CASE REPORT

Attenuation of ECG voltage in cirrhotic patients

John E. Madias*

Mount Sinai School of Medicine of the New York University and the Division of Cardiology, Elmhurst Hospital Center, 79-01 Broadway, Elmhurst, NY 11373, New York, NY, USA

Received 17 July 2006; accepted after revision 17 November 2006

Two patients with cirrhosis (CIR), ascites (ASC), and peripheral oedema (PEROD) are presented. They were followed for many years, had multiple hospital admissions and clinic evaluations, had repeated laboratory testing, and many ECGs recorded. As their condition worsened, they developed attenuation of the ECG voltage (ATTECGV), which was more pronounced during intervals of increased fluid overload. Attenuation of the ECG voltage was decreased at times of successful diuresis and amelioration of PEROD, while abdominal paracenteses for ASC did not have any influence on the ATTECGV, suggesting that ATTECGV in patients with CIR is due to the associated PEROD, and not to ASC.

KEYWORDS
Cirrhosis; Ascites; Peripheral oedema; Electrocardiography; Electrophysiology; Attenuation of the ECG voltage; Body volume conductor; Electrical impedance

Introduction

Ascites (ASC) and peripheral oedema (PEROD), occasionally reaching the extent of anasarca, are seen in patients with cirrhosis (CIR) of the liver. The Starling forces due to portal hypertension and the resultant high hydrostatic pressure contribute to ASC and PEROD, and the hypoalbuminaemia often seen in CIR leads to a low colloid osmotic pressure which further facilitates fluid efflux from the vascular system to the ‘third space’ of the abdominal cavity and the interstitial compartment. Standard textbooks of internal medicine, cardiology, and electrocardiography do not refer to CIR as a cause of peak-to-peak amplitude of ECG voltage (ATTECGV); also a comprehensive search of the MEDLINE did not disclose any report of such association. Nevertheless, we have repeatedly observed over the past 8 years a close link of CIR and ATTECGV. Indeed, the ATTECGV in such cases is so profound that the ECGs satisfy criteria for low voltage ECG (LVECG), i.e. amplitude of R wave in limb leads ≤5 mm, and in precordial leads ≤10 mm. Patients with CIR have fluctuating ASC and PEROD, with fluctuating ATTECGV or LVECG. Two of the patients in the original description of the association of ATTECGV with PEROD* had a history of CIR, but they also had other critical illnesses; the multiple pathologies in these 2 patients, and others with CIR we have observed subsequently, have prevented us from attributing clearly the ATTECGV to the ASC and/or PEROD. Furthermore, we have not been able to differentiate the effects of the ASC or PEROD in mediating this reversible ATTECGV. We have identified 2 patients with CIR, ASC, and PEROD, with multiple hospital admissions during which they showed fluctuating ATTECGV. The multiple clinical and laboratory evaluations of these 2 patients, their long clinical course, the large series of recorded ECGs, and their response to various therapies provided evidence that the ATTECGV in patients with CIR is due to the associated PEROD, and not to ASC.

Case reports

Patient 1

A 48-year-old alcoholic patient had numerous admissions to the hospital and the emergency room and was repeatedly evaluated in the outpatient clinic over the course of 4 years, with marked ASC, PEROD, episodes of acute alcoholic intoxication, syncopal attacks following significant amounts of alcohol consumption, injuries due to falls, including rib and carpal fractures while intoxicated, and an episode of presumed spontaneous bacterial peritonitis. He had a past history of well-controlled type 2 diabetes mellitus (HbA1c), hypertension, pancreatitis, peptic ulcer disease, partial gastrectomy, and Barrett’s oesophagus. He had drunk significant amounts of vodka and beer daily for 30 years, sometimes in enormous quantities. On his multiple presentations, he was found to have either well-controlled or mildly elevated blood pressure; he was obese with a very large abdominal girth, with abdominal hernias, normal cardiovascular and pulmonary examinations, and PEROD. Review of his voluminous

* Corresponding author. Tel: +718 334 5005; fax: +718 334 5990. E-mail address: madiasj@nychhc.org

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paper and electronic records revealed repeated extensive laboratory evaluations, which showed liver transaminases to be normal or slightly elevated, no bilirubin rise, highest recorded blood alcohol level of 385.3 mg/dL, lowest albumin level of 1.49 g/dL, occasional mild anaemia, and rare hypokalaemia. Although abdominal ultrasound did not reveal ASC 4 years prior to his most recent admission, there was hepatomegaly, and the inhomogeneous increased echotexture of the liver suggested diffuse parenchymal disease. Two years later abdominal ultrasound showed a small ASC, splenomegaly, a cirrhotic liver with lobulated contour, and increased coarse echotexture with fatty infiltration, suggestive of CIR. Shortly, thereafter ASC was found to be moderate in size, and subsequently massive ASC was found during the time of the ECG of Figure 1B. The associated PEROD prompted evaluation for deep vein thrombosis that was negative. There were 32 ECGs in his file recorded over the course of 4 years. The initial ECG (Figure 1A) revealed a sum of the absolute amplitude of QRS complexes from zenith to nadir, measured to the nearest 0.5 mm from all 12 ECG leads (QRS) of 87.5 mm; his weight at that time was 195 lbs. He subsequently showed fluctuation of the amplitude of QRS complexes depending on the degree of ASC and PEROD, and whether he took his diuretics, or was more or less abusive in his alcoholic addiction. On another admission to the hospital 3.6 years later his weight was 268 lbs and his ECG revealed marked attenuation of the QRS complexes (Figure 1B), with a QRS of 37.0 mm. Following diuresis in the hospital and on outpatient basis after his discharge, his weight decreased to 210 lbs, and his QRS increased to 47.5 mm, 8 days after the initiation of therapy (Figure 1C), and 76.5 mm at long-term follow-up (Figure 1D). The patient’s ASC and PEROD often was enormous. On one hospital admission his weight was 271 lbs, and the patient underwent an ultrasound-guided abdominal paracentesis, during which 4.0 litres of fluid were removed. The ECG 4 h before (Figure 2A), and 22 h after (Figure 2B) the procedure revealed a QRS of 45.5 mm and QRS of 43.5 mm, respectively. The weight of the patient was 272 lbs before the procedure and 266 lbs the following day. An echocardiogram revealed normal left ventricular size and function, and no pericardial effusion. Multiple chest radiographs revealed normal lungs and heart, and various degrees of right and occasionally left hemidiaphragm elevations due to ASC (Figure 3A and 3B). The right hemidiaphragm elevation due to ASC led to right lobe compression atelectasis, and was so marked that it simulated right phrenic nerve paralysis, as per Radiologist’s report. Multiple abdominal ultrasound examinations showed large ASC overlying the dome of the liver (Figure 3C) and within the abdominal cavity (Figure 3D).

Patient 2

A 73-year-old woman with history of hypertension, type 2 diabetes mellitus, atrial fibrillation, cerebrovascular accident, and cryptogenic CIR (no history of alcohol or drug abuse and no evidence of hepatitis), had numerous admissions to the hospital and was followed in the outpatient clinic, particularly for ASC, PEROD, and other complications of CIR. On her multiple admissions she was hypertensive, although for the last 4 years of her life her blood pressure was well controlled; she had a large abdominal girth with a large umbilical hernia. She had undergone recently a herniorrhaphy for incarcerated umbilical hernia, which was leaking clear ascitic fluid for several weeks prior to the procedure. During the procedure much ascitic fluid leaked out of her distended abdomen, in addition to the one drained and measured, which amounted to 1.7 L. The ECG did not change with QRS of 49.5 mm before and QRS of 44.5 mm following the procedure (Figure 4A and B); the weight of the patient had decreased only by 2.5 lbs, from 132.5 lbs before and 130.0 lbs after the procedure, since she both lost much ascitic fluid and received perioperative fluid infusions. She had undergone multiple abdominal paracenteses over the course of her illness, and in the last year...
of her life she had weekly such procedures. On one such occasion she had 10 L of ascitic fluid removed, and this was followed by repeat weekly paracenteses, while her PEROD was becoming more pronounced, as could be assessed by physical examination, and with her weight fluctuating between 137 and 150 lbs. These repeated removals of ascitic fluid did not lead to any augmentation of the ECG voltage; on the contrary during this particular time interval of 2 months her $\Sigma QRS$ decreased from 55.5 to 45.5 mm ($\text{Figure 5A and B}$), as her PEROD worsened. She had received, on a regular basis, infusions of albumin, while she was undergoing frequent paracenteses, and diuresis with furosemide and spironolactone.

The change in her ECG from the time that she had been diagnosed with CIR but did not have detectable ASC and PEROD by physical examination can be appreciated by comparing the ECGs obtained 5 years apart, with a $\Sigma QRS$ of 106.0 mm at the beginning, and $\Sigma QRS$ of 28.5 mm at the end of that period (73.1% attenuation) ($\text{Figure 6A and B}$). There were 48 ECGs in her file recorded over the course of her illness. The patient had 3 transthoracic and 2 transoesophageal echocardiograms over the course of the last 8 months, which showed calcification of the mitral annulus, dilatation of the right and left atria and the right ventricle, normal left ventricular size and function, a patent foramen ovale, and no pericardial effusion. Doppler

![Figure 2](https://academic.oup.com/europace/article-abstract/9/3/175/616905/Figure_2)

**Figure 2** Patient 1: ECGs showing no change in the voltage before (A) and after (B) abdominal paracentesis.

![Figure 3](https://academic.oup.com/europace/article-abstract/9/3/175/616905/Figure_3)

**Figure 3** Patient 1: Chest radiographs showed elevation of the right diaphragm (A) and (B); abdominal ultrasound studies showed large ASC (C and D).
studies revealed severe tricuspid regurgitation, and the pulmonary systolic pressure was estimated to be 40–50 mm Hg.

In the last 2 years, she had 2 abdominal ultrasonograms showing echotexture suggestive of CIR, a subsequent small and moderate ASC and splenomegaly. Shortly, thereafter she had an ultrasound of her legs, which did not disclose deep vein thrombosis. This was prompted by the PEROD, and the concern that her right ventricular dysfunction could have been due to pulmonary embolism with resultant pulmonary hypertension. In the last 4 months of her life the patient deteriorated and had multiple hospital admissions with severe ASC, PEROD, fevers, and anoxic encephalopathy, eventually dying in hepatorenal syndrome. During that time interval her portable chest radiograph did not disclose pulmonary congestion, showed mild cardiomegaly (Figure 7A), and prior to her demise severe ASC with elevation of both hemidiaphragms (Figure 7B), although the involvement of the left hemidiaphragm was not apparent in Figure 7B due to rotation and position of the patient. Computerized tomography scan of her chest showed cardiomegaly, predominantly of the right ventricle without pleural effusions (Figure 7C). Computerized tomography scan of the abdomen showed ASC, lobulated liver suggestive of CIR, and splenomegaly (Figure 7E). A pelvic ultrasound showed massive ASC (Figure 7D). An abdominal radiograph revealed intense diffuse haziness indicative of ASC (Figure 7F). The lowest albumin level noted in her medical record was 2.6 gm/dL. This patient was felt to

Figure 4 Patient 2: ECGs showing no change in the voltage before (A) and after (B) abdominal paracentesis.

Figure 5 Patient 2: In spite of abdominal paracenteses, and due to worsening PEROD, some ATTECGV developed (A and B).
have either cryptogenic or cardiac CIR. Although the latter seems less likely to be the cause of CIR, it is still possible that her hypertension had led to left ventricular diastolic dysfunction, pulmonary hypertension, right systolic dysfunction, tricuspid regurgitation, right ventricular dilatation, and passive liver congestion, leading to CIR.

Discussion

What is new about this report is that PEROD in these 2 patients with CIR led to ATTECGV, and actually LVEC. This ATTECGV was similar to the one noted in patients with PEROD of different aetiologies,6 patients on chronic haemodialysis before each dialysis,7 and patients with congestive heart failure,8–12 and involved both the limb and the precordial leads, as previously noted.6 Peripheral oedema involves the entire body, and thus both limb and precordial leads are affected. Occasionally, when patients with severe PEROD, cared for in critical care environments, are kept constantly in the supine position, there is a ‘gravity effect’ manifest in the ATTECGV, where the lateral limb leads are more affected than the right precordial leads.6 Although ASC and PEROD were associated with ATTECGV in these 2 patients, the following observations suggested that

Figure 6 Patient 2: Severe ATTECGV took place in the course of 5 years with ASC and PEROD; of note is that ATTECGV involved both the limb and the precordial leads (1B and 1C).

Figure 7 Patient 2: Chest radiographs (A and B), chest CT scan (C), pelvic ultrasound (D), abdominal CT scan (E), and abdominal radiograph (F), showing some cardiomegaly, CIR, and extensive ASC.
it was PEROD that mediated ATTECGV, while ASC did not have any observable effect on the ECG voltage: (i) Patient 1 had augmentation in ECG voltage with diuresis at different times both in the hospital and in the outpatient follow-up, in association with loss of weight and receding PEROD. (ii) Abdominal paracenteses with removal of ascitic fluid in both patients did not alter the ECG voltage. (iii) Repeated abdominal paracenteses with drainage of ascitic fluid in Patient 2 did not lead to augmentation of the ECG voltage, but further attenuation as her PEROD worsened. (iv) There was profound ATTECGV in both patients between early and later phases of their disease, developing progressively pari passu with the intensification of PEROD. (v) Attenuation of the ECG voltage was noted in both patients when there was evidence of PEROD or weight gain, irrespective of the degree of ASC, which varied in different hospital admissions, and was occasionally even minor, as assessed by abdominal radiographs and ultrasound studies. (vi) Large changes in the degree of ASC without appreciable changes in PEROD over time in both patients did not produce changes in the ECG voltage.

Ascites and PEROD in CIR have not been entirely elucidated. It is clear that patients with CIR do not develop only ASC, but they also manifest PEROD throughout the body. The mechanism of ASC accumulation is attributed to obstruction in the hepatic lymphatics, portal hypertension, and hypoalbuminemia; what initiates the excessive sodium and water accumulation initially in the abdominal cavity, and then the entire body, is not well established, but three theories, that of ‘underfilling’, ‘overflow’, and ‘peripheral arteriolar vasodilation’ have been proposed. The eventual enormous salt and water retention involves in addition to the hepatic pathology, the kidney, the sympathetic nervous system, the renin-angiotensin-aldosterone system, nitric oxide, and hypoalbuminemia.

The mechanism of ATTECGV in patients with CIR and PEROD appears to be identical to the one affecting patients with other oedematous states, and is attributed to the mediating influence of the changed passive volume conductor surrounding the heart; the sequestration of fluid in the body tissues and organs enhance electrical conductivity and decrease electrical impedance, with resultant decrease in the ECG potentials recorded at the body surface. As per this model patients with CIR are expected to develop ATTECGV as they suffer PEROD. Early in the course of their illness, this may not be appreciated on physical examination, since patients often accumulate several litres of fluid before PEROD becomes clinically apparent. However, an increase in weight provides evidence of the developing fluid overload. Diuresis leads to loss of weight and attenuation in the ECG voltage, as noted clearly in Patient 1. On the other hand, drainage of ascitic fluid, by abdominal paracentesis, reduces temporarily fluid in the ‘third-space’ (the abdominal cavity), and it is not expected to impact the ECG electrical potentials, as was noted in both patients.

Attenuation of the ECG voltage is gradual in patients with CIR and PEROD appears to be identical to the one affecting patients with other oedematous states, and is attributed to the mediating influence of the changed passive volume conductor surrounding the heart; the sequestration of fluid in the body tissues and organs enhance electrical conductivity and decrease electrical impedance, with resultant decrease in the ECG potentials recorded at the body surface. As per this model patients with CIR are expected to develop ATTECGV as they suffer PEROD. Early in the course of their illness, this may not be appreciated on physical examination, since patients often accumulate several litres of fluid before PEROD becomes clinically apparent. However, an increase in weight provides evidence of the developing fluid overload. Diuresis leads to loss of weight and attenuation in the ECG voltage, as noted clearly in Patient 1. On the other hand, drainage of ascitic fluid, by abdominal paracentesis, reduces temporarily fluid in the ‘third-space’ (the abdominal cavity), and it is not expected to impact the ECG electrical potentials, as was noted in both patients.

In the case of Patient 1, with only mild or mostly well-controlled hypertension, and preserved left- and right-ventricular function during multiple hospital and clinic visits, it is unlikely that other conditions than complications of CIR led to his profound ATTECGV. Patient 2 needs some elaboration, since in addition to her CIR there was right ventricular dysfunction with tricuspid regurgitation. Indeed, Patient 2 was included herein to underscore the importance of dissection the cause of PEROD when multiple pathologies, including CIR, are present in a patient. It is conceivable that right ventricular dysfunction present in Patient 2 led to cardiac CIR; however, for the last 4 years her blood pressure was well controlled, and the enormity of her ASC and CIR pointed to non-cardiac CIR, or cryptogenic CIR, since no definitive cause had been identified. Also it is possible that her CIR had a dual pathophysiology. In both patients, the underlying hypoalbuminemia probably contributed to the intensification of PEROD, resulting in ATTECGV, and ASC as was previously shown.

Beyond the pathophysiological insight that this link between ATTECGV and PEROD, complicating CIR provides, this association has clinical implications: (i) Attenuation of the ECG voltage seen in patients with CIR can be attributed to PEROD, and can be considered as an index of severity of CIR. (ii) The ECG can be employed for the follow-up of patients with CIR, independent of weight, since changes in the former reflect the oedematous state of the patient, while changes in the latter may include ASC. (iii) Weighing the patient often is performed in a cursory fashion, and errors are associated with recording and measuring it in different settings, with different scales, with the patient wearing different clothes. Thus, weights are notoriously inaccurate, and serial ECGs can provide an index of parallel evaluation of fluid retention in patients with CIR.

(iv) The ATTECGV also could provide a measure of volume overload in patients with CIR, even in the absence of apparent evidence of PEROD on physical examination, as noted above. (v) Finally, knowledge of this phenomenon in patients with CIR prevents attribution of ATTECGV to pericardial effusion, or other specific, or unknown cardiac, or non-cardiac illnesses.

References