CHAPTER

13

ECG in Non-cardiac Conditions

M Chenniappan, Kader Sahib

INTRODUCTION

Since its discovery in 1902 by William Einthoven, the electrocardiogram (ECG) has served as the most cost effective investigation. Its usefulness in cardiac conditions, both in coronary and non coronary heart disease is well established. However, most often it is believed that the ECG is a cardiac investigation, utilised only for diagnosing cardiac condition. The beauty of ECG is that it can provide valuable information in variety of non-cardiac conditions also. In this article we explore the usefulness of ECG in many non cardiac situations.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

This ECG is very useful in COPD to assess the prognosis. A peculiar ECG sign in COPD is 'Lead I sign' or 'Schamroth sign' which is low voltage P, QRS, T in LI because of vertical axis of all the vectors[1] (Figure 1). The ECG may also show right ventricular hypertrophy and right axis deviation which are the signs of cor pulmonale where the prognosis is bad. In addition to this, the symmetrical T inversion in chest leads may be due to right ventricular ischemia rather than coronary artery disease which also indicates a bad prognosis.

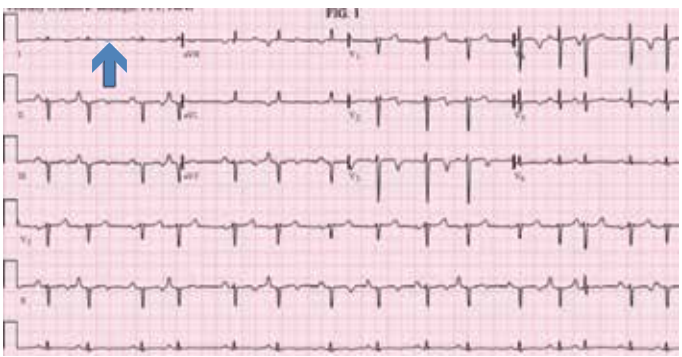

Fig. 1: ECG showing Schamroth sign (Arrow)

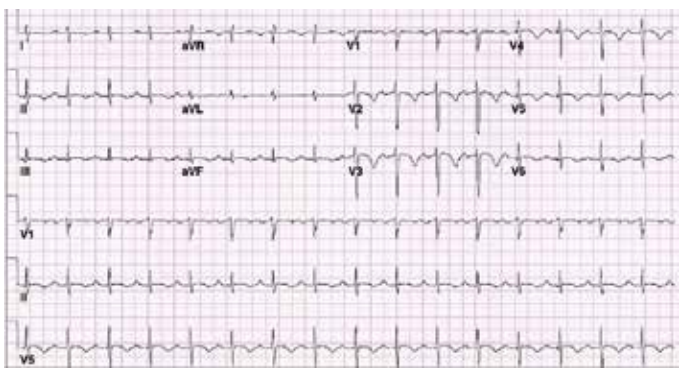

Fig. 2: ECG in acute pulmonary embolism showing symmetrical T inversion in V1 –V4

Pulmonary Thrombo Embolism

There are many ECG signs described in pulmonary thromboembolism. The most important and common ECG

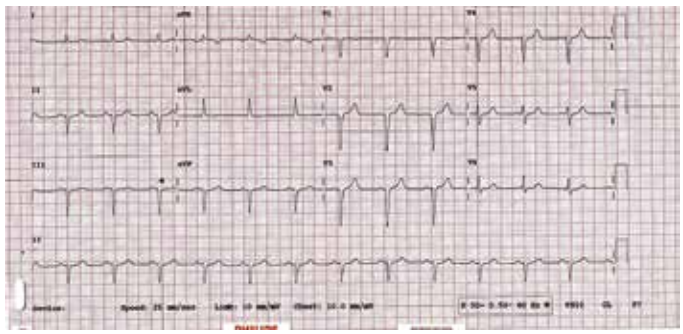

Fig. 3: ECG mimicking anterior MI in a patient with skeletal abnormalities

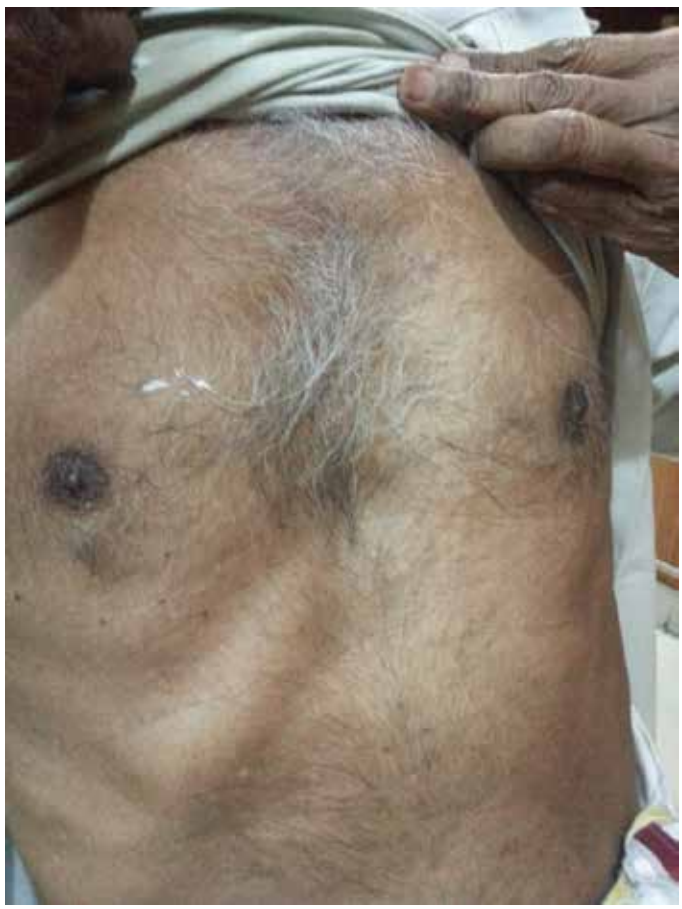

Fig. 3a: The patient showing skeletal abnormality whose ECG is shown in Fig 3.

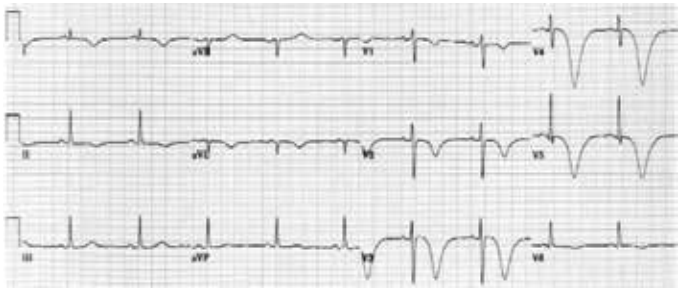

Fig. 4: Deep broad T inversion with prolonged QT interval in SAH

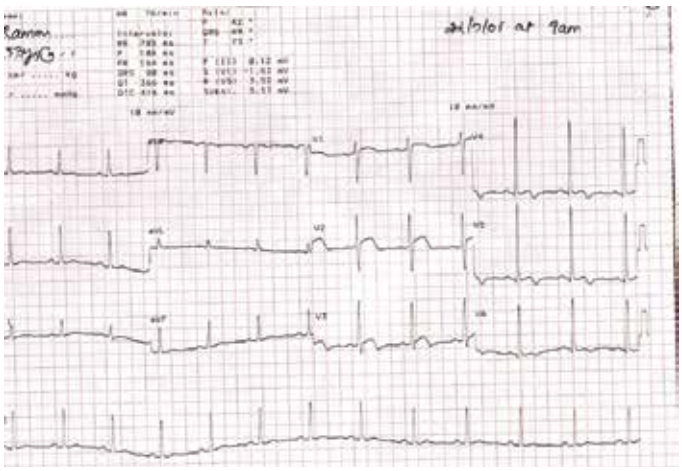

Fig. 5: ECG in a patient with acute pancreatitis showing ST coving mimicking acute coronary syndrome

sign is symmetrical inversion of T wave in anterior chest leads[2] (Figure 2). This is due to right ventricular ischemia and dilatation where Right ventricle occupies region of V1-V3. This ECG sign in appropriate clinical setting not only establishes the diagnosis but also indicates poor response to treatment as well as poor prognosis.

SKELETAL ABNORMALITIES

ECG may be abnormal due to skeletal abnormalities such as kyphoscoliosis. The common ECG sign is non progression of R wave in chest leads due to shifting of the heart (Figure 3 & 3a) Non progression of R wave is defined as R wave less than 3mm in V3 when chest electrodes are correctly placed. In this situation, taking ECG one space below or above may increase the R wave voltage in V3 in which case anterior MI as the cause of non-progression of R wave is unlikely.

CENTRAL NERVOUS SYSTEM DISORDERS (CNS)

ECG can be abnormal in certain CNS disorders. Sub arachnoid haemorrhage (SAH) and some cases of stroke usually produce deep, broad T inversion (Figure 4). CAD also produces deep T inversion in chest leads. But in SAH, T inversion is deep; broad with prolonged QT interval[3] (Figure 4). Rarely in SAH, ECG may show ST elevation mimicking acute ST elevation MI. This is due to excessive catecholamines released from brain producing extensive myocardial injury. Thrombolysis here is disastrous. In some patients with vertebro basilar insufficiency, atrial fibrillation can occur.

GASTRO INTESTINAL DISORDERS (GID)

Some GID may also produce ECG changes. Acute

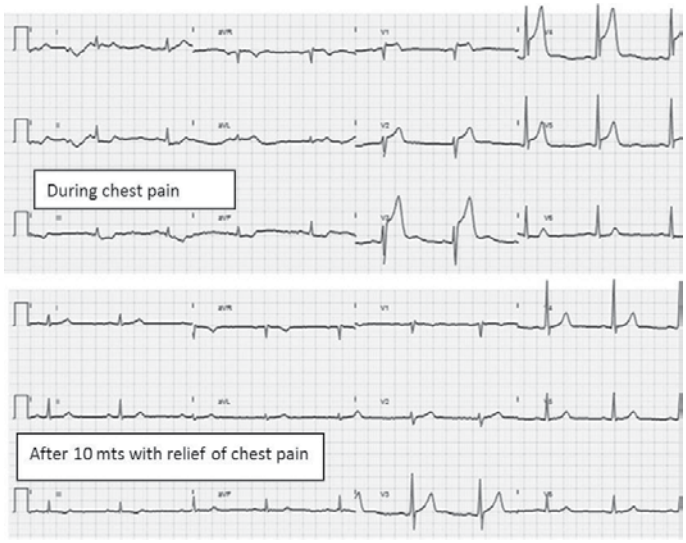

Fig. 6: A patient with esophageal spasm showing transient ST elevation due to transient coronay spasm

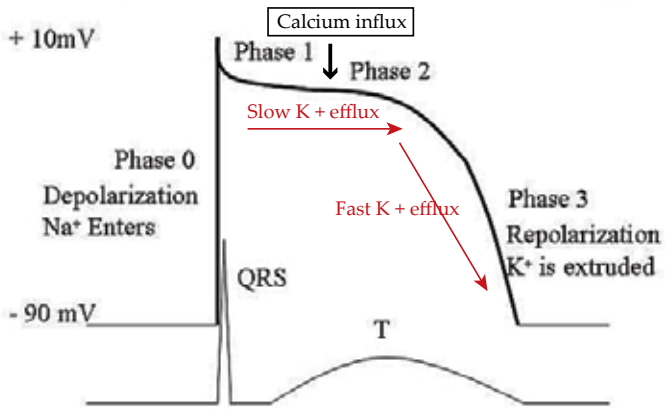

Fig. 7: Relationship between action potential, movement of ions and ECG.

pancreatitis can sometimes produce ECG changes mimicking acute coronary syndrome (Figure 5) The ECG changes in pancreatitis are due to proteolytic enzymes released by pancreas injuring the myocardium. The clinical correlation with ECG interpretation in this situation is crucial as the treatment given for Acute Coronary Syndrome will worsen pancreatitis. In some patients, oesophageal disorders not only mimic CAD but can also produce ECG changes[4] due to associated coronary spasm known as 'Linked Angina' (Figure 6)

ELECTROLYTE DISTURBANCES

Electrolyte disturbance can cause significant ECG changes. The relationship between active potential and ECG is shown Figure 7. The QRS corresponds to sodium entry, calcium to ST segment and potassium to T wave.

Potassium

Hyperkalaemia initially produces Tall T waves (Figure 8), with increasing levels producing P and QRS changes[5]. The ECG changes appear beyond 6mEq/L. When hyperkalaemia produces tall T waves, it may be mistaken for acute subendocardial ischemia (Figure 9). Hyperkalaemia produces Tall T with narrow base and

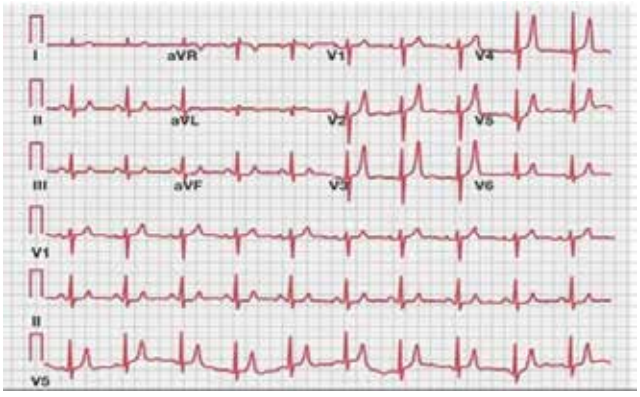

Fig. 8: ECG showing hyperkalaemia - Tall T with a narrow base and sharp apex

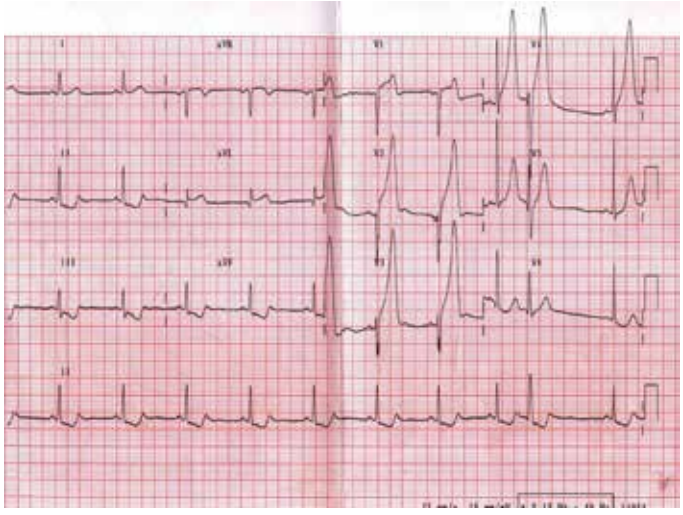

Fig. 9: ECG showing tall T waves due to sub endocardial ischemia. (Broad base with blunt apex)

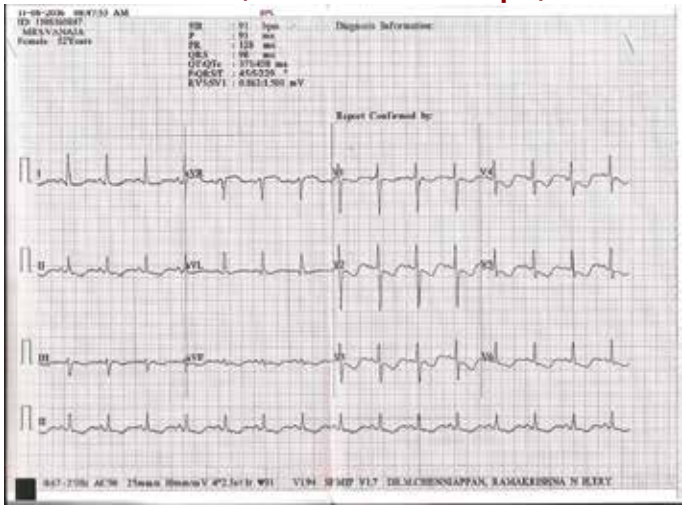

Fig. 10: ECG in severe hypokalaemia showing down sloping ST depression, low voltage T wave and prominent U extending into next P wave

sharp apex; acute ischemia produces Tall T waves with wide base and blunt apex.

Hypokalaemia: Hypokalaemia produces low voltage T waves with prominent U waves. Usually the ECG changes occur when potassium is <2.7mEq/l. Whenever

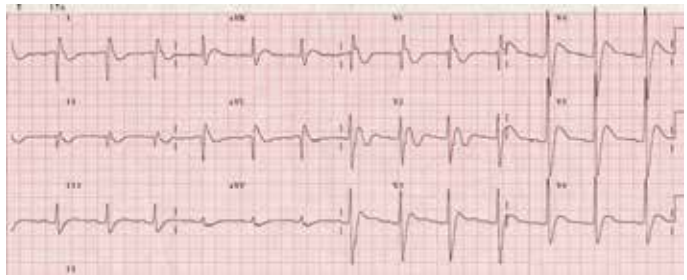

Fig. 11: ECG showing short QT due to shortened ST segment interval due to hypercalcemia

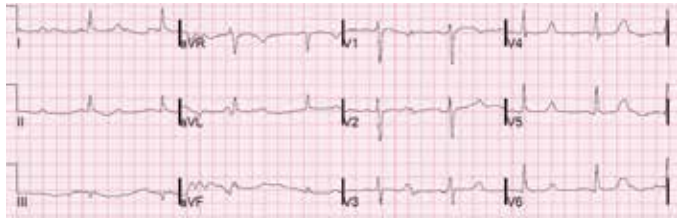

Fig. 12: ECG showing Prolonged ST segment due to hypocalcaemia

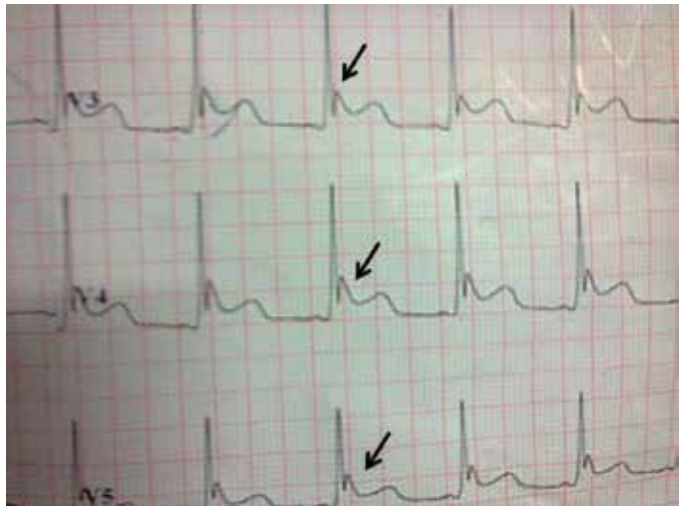

Fig. 13: Hypothermia showing Osborn Wave. (Arrow).This hypothermia was due to paracetamol poisoning

there is a low voltage T wave, one should look for 'u' wave to rule out hypokalaemia. When K is less than 1.7 mEq./L, it produces significant ST depression, low voltage T and prominent U mimicking acute coronary syndrome[5](Figure 10).The apparent QT prolongation in hypokalaemia differentiates it from acute myocardial injury.

Calcium

The abnormalities in calcium produce ST changes. Hypercalcemia produces short QT interval due to a short ST segment and hypocalcaemia produces prolonged QT interval due to a prolonged ST segment[5] (Figures 11, 12).

Digoxin produces short QT interval due to shortening of ST segment because of intracellular hypercalcemia.

HYPOTHERMIA

Hypothermia is defined as core body temperature below 95° Fahrenheit. ECG changes appear below 90° F and when the temperature approximates 86° F, 80% of patients show an extra deflection at the end of QRS which

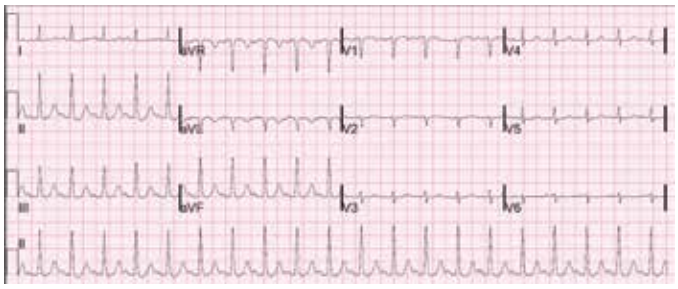

Fig. 14: ECG showing sinus tachycardia, low voltage and non-progression of R wave in chest leads due to Pneumothorax on left side shifting the heart to right side.

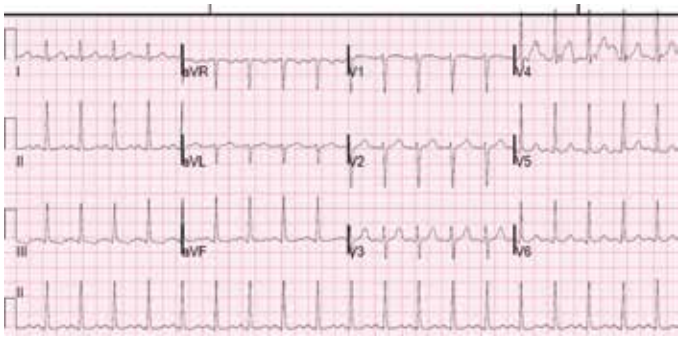

Fig. 15: ECG after the relief of Pneumothorax .Please note progression and good voltage of R wave in left sided leads

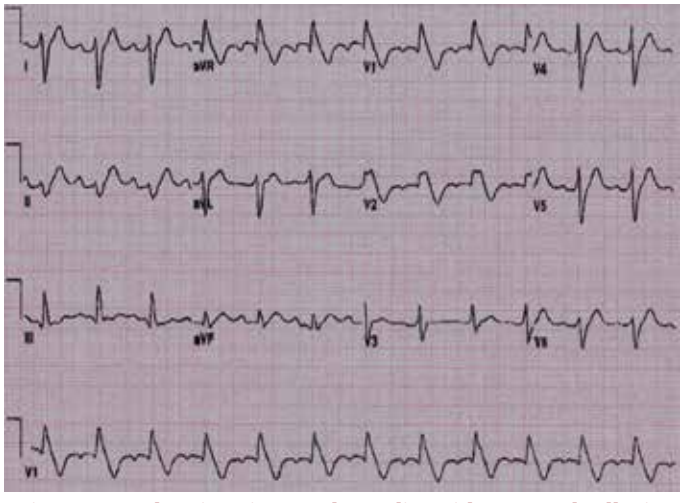

Fig. 16: ECG showing sinus tachycardia, wide QRS and tall R in avR due to tricyclic antidepressant toxicity

is known as Osborn wave[6] (Figure 13). This change which was described by Dr.John Osborn is due to the gradient of potassium current between epicardial and endocardial surfaces.

PNEUMOTHORAX

Diagnosis of pneumothorax is purely clinical. ECG changes are due to shifting of the heart which gets normalised immediately after the relief of pneumothorax. (Figure 14, 15).

DRUG TOXICITY

Many non-cardiac drugs produce ECG changes at their toxic levels. Tricyclic antidepressant toxicity typically produces wide QRS, sinus tachycardia and terminal R in avR. Terminal R wave in avR more than 3mm, QRS duration more than 100m.sec and sinus tachycardia are

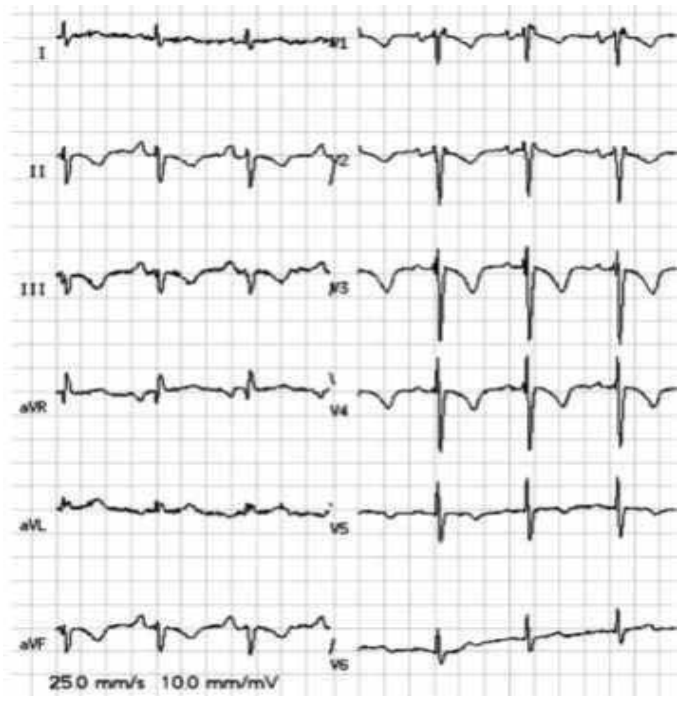

Fig. 17: ECG showing diffuse T inversion due to CO monoxide poisoning which is an indication for hyperbaric therapy

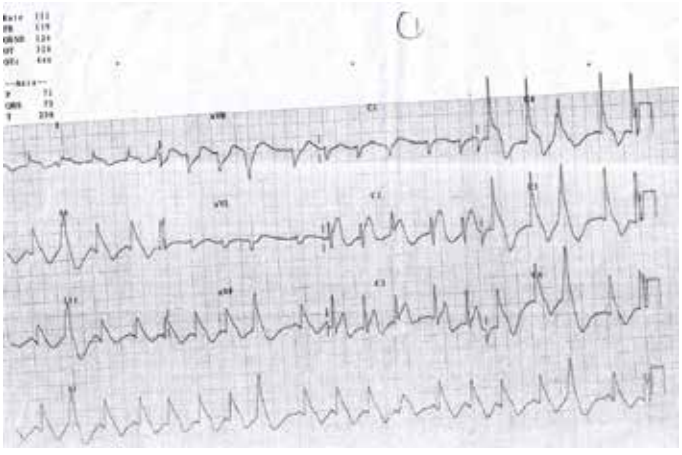

Fig. 18: ECG showing diffuse ST elevation due to ALP poisoning. (See text)

bad prognostic signs[7] (Figure 16). Many chemotherapeutic drugs especially anthracyclines cause cardiac dysfunction and induce changes of myocardial ischemia.

POISONING

Cardiac toxicity is a common finding in patients who have been poisoned with wide variety of chemical agents. Carbon monoxide (CO) poisoning typically produces ischemic changes in ECG due to inhibition of cellular respiration[8] (Figure 17).

Organo phosphorous poisoning, cyanide poisoning and heavy metal poisoning produce arrhythmias and ECG changes. One of the common insecticides which are used in South India is Aluminium Phosphide (ALP). ALP poisoning produces cellular hypoxia due to inhibition of cytochrome oxidase in mitochondria. This may produce diffuse ST elevation mimicking Acute Myocardial Infarction (Figure 18).

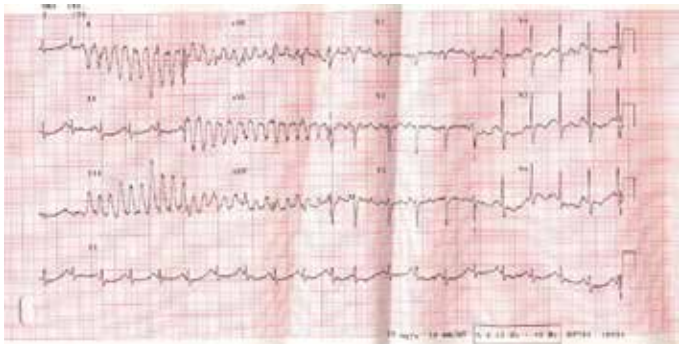

Fig. 19: ECG showing Parkinson tremor mimicking Torsade de Pointes. Note that LII which is simultaneously recorded with LI and LIII does not show same ECG changes confirming STA

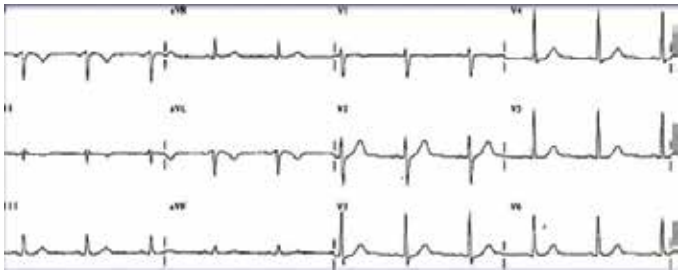

Fig. 20: Right arm and left arm lead reversal leading to positive complexes in lead aVR and negative complexes in lead I

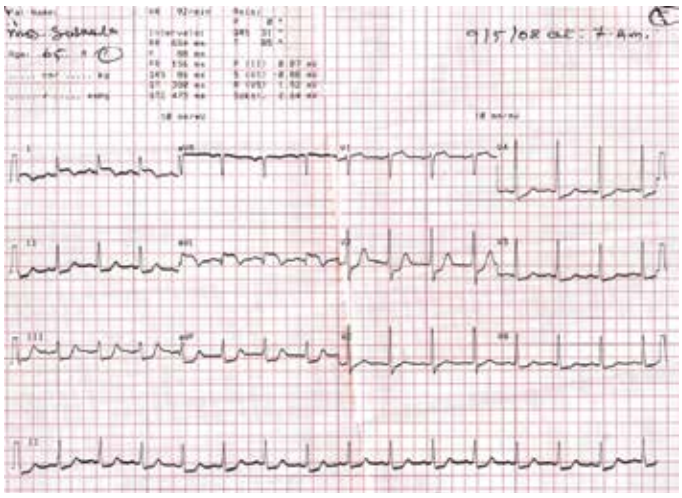

Fig. 20a: ECG showing High lateral MI like picture because of upper limb, lower limb lead reversal

TREMORS

Tremors due to various reasons especially Parkinsonism produce somatic tremor artefacts (STA). This STA will mimic arrhythmias such as atrial flutter, Torsade de pointes and may be wrongly treated with powerful antiarrhythmic agents and DC shock[9]. The clinical examination during the arrhythmia will show disparity between pulse and ECG. The ECG in Parkinsonism is shown in Fig.19, which exactly looks like Torsade de pointes. Careful examination of L II which is simultaneously recorded with other leads did not show the arrhythmia, confirming the diagnosis of tremors. Further careful examination of limb leads confirm that the leads using left arm such as L1, L III, and avL showed the ECG changes and not L II which is not using left upper limb indicating the tremor is maximum in left upper limb. So the ECG can be utilized not only to diagnose tremors but also the limb of tremors!

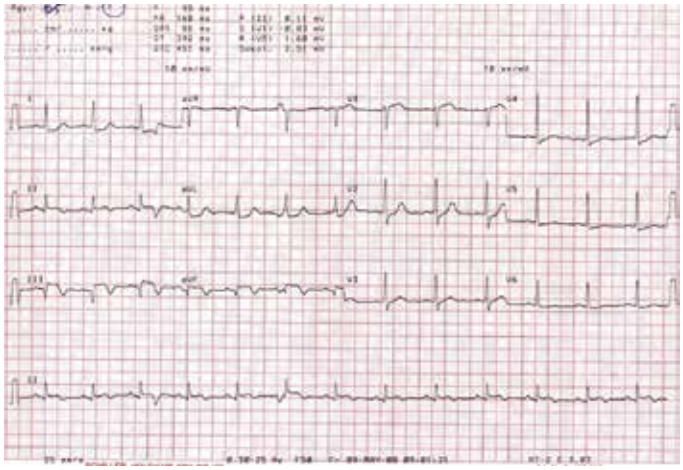

Fig.20 b. Correctly recorded ECG showing actual inferior MI

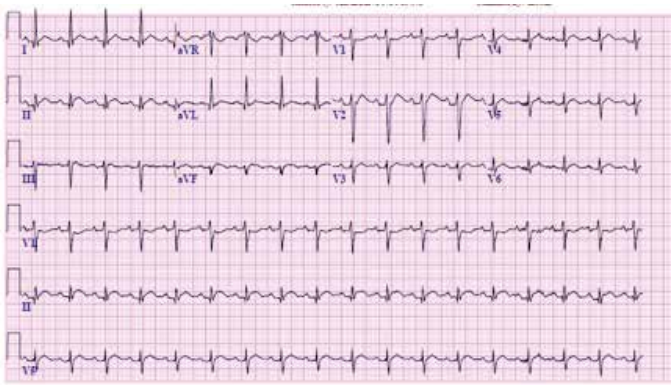

Fig. 21: ECG changes in pregnancy

Table 1: Pathologic changes in ECG in pregnancy
Sinus Bradycardia
A.V. Blocks (New onset)
Complex Premature beats
Atrial Fibrillation
Significant chamber enlargements (LA,LV,RV)
Ischemic changes(deep T inversion, ST elevation or depression)

LEAD MISPLACEMENT

Upper arm lead reversal is well known to cause technical dextrocardia where limb leads show the evidence of dextrocardia (P, QRS negative in L I and positive in avR) but chest leads show normal R wave progression (Figure 20).

Less well known is the reversal of electrodes between upper and lower limbs[10]. In Figure 20a & b upper, lower limb lead reversal actually changes site of infarction. The actual inferior wall MI is shown as high lateral MI due to upper, lower limb lead reversal.

PREGNANCY

Pregnancy produces a lot of ECG changes such as Sinus tachycardia, nonspecific ST T changes, short PR, rare

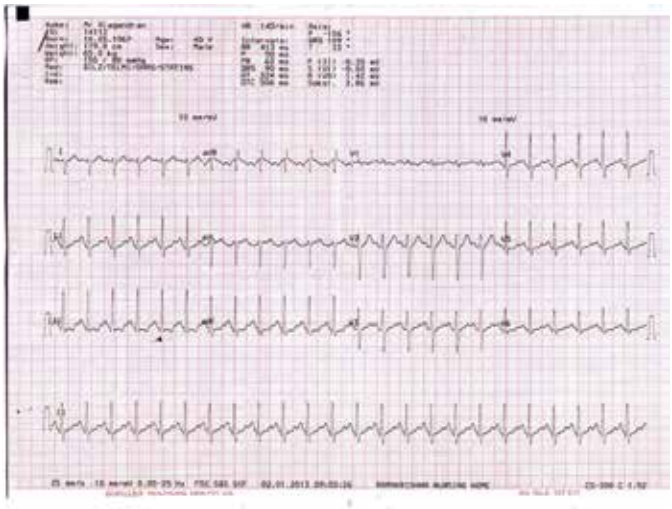

Fig. 22: ECG in erect posture;compare the ECG in lying posture (Fig. 22a)

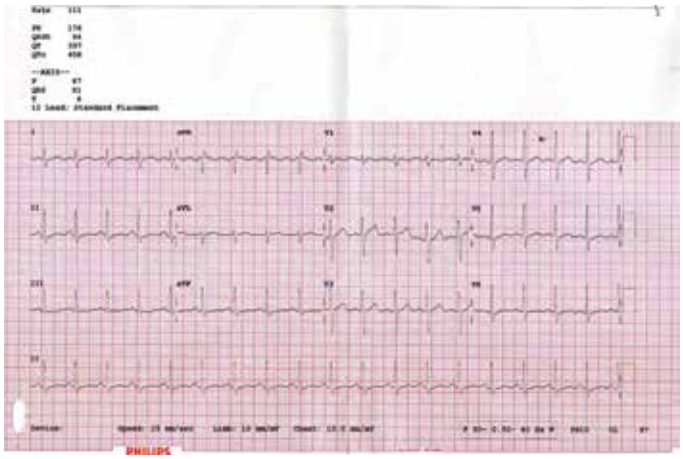

Fig. 22a: ECG of the same pt. in fig .22 in lying posture

premature beats and minor axis deviation towards left due to elevation of diaphragm[11] (Figure 21).

The pathological changes in ECG during pregnancy are listed in Table 1.

POSTURE

Changes in posture itself can produce significant ECG changes. Standing may produce T wave changes and axis shift (Figure 22 a & b); so when interpolating ECG it is important to know in which position the ECG has been taken.

RENAL DISEASE

ECG in chronic kidney disease (CKD) usually shows LVH, Left Atrial Enlargement and most often hyperkalemia[12]. Sometimes combination of electrolyte abnormalities may produce some typical ECG changes which are diagnostic of chronic renal diseases. The combination of hypocalcaemia and hyperkalaemia show prolonged ST segment (hypocalcaemia) and peak T waves (hyperkalaemia) (Figure 23). Although in this ECG, T wave is not typical of hyperkalaemia because of decreased amplitude, one must suspect associated hyperkalaemia because of T waves with a sharp apex.

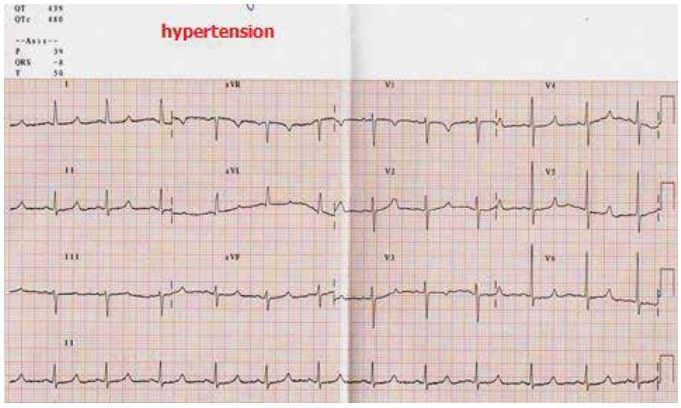

Fig 23: ECG in a CKD patient with hypocalcaemia and hyperkalaemia

CONCLUSION

Most often, whenever there are ECG changes it is presumed, it is due to cardiac disease. It should be realised that many non cardiac conditions can produce significant ECG changes which are mistaken for cardiac disease and wrongly treated especially in critical care settings. The clinical correlation, careful study of ECG and awareness of ECG changes in non cardiac conditions will prevent many such therapeutic disorders.

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