"Perioperative management of a case of accelerated idioventricular rhythm posted for caesarean section "

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Abstract: Premature ventricular contractions (PVCs) do not necessarily trigger ventricular tachycardias or ventricular fibrillations unless are associated with structural heart disease. Accelerated idioventricular rhythm (AIVR) may merely indicate other treatable conditions such as hypokalemia, hypomagnesemia, hypoxia, hypothyroidism, or overdose of medications such as digoxin, aminophylline, ephedrine, alcohol, caffeine. It must be emphasized that if the work up for underlying heart disease is unrevealing, the patients can be assured that their prognosis is excellent. The anaesthesiologist managing a case of AIVR posted for LSCS may avoid the use of medications (antiarrhythmics, diuretics, nitrates, anticoagulants) hazardous to the foetus; until the symptoms of heart failure appear. Here, we report a case of subclinical hypothyroidism which presented as AIVR. High index of suspicion for hypothyroidism lead us to accept the challenge of avoiding use of medications and vigorous therapeutic intervention; which resulted into satisfactory fetomaternal outcome.

Keywords: Accelerated idioventricular rhythm, Subclinical hypothyroidism

Introduction: In the last trimester of pregnancy symptoms like breathlessness, dizziness, palpitations are frequently found with stable haemodynamics. The anaesthesiologist managing a case of accelerated idioventricular rhythm (AIVR) is often in dilemma whether to use medications (antiarrhythmics, diuretics, nitrates, anticoagulants) hazardous to the foetus and safeguard the mother from ventricular tachycardias and ventricular fibrillations or ignore such symptoms. Though, AIVR is often a clue to certain underlying conditions like myocardial ischemia-reperfusion, peripartum cardiomyopathy (PPCM) or structural heart disease; [1,2] sinus bradycardia resulting from any cause like hypokalemia, hypomagnesemia, hypoxia, hypothyroidism, or overdose of medications such as digoxin, aminophylline, phenylephrine [3] ephedrine, alcohol, caffeine may also facilitate the appearance of AIVR. [1]

AIVR was first described by Thomas Lewis in 1910 [1, 2] and is currently defined as an enhanced ectopic ventricular rhythm with at least 3 consecutive ventricular beats, which is faster than normal intrinsic ventricular escape rhythm (≤40 bpm), but slower than ventricular tachycardia (at least 100-120 bpm). [1, 2] Patients with AIVR should be treated mainly for its underlying causes, such as digoxin toxicity, myocardial ischemia, and structure heart diseases. AIVR is usually hemodynamically tolerated and self-limited; thus, it rarely requires treatment with anti-arrhythmia medications. Atropine can be used to increase the underlying sinus rate to inhibit AIVR. [1] Hypothyroidism may present as

AIVR and diagnosis of subclinical hypothyroidism (nutritional/hormonal/autoimmune) needs high index of suspition.

Case history:

A 21 year old primigravida in labour was admitted to our hospital with 38 weeks of gestation for emergency LSCS. Patient presented with complaints of increased fatigability with difficulty in breathing since one week.

ECG Findings of referring physician were multiple PVCs more than 10 per min, often in bigeminy with generalised ST-T changes persistent in spite of inj. Xylocard 50 mg IV bolus followed by 100 mg/hour in a drip.She never gave any history of abortions, chest pain or wheezing and was not a known case of diabetes, hypertension, IHD or bronchial asthma.

Clinical findings: She was an edematous, dull patient, complaining of (grade II) breathlessness and palpitations.

On examination, her pulse rate was 76 per minute with 8-10 ectopics and BP 130/80 mm of Hg with pitting type of pedal edema. Respiratory system showed bilaterally equal, normal vesicular breath sounds with no ronchi or crepitations.

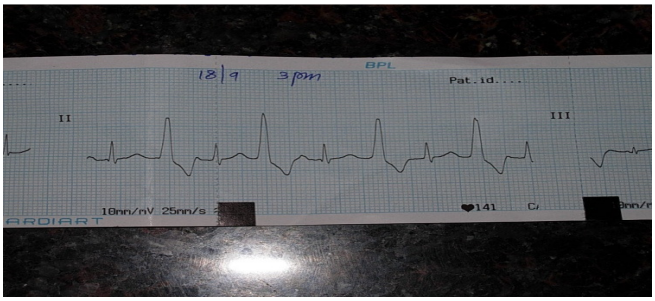
Her Haemoglobin was 10.5gm %. HIV, Serum electrolytes, random blood sugar, urea, creatinine, VDRL, HbsAg reports were normal. Her Blood Group was O Rh negative. Her ECG showed sinus bradycardia, premature ventricular contrations (8-10 per min) with ST depression in chest leads. Chest X-ray showed mild cardiomegaly with basal haziness on the right side.

Her ECHO was normal with LVEF 57%. Her thyroid function report was awaited. She was taking T. Furosemide 20 mg twice daily since last 3 days as prescribed by the referring Physician.

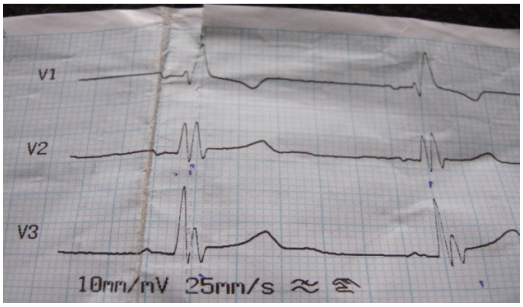
Anaesthesia technique: After reassuring the patient, injection Glycopyrrolate 0.2 mg was given intravenously as a premedication. Lumbar spinal anesthesia was administered between L4 – L5 interspace with injection Lignocaine 5% 1ml. Adequate analgesia was obtained till T8 level.

The patient was haemodynamically stable throughout the operation. Heart rate was 70 beats per min with 4-6 PVCs detected on ECG monitor. 800 ml of Ringer Lactate was administered. A 2.4 kg male baby with APGAR score of 7 and 9 at 1 & 5 minutes was extracted. 20 units of Oxytocin was given in infusion and injection Fentanyl 50 microgram was injected IV. Inj. Amiodarone 300 mg /500 ml RL was kept ready but was not used.*Postoperative course:* Patient was shifted to ICU for observation and further management with instructions to keep the patient in supine position and give Oxygen through ventimask. Four hours later, in the ICU patient developed ventricular bigeminy, with new development of M pattern in right sided leads (RBBB). Her Blood Pressure was 90/70 mm Hg and fresh investigations revealed no hypoxia, hypoglycemia, hypokalemia or hypomagnesemia.

[Figure 1- Ventricular bigeminy]

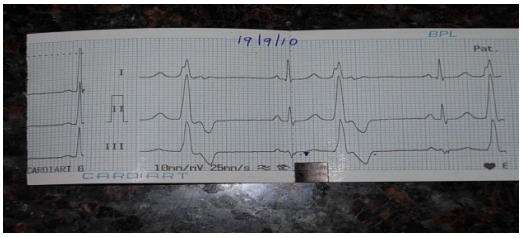


[Figure 2 –RBBB (M pattern in right sided leads)]



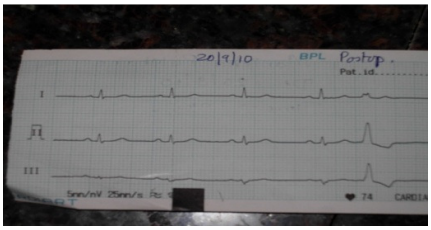
Amiodarone 150 mg bolus in 10 ml of normal saline over 10 min followed by (then 1 mg per minute for 6 hours) 300 mg in IV drip with a rate of 4 drops /min was administered. Though ECG findings of incomplete RBBB with Bigeminy (PVCs) persisted throughout the first postoperative day, we did not increase the dose of Amiodarone since there was symptomatic relief with correction of hypotension .

[Figure 3 - Incomplete RBBB with Bigeminy (PVCs)]



On second postoperative day, frequency of PVCs reduced to 2-3/min. The patient was put on Tab. Amiodarone 100 mg TDS; tapered to 100 mg OD over one month. The patient maintained stable hemodynamics and normal blood sugar level.

[Figure 4 – occasional PVCs on second postoperative day]

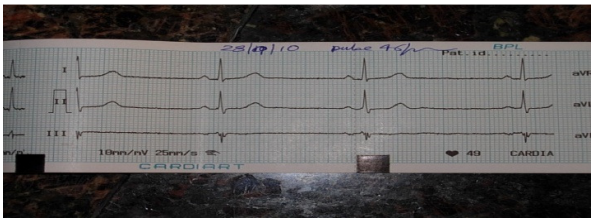


Thyroid function test report obtained on fifth postoperative day was characteristic of subclinical hypothyroidism (an elevated TSH level with a T_4 level at the lower limit).

T3- 67ng/dl (N range 60-200), T4- 4.6 mcg/dl (N range 4.5-12),

TSH - 9.16 mcIU/ml (N range 0.3-5.5).

[Figure 5- At discharge on 10th postoperative day - bradycardia without ectopics]



At discharge, the patient was adviced high Iodine and sugar content in the diet,

T. Alupent 10 mg thrice daily for one week, T. Levothyroxine 50 mcg once daily for 3 weeks.

Thyroid function test was repeated after three weeks which turned out to be normal.

Discussion:

Ventricular extrasystoles are a common finding in patients with and without heart disease. [4, 5, 6] PVCs have been described in 1% of clinically normal people as detected by standard ECG and 40–75% of apparently healthy persons as detected by 24–48 hour ambulatory (Holter) ECG recording. [5] Increasing "severity" of ventricular ectopic activity was directly related to the risk of malignant ventricular arrhythmias. [5] The first recorded description of PVCs was from the early Chinese physician Pien Ts'Io, around 600 BC. [5] In the presence of heart disease, frequent and repetitive extrasystoles are an independent predictive factor of total mortality and sudden death. On the other hand, very frequent monomorphic ventricular complexes and even bursts of ventricular tachycardia in subjects without evidence of heart disease are generally considered benign. [1, 2, 4, 5, 7]

AIVR can be labelled as benign once diagnosis of certain underlying conditions like myocardial ischemia-reperfusion, digoxin toxicity, cardiomyopathies is ruled out.

Peripartum cardiomyopathy (PPCM) is a form of dilated cardiomyopathy with left ventricular systolic dysfunction and is associated with heart failure and the reduction in LVEF below 45%.[8] Symptoms

of worsening of cardiac failure occur in the last trimester of pregnancy and diagnosis is usually made by exclusion of any other cause of heart failure. [8]

The mechanism of AIVR appears to be related to the enhanced automaticity in His-Purkinje fibers and/or myocardium, sometimes accompanied with vagal excess and decreased sympathetic activity.[1] Ischemia, reperfusion, hypoxia, drugs, [3] and electrolyte abnormalities can all accelerate the phase 4 action potential depolarization rates in His-Purkinje fiber and myocardium, leading to faster spontaneous cell depolarization (enhanced automaticity).[1] When the enhanced automaticity in His-Purkinje fiber or myocardium surpasses that of sinus node, AIVR manifests as the dominant rhythm of the heart. Sinus bradycardia may facilitate the appearance of unifocal PVCs. [1]

Subclinical hypothyroidism (nutritional, hormonal or due to antibodies) in early pregnancy, compared with normal thyroid function, is associated with the occurrence of pre-eclampsia, increased risk of perinatal mortality, unexplained subfertility, recurrent miscarriage and maternal post-partum thyroiditis. Hypothyroidism can lead to well-known and classic cardiovascular abnormalities such as a reduced cardiac output, arterial hypertension, sinus bradycardia, prolonged QT interval, and pericardial effusion. [11]

Recent studies have shown that aggressive attempts to maintain sinus rhythm using amiodarone or other drugs do not improve outcomes in relatively asymptomatic patients. [14] Moreover, Amiodarone crosses the placenta and reaches measurable levels in breast milk hence we kept the dosage of amiodarone at the lowest effective level. Due to the structural similarity between Amiodarone and thyroid hormone, it is possible that the drug could inhibit the activity of the 5'-thyroxine-deiodinase. [14, 15] The drug's high iodine content is a factor in its effects on the thyroid gland. In hypothyroid patients with a strong clinical indication for amiodarone, the drug may be continued with appropriate thyroid hormone supplementation. Bradycardia and heart block occur in 1 to 3 percent of patients receiving amiodarone. [14]

Conclusion : We believe this case report is of interest because it is unusual and because it serves as a reminder that there is a form of PVCs known as AIVR that can be caused by hypothyroidism. A multidisciplinary team approach, rational use of drugs unsafe in pregnancy & lactation is needed to get satisfactory fetomaternal outcome.

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