CASE REPORT

Total electrical alternans in a patient with malignant pericardial tamponade

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SUMMARY

We present a case of a 59-year-old man with acute abdominal pain and progressive shortness of breath. A focused assessment with sonography for trauma scan showed free fluid in the hepatorenal recess and in the recto-uterine recess. Later, due to clinical deterioration and the differential possibility of a pulmonary embolism or aortic dissection, a CT scan was performed which revealed large pericardial effusion. This, together with a dilated vena cava inferior and portal system, raised the suspicion of cardiac tamponade. The diagnosis was confirmed by transthoracic echocardiography (TTE). In retrospect, the ECG at admission showed a sinus tachycardia, low-voltage QRS complexes and a total electrical alternans corresponding with the swinging heart seen on TTE. An electrical alternans on ECG is an important diagnostic clue but is often missed, causing an unnecessary delay to proper diagnostic and therapeutic measures.

BACKGROUND

Total electrical alternans consisting of alternation of the P wave as well as the QRS and ST wave is a rare ECG finding. This ECG sign has a broad differential diagnosis including various rhythmic disorders, such as supraventricular tachycardia and bundle branch blocks. The most common form is electrical alternans associated with pericardial effusion. Such electrical alternation reflects mechanical oscillation of the heart in the pericardial fluid: the 'swinging heart' phenomenon.

As in the presented case, this diagnostic clue is often missed, causing an unnecessary delay to proper diagnostic and therapeutic measures.

CASE PRESENTATION

We present a case of a 59-year-old man, with a medical history of marijuana dependence and schizophrenia. His regular medication included penfluridol, and he was a heavy smoker (over 50 pack years). He was presented to our hospital emergency room with colicky abdominal pain in the right upper quadrant and epigastric area with urge to move, nausea, vomiting, decreased appetite and increasing shortness of breath for the past 2 days. On examination, the patient was tachypnoeic (with a frequency of 28 per minute), peripheral oxygen saturation was 91% with 15 L of O₂ via a non-rebreather mask, and he had an increased heart rate of 110 beats per minute. There was no sign of hypotension, however, a narrow pulse pressure

(128/103 mm Hg) and elevated jugular venous pressure were observed. His heart sounds were classified as normal. No pulsus paradoxus was reported. The patient had a maximum score on the Glasgow coma scale and was oriented in time, place and person. He had a normal temperature (37.1°C) and his glucose level was 8.6 mmol/L. He had crackles at the base of the right lung and palpation of the right upper quadrant of the abdomen revealed direct tenderness.

INVESTIGATIONS

The admission ECG ([figure 1](#)) showed a sinus tachycardia of 110 beats per minute, low voltage QRS complexes in the anterior leads and no signs of acute ischaemia. On focused assessment with sonography for trauma (FAST) scan, performed by a trained resident of internal medicine, free fluid was observed in the hepatorenal recess and in the rectouterine recess. There was no sign of an abdominal aortic dissection. Pericardial fluid was not observed. The gallbladder was possibly distended.

Blood investigations revealed haemoglobin 15.0 g/dL (13.7–17.0 g/dL), total leucocyte count $18.6 \times 10^9/L$ ($4.0\text{--}10.0 \times 10^9/L$), platelet count $285.0 \times 10^9/L$ ($150.0\text{--}450.0 \times 10^9/L$), C reactive protein 49.3 mg/L (<5.0 mg/L), lactate 9.5 mmol/L ($0.4\text{--}2.0$ mmol/L), aspartate aminotransferase 906.0 U/L (<40.0 U/L), alanine aminotransferase 996.0 U/L (<45.0 U/L), alkaline phosphatase 172.0 U/L ($40.0\text{--}120.0$ U/L) and gamma-glutamyl transferase 217.0 U/L (<60.0 U/L), serum creatinine 122.0 $\mu\text{mol/L}$ ($75.0\text{--}110.0$ $\mu\text{mol/L}$), urea 13.5 mmol/L ($2.1\text{--}7.1$ mmol/L), albumin 46.0 g/L ($35.0\text{--}50.0$ g/L) and lactate dehydrogenase (LDH) 511.0 U/L (<248.0 U/L). The arterial blood gas revealed a wide-anion-gap metabolic acidosis caused by the high lactate level and a high A–a gradient respiratory alkalosis exhibiting a ventilation–perfusion mismatch. Cardiac biomarkers such as NT-proBNP, troponin and CK-MB were not measured. Because of acute deterioration due to dyspnoea with high oxygen demand, a CT angiogram was made to exclude a pulmonary embolism and aortic dissection. This scan displayed bilateral pleural effusions, a large pericardial effusion and multiple enlarged lymph nodes of the right hilum and mediastinum. It also showed a dilated vena cava inferior and portal system suggestive of backward failure. The transthoracic echocardiography (TTE) ([video 1](#)) revealed a large amount of pericardial effusion with swinging of the heart freely in an anterior–posterior fashion and an increased

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Figure 1 ECG at admission showing sinus tachycardia of 110 beats per minute, low voltage QRS complexes in the anterior leads and no signs of acute ischaemia. In retrospect, an electric alternans was seen. Note, the alternating height of the P–QRS–T complexes.

respiratory inflow variation over the mitral valve, consistent with cardiac tamponade. Left and right ventricular systolic function was not impaired. In retrospect, the 12-lead ECG did demonstrate an electrical alternans of the P–QRS–T complexes.

DIFFERENTIAL DIAGNOSIS

A wide variety of conditions can result in moderate to large pericardial effusion. Although there is substantial heterogeneity between studies investigating the aetiology and subsequent incidence of moderate to large effusion, the differential diagnosis includes: neoplastic, infectious (viral/bacterial), uraemia, post-myocardial infarction, post-cardiovascular surgery, post-radiation and idiopathic.

TREATMENT

The patient initially received antibiotics (gentamycin, ceftriaxone and metronidazole) as treatment of (severe) sepsis with abdominal focus. The patient was transferred to the intensive care unit for haemodynamic stabilisation. He received a therapeutic pericardiocentesis where 900 mL of serosanguineous fluid was subsequently drained. Following extended drainage, the symptoms as well as the abnormal ECG and echocardiographic findings resolved (figure 2). No quick reaccumulation of pericardial effusion was observed on TTE after removal of the pericardial drain.

OUTCOME AND FOLLOW-UP

Pericardial fluid analysis revealed a total protein content of 55.3 g/L and LDH of 2399.0 U/L in combination with an albumin level of 37.0 g/L, which is associated with an exudate. Microbiological analyses of the pericardial fluid showed no infectious agent, excluding a viral or bacterial origin. Pericardial fluid cytology was suggestive of a malignant aetiology. All peripheral blood cultures were negative. A follow-up Positron Emission Tomography-CT scan exposed a small lesion of 1.8 cm

in the base of the inferior lobe of the right lung with moderate to greatly elevated FDG uptake, suspected for primary lung carcinoma. In addition, extensive hypermetabolic lymphadenopathy of the right hilum, mediastinum, supraclavicular and intra-abdominal was seen, indicative for lymph node metastases. The patient remained clinically well post-pericardiocentesis. He was discharged 7 days post-admission.

The pathological diagnosis was pT1aN3M1a, stage IVA non-small cell lung adenocarcinoma, for which palliative chemotherapy was initiated. Four cycles of carboplatin/pemetrexed were advised. However, due to side effects and a mixed response, the chemotherapy was discontinued after three cycles. Best supportive care was given. Unfortunately, the patient passed away within 16 weeks following his diagnosis.

DISCUSSION

Electrical alternans has been defined as an alternation in the configuration of the ECG complexes arising from the same pacemaker.¹ Any electrographic complex can display alternation,

Video 1 Transthoracic echocardiography, swinging of the heart.

Figure 2 ECG post-pericardiocentesis.

most commonly the QRS complex, but also the ST wave, or T wave alone and the P wave rarely.² Through the years, additional variations and aetiologies have been described with regard to electrical alternans and its association with rhythmic disorders, such as supraventricular tachycardia and bundle branch blocks.³ However, when all main complexes alternate simultaneously the phenomenon is called total electrical alternans.² This rare ECG finding is associated with pericardial effusion and tamponade, but when present it is of important diagnostic value.⁴ The changes in shape and amplitude of the ECG complexes have been attributed to excessive cyclic and rotational cardiac motion within an enlarged fluid-filled pericardial sac. The ECG electrodes detect this cyclic change in relation to the relative anatomical position of the heart resulting in the typical alternation.^{3 5} Interestingly, this cycle repeats itself after every heartbeat; 2:1 alternans. Echocardiographic studies demonstrate that cardiac oscillation is already present before the onset of tamponade but in a frequency similar to the heart rate, and therefore not visible on the ECG as an electrical alternation.¹ Nevertheless, when the effusion accumulates to a critical extent and cardiac tamponade supervenes, the frequency decreases to one half of the heart rate. As a result, the P–QRS–T amplitude varies periodically from one beat to the next. Conversely, electrical alternans may disappear following the removal of pericardial fluid, as seen in our case.⁶

In our case, the CT scan was decisive for the diagnosis of pericardial tamponade. Although CT provides detailed information on size and localisation of pericardial effusion and extracardiac causes of pericardial effusion, it should be regarded as second-line imaging compared with TTE. TTE is recommended as first-line imaging since it is portable, non-invasive and accurately detects pericardial effusion and cardiac tamponade.⁷ The development of cardiac tamponade depends on the rate and viscosity of fluid accumulation, the distensibility of the pericardium, the filling pressures and compliance of the cardiac chambers. Small but rapidly accumulated pericardial effusions can lead to dramatic tamponade. Whereas large pericardial effusions that accumulate

slowly can be tolerated better. To differentiate between pericardial effusion and tamponade, clinicians can correlate echocardiographic findings, such as right ventricular or atrial collapse, respiratory variation in the mitral and tricuspid flow and inferior vena cava plethora, with the symptoms of clinical tamponade, such as dyspnoea, hypotension, tachycardia, elevated jugular venous pressure and pulsus paradoxus.⁸ A recent study among patients with large malignant pericardial effusion, demonstrated tricuspid valve annular plane systolic excursion and peak systolic annular velocity at the lateral margin of the tricuspid valve annulus as independent predictors for the development of cardiac tamponade.⁹

Regarding our case, electrical alternans has been shown to be more prevalent in patients with large effusions of relatively viscous fluid such as malignant pericardial effusion.¹⁰ Furthermore, the occurrence of cardiac tamponade, in contrast to the general population is relatively common in patients with malignant pericardial effusion, has a poor prognosis and a patient remain at risk for recurrence of tamponade following pericardiocentesis.¹¹ In cases with reduced life expectancy, surgical interventions such as a pericardial window can be considered in patients with symptomatic effusions in whom medical therapy and repeated pericardiocentesis were not successful.⁷ In hindsight, the 12-lead ECG did demonstrate a true 2:1 electrical alternans of the P–QRS–T complexes in combination with a sinus tachycardia, not readily recognised at first. In addition, pericardial fluid was not detected by FAST scan due to premature closure when free intraperitoneal fluid was observed in a patient in severe respiratory distress. Our case illustrates the importance of carefully analysing the admission ECG, completion of the FAST scan protocol and to contemplate the clinical condition of the patient as a whole. The electrical alternans in combination with the acute deterioration of the clinical condition was in retrospect highly suggestive of a cardiac tamponade. Although total electrical alternans is uncommon, it is a very specific marker of cardiac tamponade and seen relatively often in malignant pericardial disease.

Learning points

- ▶ Total electrical alternans of the P–QRS–T complexes is very specific for cardiac tamponade.
- ▶ Completion of the focused assessment with sonography for trauma scan protocol is of vital importance to avoid premature closure and hence a delay in the diagnosis or adequate treatment.
- ▶ Despite the absence of chest pain and ECG signs of myocardial ischaemia, cardiac biomarkers should be included in the initial workup of patients with severe respiratory distress.
- ▶ The incidence of cardiac tamponade in the general population is low, but the occurrence is relatively common in patients with malignant pericardial effusion.

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