sinus tachycardia in a patient with idiopathic myopericarditis following a viral upper respiratory infection with ST segment elevation in leads II, III, avF, and V2-V5. The ST segment elevation is widespread with a concave morphology in the precordial leads (reciprocal ST segment depression is seen in lead avR). PR segment changes are also seen in leads II, III, and avF (depression) as well as in lead avR ("reciprocal" elevation).

importantly, is very specific for the diagnosis of acute myopericarditis. Investigations suggest that PR segment depression may occur earlier, and be of shorter duration, than ST segment elevation in the initial stage of inflammation (22,23). In fact, PR segment depression may be the earliest sign of acute myopericarditis and, with certain etiologies, the only sign of the disease on the EKG (22). Similar to ST segment elevation, PR segment de-

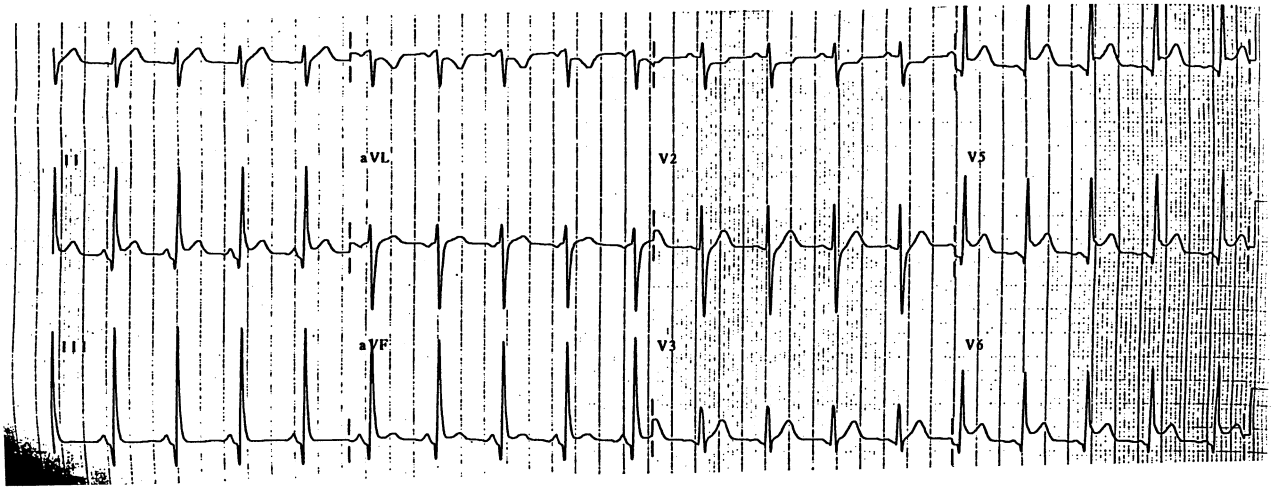

Figure 2. (Case Two): Sinus tachycardia in a patient with uremic myopericarditis with numerous electrocardiographic changes consistent with pericarditis: (1) ST segment elevation in leads I, II, avL, avF, and V3-V6 that is either convex or obliquely flat in morphology; (2) "reciprocal" ST segment depression in leads avR and V1; (3) PR segment depression in leads II, III, avF, and V3-V6; and (4) "reciprocal" PR segment elevation in lead avR.

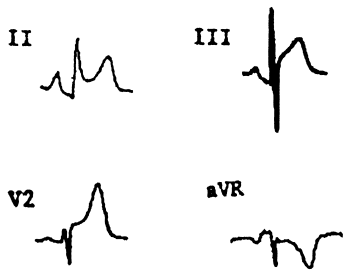

Figure 3. Four electrocardiographic complexes from different patients with ST segment and PR segment abnormalities seen in acute myopericarditis. Lead II reveals both ST segment elevation and PR segment depression; note that the ST segment elevation in this lead is obliquely flat, worrisome for AMI. In lead III, concave ST segment elevation and PR segment depression are seen. Lead V2 reveals ST segment elevation while lead avR demonstrates the "reciprocal" changes seen in acute myopericarditis: ST segment depression and PR segment elevation.

pression occurs diffusely in the limb and precordial leads, with the exception again of avR and V1, which may have reciprocal PR segment elevation (Figures 1–3). PR segment abnormalities may be most prominent, however, in leads II, V5, and V6 (5). In assessing the PR segment, it is important to use the TP segment as baseline, otherwise PR segment depression may be misinterpreted as ST segment elevation.

The PR segment vector shift is directed primarily to the left of the QRS axis (16). While it was once believed that vector analysis of electrocardiographic changes, including PR segment depression, could be used as a discriminator for acute myopericarditis, this has been discredited by recent studies (17,21,24). The finding is not surprising since electrical force vectors can be affected by factors not related to the disease, such as body habitus and heart position (14,16,25).

T Wave Inversion

T wave inversions are encountered diffusely on the EKG at later stages in acute myopericarditis. Discordant T wave inversions occur only after ST segment elevations have resolved. While typical myopericarditis findings are rare in postinfarct myopericarditis, new data suggest that abnormal T wave persistence or gradual reversal of T wave deflection may help in the diagnosis of pericardial inflammation following AMI (5,7,26–29).

Electrocardiographic Rhythm

The most common rhythm associated with acute myopericarditis is normal sinus rhythm or sinus tachycardia, though there are reports of severe sinus bradycardia with the disease (30). Because of the proximity of the sinus node to the pericardium and the occasional occurrence of atrial dysrhythmias in association with acute myopericarditis, it was previously thought that the disease could precipitate atrial fibrillation and flutter (14,31). But recent studies indicate that the disease does not cause significant dysrhythmias. In fact, anatomic studies suggest that the sinus node is virtually immune to involvement from surrounding inflammation (14). Clinically, this finding has been confirmed; the only group of pericarditis patients with dysrhythmia are those with a history of past heart disease or dysrhythmia due to another cardiac process (15,23,31,32). Spodick prospectively studied 100 acute myopericarditis patients and found that only seven experienced any dysrhythmia, primarily supraventricular (atrial fibrillation, atrial flutter, and junctional tachycardia). In all cases, the dysrhythmias were associated with underlying cardiac disease, particularly valvular etiology (31).

Other Electrocardiographic Issues

Other electrocardiographic findings in acute myopericarditis may be related to the development of complications from the disease. Pericardial effusion may result in widespread low voltage across all leads as a result of increased resistance from the accumulated fluid. Electrical alternans, beat-to-beat alternation in the QRS complex from shifting fluid and heart position, may occur in the setting of a large pericardial effusion with or without cardiac tamponade (Figure 4).

Electrocardiographic Differential Diagnosis

The clinical differential diagnosis of acute myopericarditis includes potentially life-threatening syndromes such as pneumothorax, pulmonary embolism, and aortic dissection, which are often distinguished by presentation and additional clinical or radiologic studies. Two entities, benign early repolarization (BER) and AMI, present with similar electrocardiographic findings to acute myopericarditis (Table 3).

BER was the topic of a prior article on electrocardiographic manifestations (33). It occurs most commonly in young, black males. On the EKG, BER has ST segment elevation similar in morphology to that of acute myopericarditis (Figure 5) with an initial concave upsloping and an indistinct J point. However, unlike acute myopericarditis, ST segment elevation in BER is limited primarily to the precordial leads, most prominent in the right precordial leads and least prominent in lead V6 (in

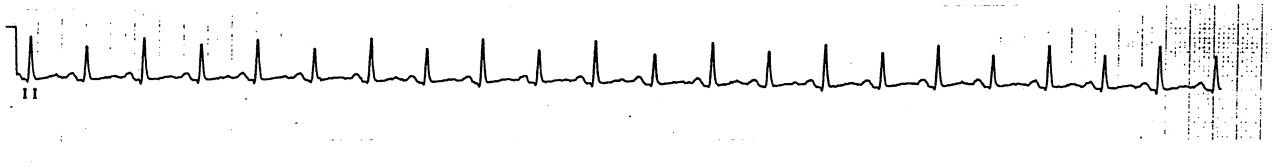

Figure 4. Electrical alternans is seen in this electrocardiographic rhythm strip from a patient with acute myopericarditis related to lymphoma. Note the alternating large and small QRS complexes seen in lead II.

which the ST segment is often isoelectric) (1). Moreover, while ST segment elevation in BER may transiently return to baseline with exercise, it does not resolve over the short term as with acute myopericarditis.

One of the most reliable discriminators of BER and acute myopericarditis on EKG is the ratio of ST segment to T wave (ST/T ratio) in lead V6 (Figure 6). Using the PR segment as the baseline for the ST segment and the J point as the beginning of the T wave, the heights are measured and a ratio calculated. A ratio of 0.25 or more points to a diagnosis of acute myopericarditis while a value less than 0.25 strongly suggests BER. Essentially, this ratio describes the electrocardiographic differences in the two syndromes. In BER, the J point is minimally elevated with a prominent T wave. In acute myopericarditis, the J point and ST segment elevation are more pronounced with a less prominent T wave. Additionally, PR segment depression can augment this ratio. Previous studies suggest that this ratio is both highly sensitive and specific for the disease. In a study of 19 acute myopericarditis and 20 BER patients, the ST/T ratio criteria in lead V6 had a positive and negative predictive value of 1 (25). If lead V6 is unavailable, the ratio can be applied to leads V4, V5, and I, though with less sensitivity and specificity (25).

The distinction between acute myopericarditis and AMI on the EKG can be difficult. Both syndromes commonly result in electrocardiographic changes including ST segment elevation and T wave inversion. In fact, with increasing emphasis on rapid revascularization in the setting of AMI, difficulty in distinguishing the two syndromes has led to administration of thrombolytic therapy in patients with acute myopericarditis. Though the overall risk is low from such treatment, there have been reports of significant complications, including bleeding, tamponade, and death (9–13). While electrocardiographic findings may be similar, important differences help to distinguish acute myopericarditis from AMI. Regarding ST morphology, the elevation in AMI is convex initially, as opposed to concave in acute myopericarditis (Figure 7). An analysis of the ST segment waveform may be particularly helpful in distinguishing between AMI and acute myopericarditis. This technique uses the morphology of the initial portion of the ST segment/T wave. This portion of the cardiac electrical cycle is defined as beginning at the J point and ending at the apex of the T wave. Patients with noninfarctional ST segment elevation, such as acute myopericarditis, tend to have a concave morphology of the waveform while AMI frequently produces either an obliquely flat or convex shape; however, this approach is fallible and should be used only as another useful electrocardiographic analysis tool. In addition, other electrocardiographic features that may be of value in discriminating between the two

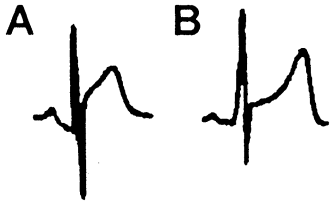

Figure 5. A comparison of BER and acute myopericarditis. Example A was obtained from a patient with acute myopericarditis; note the PR segment depression with concave ST segment elevation. Example B represents BER with its classic J point elevation, ST segment elevation, markedly concave initial ST segment/T wave morphology, and prominent T wave.

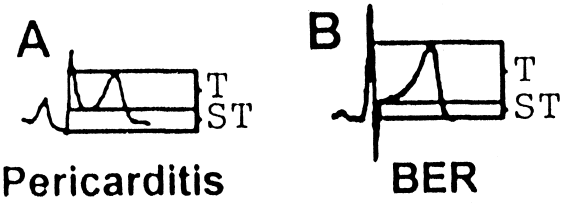

Figure 6. This PR segment–ST segment discordant ratio may also help in discriminating between the ST segment elevation resulting from BER and acute myopericarditis. It is objectively assessed by comparing the heights of the ST segment and T wave in lead V6: the ST segment/T wave magnitude ratio. Using the PR segment as the baseline for the ST segment and the J point as the beginning of the T wave, the heights are measured with calculation of the ratio. If the ratio is 0.25 or greater, pericarditis is the likely diagnosis; with results less than 0.25, one should consider BER.

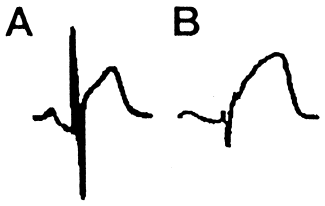

Figure 7. A comparison of acute myopericarditis and AMI. Example A was obtained from a patient with acute myopericarditis: note the PR segment depression with concave ST segment elevation. Example B represents AMI with a convex morphology to the ST segment elevation.

syndromes include T wave inversions, Q waves, and PR segment characteristics. ST segment elevation often occurs with T wave inversion in the setting of AMI. Such inversions occur only after ST segment elevation has resolved (after Stage I) in acute myopericarditis. Q waves can be present in AMI but are rare in acute myopericarditis. Conversely, PR segment depression is much more suggestive of acute myopericarditis than AMI.

The distribution of ST segment elevation also can help. In AMI, ST segment elevation is commonly in a territorial distribution, with reciprocal depressions in appropriate leads. In fact, the finding of anatomic reciprocal ST segment depression is highly predictive of AMI. ST segment changes in acute myopericarditis are more diffuse in distribution. Reciprocal ST segment depression can occur in leads avR and V1 in acute myopericarditis, and local myopericarditis can occur with territorial electrocardiographic changes, particularly with postinfarct myopericarditis.

Finally, the evolution of electrocardiographic changes occurs over a much shorter time in the setting of AMI (hours to a few days) than in acute myopericarditis (days to weeks). Thus, serial EKGs performed during the evaluation of a patient may help in distinguishing AMI from acute myopericarditis (4).

CONCLUSION

Acute myopericarditis from diffuse inflammation of the pericardium and superficial epicardium results from repolarization abnormalities of both the atria and ventricles that can be detected on the EKG. Classically, the electrocardiographic changes have been described as an evolution through four stages involving PR segment depression, ST segment elevation, T wave inversion, and eventual normalization. These changes are distributed widely over the EKG and can occur over a period of time from days to weeks. Though atypical findings are common, specific features of these electrocardiographic changes can help in establishing the diagnosis of acute myopericarditis as opposed to other syndromes such as BER and AMI.

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