Correspondence

ECG in Hypoglycemia: Mimic Isoelectric ECG

Dear Sir,

ECG changes in Diabetes Mellitus are probably due to other associated diseases like Coronary Artery Disease, Dilated Cardiomyopathy. The most common cause of reversible transient ECG changes in diabetics, are in diabetic coma or in insulin shock (Hypoglycemia). Among both of these conditions marked cardiac disturbances may occur and may cause alteration in electrocardiogram.

Literature was reviewed, not much work had been done on this subject i.e. about the ECG changes in hypoglycemia / insulin shock. Samuel1 had established that insulin shock can produce similar changes in electrocardiogram as in hypoglycemia. The first electrocardiographic study of insulin in human beings was conducted by Wittgenstein and Mandel in 1924, showed that, initially there is inversion of T wave and later on decreases the amplitude of R wave.2 Marked Bradycardia has been recorded during insulin shock by Haynal.3 Insulin shock produced profound changes in (1) heart functions i.e. due to hypoglycemia, circulation is affected adversely, generally blood pressure is maintained and some time due to marked bradycardia circulatory support is of utmost importance;4 (2) reversible electrocardiographic changes like decrease in amplitude of wave components ( P wave, QRS complex and T waves) are recorded;5 (3) augmentation of electrical potential impairment or there is a poor circulation.6

Isoelectric electrocardiogram (ECG without P wave, QRS complex and T waves) are recorded: pathologically, when there is no cardiac activity i.e. cardiac asystole and rarely in extreme hypothermia or hypothyroidism; physiologically, when all the electrodes record electrical potential in equal concentration or conduction of electrical potential is impaired or there is a poor circulation.6

A, thin built, 60 years old lady, not a known diabetic or hypertensive, on some spurious medicines for body aches, had an episode of giddiness in the toilet in midnight, brought to the bed by her husband, went to sleep and in the morning at 7 a.m. her husband tried to awoke her but could not and finally brought her to the hospital in drowsy state at about 9 am.

On examination: she was comatose, bilateral radial pulse not felt, brachial and carotids were weak and slow (30 per minute). BP was 90/60 mm of Hg. in both the upper limbs. Respiratory rate was 18 per minute and it was shallow. Oral temperature was 99.2°F. There was no external sign of head injury. Chest revealed bilateral crackles. Heart sounds could not be clearly heard (chest was noisy). P/A soft. CNS examination revealed, coma grade II, bilateral pupil miotic, slightly reacting to light. Fundus was normal. Bilateral planters were extensor and there was no focal neurological deficit.

A, 12 lead ECG was done, the tracing did not reveal any P wave, QRS Complex and T wave (Fig. 1, apparently Isoelectric ECG) in all the leads. Again leads were applied with adequate jelly and recorded, had similar observation. Again 2 times repeated, but all in vain (to exclude any technically fault). Then ECG was recorded with another machine, but again same observation. Three times again repeated, but findings were still same. To exclude mechanical fault, with both the machines, at the same time ECG’s were recorded on another patients in the ward, which were normal. CCU facilities were not available in our hospital (secondary level health institution).

By that time blood biochemistry revealed, blood sugar (R) 23 mgm%, blood urea 36 mgm% and S. creatinine 1.4 mgm%. S electrolytes, Haemogram and Urinalysis were normal. Patient was treated with infusion of 100 ml 25% dextrose, and then 1 litter 10% dextrose fast, followed by 10% dextrose slowly and 2 litters normal saline infused with another intravenous line. At 11 a.m. She regained consciousness, blood sugar was 90 mgm%, radial pulse was of good volume with rate of 76 per minute. ECG was repeated and it was found to be normal (Fig. 2, with the same both machines). Follow-up in the hospital was uneventful, she was euglycemic without any medicine (OHA’s), Thyroid profile and CT Head was normal. She was finally discharged after 4 days.

In the present reported case, hypoglycemia (Insulin Shock), probably was due to intake of some spurious medicines presenting in the form of circulatory failure with electrocardiographic changes i.e. profound bradycardia with marked loss of amplitude of waves (P wave, QRS Complex and T waves) mimic Isoelectric electrocardiogram.2,3,6 Circulatory failure was managed with i/v normal saline and hypoglycemia with i/v dextrose, patient regained consciousness, her radial pulse was of good volume at the rate of 76 per minute and apparent isolectric electrocardiogram changes to normal electrocardiogram (recorded with the same both machines). Thus, in this case, the reversible isolectric ECG (apparent) was due to insulin shock i.e. due to poor circulation and Hypoglycemia.

India will become the capital of diabetes, with the most
ABG Clue Leads to Diagnosis of Bronchogenic Carcinoma

Sir,

We report two cases where arterial blood gas (ABG) analysis led to suspicion and detection of bronchogenic carcinoma and subsequently confirmed by imaging and fine needle aspiration (FNAC).

Case 1: A 60 years old male chronic smoker was admitted in intensive care unit on ventilator on with diagnosed as COPD with type 2 respiratory failure precipitated by pneumonitis. Initial clinical evaluation showed a cachectic, pale, drowsy, dyspneic elderly patient with bilateral crackles and rhonchi. Investigation revealed anemia (Hb-6.6mg/dl), leukocytosis, and hyperglycemia (blood glucose-283 mg/dl) with normal liver and renal function test. X ray chest showed emphysema chest with consolidation left upper zone. ABG revealed hypoxemia, hypokalemia with metabolic alkalosis with compensated rise in pCO2 (pCO2= HCO3 +15=52+15=67) (Table 1). From clinical condition the patient was expected to have acute on chronic respiratory acidosis. There was no history of vomiting, or diarrhea. He was not on diuretics or steroid therapy. In view of recent hyperglycemia, metabolic alkalosis with hypokalemia, a cachectic patient with history of smoking ectopic ACTH secretion due to bronchogenic carcinoma was strongly suspected. CECT thorax was done which revealed enhancing nodular lesion adjacent to posterior segment of RUL with pleural thickening, and a 14x10 mm thick walled cavitatory lesion in anterior segment of LUL with multiple nodular lesions in both lung fields. His overnight dexamethasone suppressed cortisol was 9.6µg/dl and serum ACTH was 523pg/ml. LDDST and HDDST could not be done due to condition of patient and associated infection. CT guided FNAC of lung confirmed diagnosis of small cell carcinoma of lung. He succumbed to his illness.

<table>
<thead>
<tr>
<th>Case</th>
<th>pH</th>
<th>PO2 (mmHg)</th>
<th>PCO2 (mmHg)</th>
<th>HCO3 (meq/L)</th>
<th>K+ (meq/L)</th>
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<tbody>
<tr>
<td>Case – 1</td>
<td>7.524</td>
<td>55.9</td>
<td>65.9</td>
<td>52.6</td>
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<tr>
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<td>63.4</td>
<td>67.8</td>
<td>53.2</td>
<td>2.5</td>
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Case 2: A 70 years old darkly pigmented man presented with fever, cough and left sided pleuritic chest pain. Clinically he had crackles in the left infrascapular region. Chest x ray showed a homogeneous opacity in the left middle zone. His blood glucose was 212 mg/dl without prior history of diabetes. ABG showed compensated metabolic alkalosis with hypokalemia. On suspicion of malignancy CT scan chest was done which showed mildly enhancing hypodense lesion at left hilar region (likely lymph nodes) partially compressing left main bronchus; bronchiectasis left hilar, lingular and lower lobe of left lung with pleural effusion and pleural based fibronodular lesion in apicoposterior segment of the left upper lobe. Post dexamethasone morning cortisol was 13.9µg/dl and serum ACTH was 229µg/ml. CT guided FNAC confirmed diagnosis of small cell carcinoma of lung. In view of clinical condition he was discharged to home for terminal care on request.

In patients with COPD, deterioration often occurs with concomitant infection, however in above cases weight loss, recent onset hyperglycemia, and finding of metabolic alkalosis with hypokalemia indicating ectopic ACTH syndrome (EAS) led to suspicion and diagnosis of bronchogenic carcinoma. None of the patient had classical signs of Cushing’s syndrome. The ectopic adrenocorticotropic hormone (ACTH) syndrome (EAS) was first described in 1928 by Brown and occurs in around 5–10% of all cases of ACTH-dependent hypercortisolism. About 12% of cases of carcinoma of lung manifest with endocrine syndrome, however immunoreactive ACTH staining is detected in all biopsy specimen. Most of these patients do not have manifestation of Cushing’s syndrome, because tumour secretes big-ACTH which is biologically inactive. Most of patients with ectopic ACTH syndrome associated with malignancy present with weight loss, recent onset hyperglycemia, metabolic alkalosis and hypokalemia. A case with similar characteristics as our case has been reported in the literature.

MK Garg*, R Pakhetra**, MK Dutta’, Abhay Gundurthy*

Senior Adviser and HOD, *classified specialist, **DNB Resident, Dept of Endocrinology, Army Hospital (R & R), Delhi Cantt - 110010

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