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Peri-operative silent ischemic changes on ECG in a primi gravida posted for LSCS under SAB

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Abstract:*A large number of healthy females posted for LSCS under SAB exhibit transient electrocardiographic (ECG) changes consistent with myocardial ischemia. Interestingly a large subgroup of these patientpresentswith symptoms typical of myocardial ischemia. Various mechanisms have been proposed in literature for this phenomenon including but not restricted toalready increased myocardial oxygen demand during pregnancy and when complemented with acute hypervolemia due to auto-transfusionfrom uterus and co loading prior to spinal anaesthesia resultsin an increased end-diastolic volume and pressure thus increasing myocardial oxygen demand.The disruption in myocardial oxygen demand and supply inducesischemia with subsequent chest symptoms and ECG changes. The present case report is of dynamic ECG changes in the absence of any symptoms noted peri-operatively in a young healthy female posted for LSCS under Sub Arachnoid block fight*

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I. Introduction

Sub arachnoid blocks are shown to be an easy, safe, and most commonly followed and accepted anaesthetics technique worldwide for LSCS [1]. Nonetheless its association with potential lethal side effects including but not restricted to decreased cardiac output leading to end organ ischemic changes cannot be overlooked [2]**.**While, various clinical trials advocate the benefits of co loading for maintaining cardiac outputand preserving end organ perfusion post sub arachnoidblock;others have proposed the associated increase in end diastolic volume and pressuremore so when complemented with auto transfusion from uterus resulting in increased myocardial oxygen demand leading to dynamic ischemic changes peri-operatively[3,4]. In the of absence of pre-existing coronary artery disease, a disruption between myocardial oxygen demand and supply has been attributed as a single most important reason for peri-operative myocardial ischaemia[5].

The present case report is of dynamic ischemic changes observed in the peri operative period in a 31-year-old primi-gravida patient accepted in ASA I for elective caesarean section under sub arachnoid block which reverted post 24 hours of surgery.

II. Case

The present case report is of a 31-year-old adult female patient with no known comorbidities except hypothyroidism for which she was on 25 mcg Thyroxin once daily for last 03 months. Patient had no history of cardiovascular disease, hypertension or any respiratory illness. She is a non-smoker, non-drinker, vegetarian and there is no use of any illicit drug usage in the past. The family history of patient is non-significant for any coronary artery disease. Patient was taking daily walks for 2-3 km at normal pace in the last trimester of present pregnancy. There is no history of orthopnea/paroxysmal nocturnal dyspnea or any decrease in physical capacity in the recent past. This was the first surgery that the patient was undergoing and there was no history of any anaesthesia exposure in the past.

Since there was no indication of fetal distresspatientwasplannedforanelectiveLSCSunderSubArachnoidBlock. After attaching standard ASA monitors and securing i.v. access patient was co-loaded with 500 ml of Ringers Lactate. Baseline blood pressure and heart rate recordings were noted and 2.2 ml of 0.5 % Bupivacaine (heavy) was administered at the L3-L4 level in the sitting position using 26-gauge spinal needle. Post administration of the drug patients was placed in supine position with 15^0table tilt. Sensory block was assessed to the loss of temperature sensation every 2 minutes for a

period of 10 minutes. After the confirmation of the successful sub-arachnoid block up to dermatomal level of T4 the patient was cleaned draped and the surgery commenced.

Within initial 05 minutes of the commencement of surgery ST depression was noted in lead 2 in the Space Lab Monitor (Fig. 1). However, since there was no associated complains of chest pain, vomiting, diaphoresis, altered sensorium, breathlessness, giddiness or signs of hemodynamic instability the surgery was continued and the patient was closely monitored for any ominous signs of cardio vascular collapse or otherwise. Intraoperative readings (Table 1) were recorded.

TIME	SYSTOLIC BP	DIASTOLIC BP	MAP	HEART RATE
1505	113	69	82	109
1509	124	77	93	96
1512	125	78	95	93
1515	128	78	88	95
1518	133	82	100	92
1520	122	76	90	86
1530	126	80	94	90
1540	132	74	92	92

Table 1. Intra operative readings of NIBP and heart rate

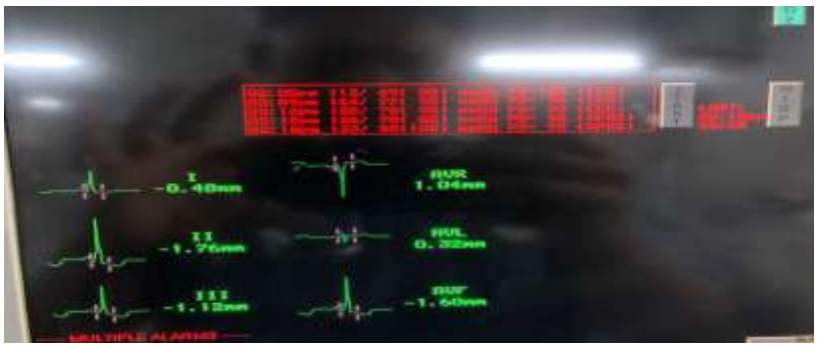
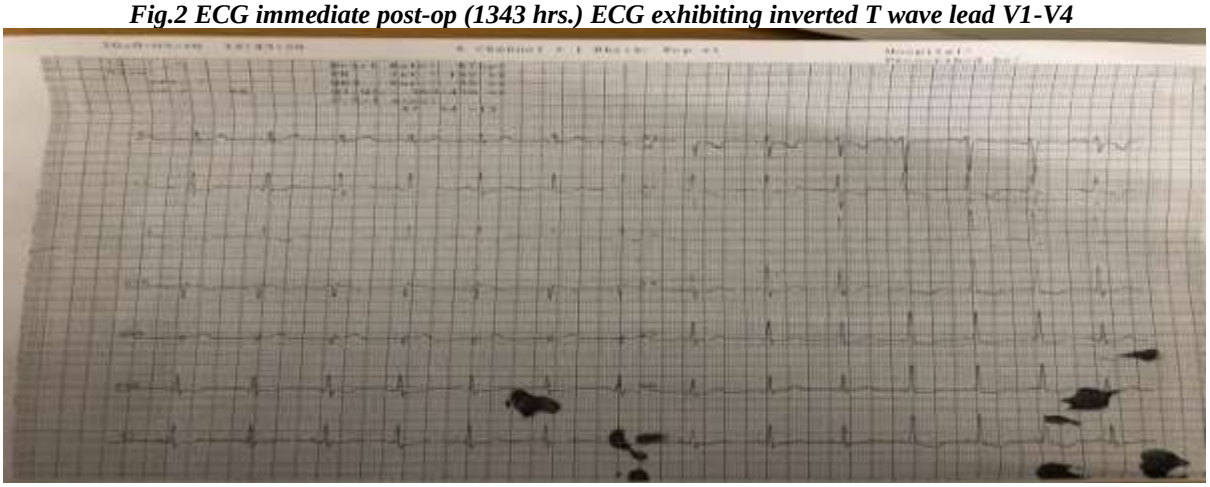


Fig 1. Intra Op ECG changes and NIBP

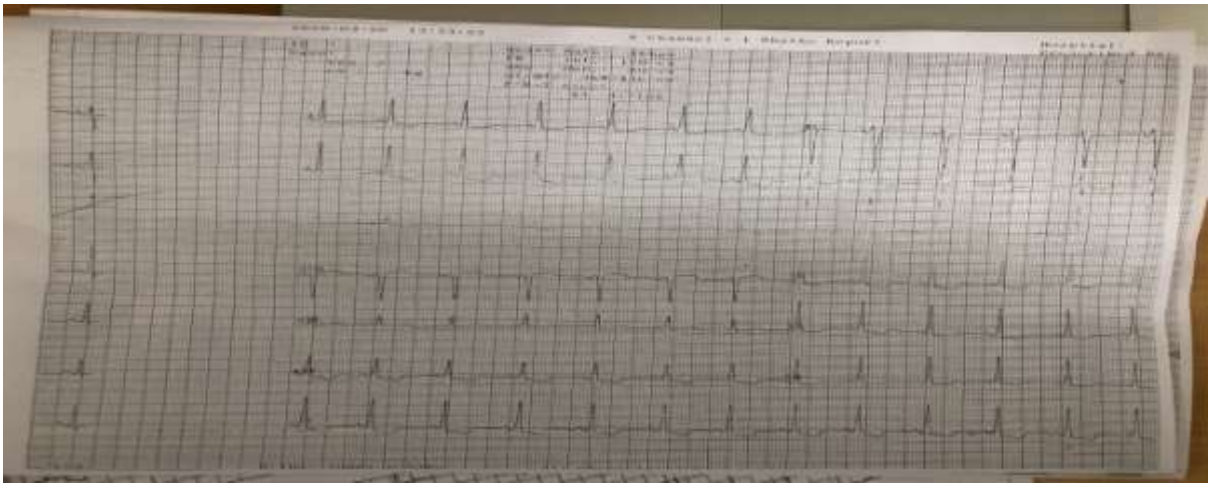
Aftertheskinclosurepatient was shiftedtopost-oproom and a12 leadECGwas recorded which exhibitedTwaveinversioninleads V1-V4(Fig.2). The patient vitals in the immediate post op period were stable and no ominous signs were noted.

Fig.2 ECG immediate post-op (1343 hrs.) ECG exhibiting inverted T wave lead V1-V4



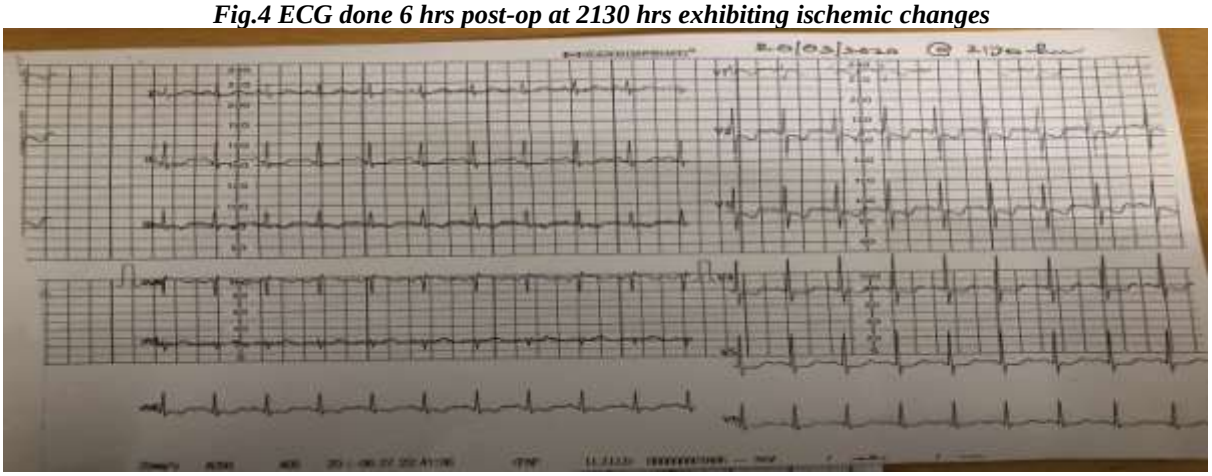
Serial ECG recordings of the patient done at an interval of 10 minutes(Fig.3) exhibited dynamic ischemic changes in the form of invertedT wave in Inferior as well as the precordial leads. Patient at this stage too had no clinical signs of an ischemic event, she was comfortable maintaining heart rate of 86 bpm and NIBP of 126/74 mm Hg, and saturation of >95 % at roomair.

Fig.3 ECG 10 mins post-op (1353 hrs) exhibiting inverted T wave inversion in inferior and all precordial leads



A repeat ECG of the patient was done post 6 hours of the surgery at 2130 hrs. (Fig.4)which continued to show ischemic changes with inverted T wave in inferior leads and V1-V4though patient still had no ominous clinical features. A point of care testing including a bed side 2 D echocardiography and CKMB level was carried out. 2D echo of the patient was unremarkable however CKMB levels were 51 U/L (Normal range 0-25). Consequently, the patient was started on therapeutic dose of Inj.LMWHalongwithloading dose 300 mgAspirin after confirming no signs of active bleed and a normal coagulation profile.

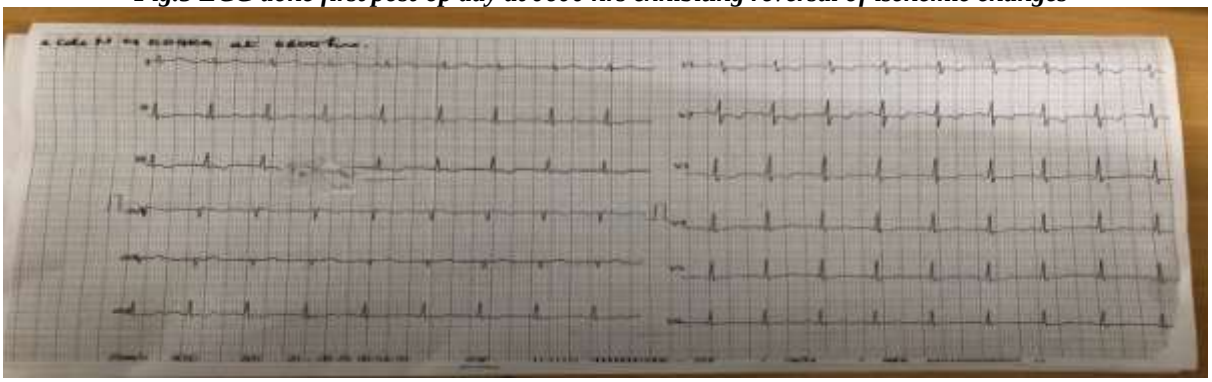
Fig.4 ECG done 6 hrs post-op at 2130 hrs exhibiting ischemic changes



A repeat ECG done in the morning of first post op day at 0600 hrs (Fig. 5) exhibited the reversal of dynamic ischemic changes.Patient continued to be in the ICU for observation for next 24 hrs.

In view of the reversal of ECG changes and in the absence of any clinical features she was subsequently discharged to ward after 24 hours of uneventful stay in the ICU. The entire duration of hospital stay of the patient remained unremarkable and post 5 days of surgery she was discharged to home.

Fig.5 ECG done first post-op day at 0600 hrs exhibiting reversal of ischemic changes



III. Discussion

In the plethora of complications that can occur peri-operatively myocardial ischemia is most dreaded. If not identified and corrected early it can lead to life threatening complications [6]. Detailed pre-anaesthetic history and cardio-pulmonary assessment is pivotal in prevention, early diagnosis and effective treatment of any cardiac event peri-operatively. While conducting caesarean section unless contra-indicated central-neuraxial blockade is regarded safe, rapid and effective mode of anaesthesia [1,7]. Its association with over all lower complication rates, than general anaesthesia has already been established in literature. However, amongst all regional anaesthesia techniques association of cardiovascular complications is most commonly seen with sub arachnoid blocks [8].

Although various clinical publications have indicated that since coronary artery disease is particularly rare in parturient transient ECG changes although frequently seen in this subset of population bears no clinical relevance [9].

Kulka et al. [10] reviewed 136 cases of myocardial infarction reported during pregnancy and reported that half of the patients had normal coronary vessels. This shows that peri-partum myocardial ischemia/infarction can occur even without evidence of coronary artery disease. [11]. In small percentage of patients during continuous Holter monitoring per- and post-operatively Frequent episodes of ST segment depression have been reported.[12] Several case reports of myocardial ischemia/infarction and cardiac death in pregnant women show that cardiac complication is a risk that must be considered in some parturient.[13] Various mechanisms including but not restricted to coronary artery spasm, pregnancy associated cardiovascular changes and hypercoagulability, and surges in myocardial oxygen demand have been proposed in literature.[14] Palmar et al. speculated that increased demand of oxygen by myocardium in pregnant patients along with acute hypervolemia due to pre loading plus auto-transfusion from uterus results in increased end-diastolic volume (pressure) [15] with consequent excessive oxygen demand of myocardium. In addition to this post spinal sympathetic blockade causes reduction in diastolic pressure and decreased coronary perfusion which impairs myocardial oxygen supply. This disruption in myocardial oxygen demand and supply is sufficient to induce cardiac ischemia with subsequent chest symptoms and ECG changes [15]. In healthy population with intact sympathetic nervous system reflex veno-dilatation prevents a sudden increase in volume status does not lead to increased myocardial oxygen. However, in healthy parturient raised anxiety states (neuro-circulatory asthenia), vago-regulatory asthenia (hyper dynamic central and peripheral circulation), hyper dynamic heart syndrome, mitral valve prolapsed or autonomic system imbalance is an important aetiology of ST segment. [16] The present case reports we could not follow up the patient with coronary angiography to assess a pre-existing coronary artery disease that might have accentuated under the stress of surgery.

IV. Conclusion

Increased myocardial oxygen demand during pregnancy when complemented with acute hypervolemia due to auto-transfusion from uterus and co loading prior to spinal anaesthesia results in an increase in end-diastolic volume and pressure. This phenomenon consequently accentuates the pre-existing high myocardial oxygen demand during pregnancy. Concurrent to this sub arachnoid block induced sympathetic blockade causes reduction in diastolic pressure and decreased coronary perfusion thereby further decreasing myocardial oxygen supply. The gambit of these events which cause disruption of myocardial oxygen demand and supply induces ischemia.

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