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RESEARCH ARTICLE

HYPOXIC HEPATITIS AFTER IN-HOSPITAL CARDIAC ARREST - A CASE REPORT

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Abstract

Hypoxic hepatitis (HH) is a condition of insufficient hepatic blood flow, due to shock or hypotension. It is characterized by an elevation of aminotransferases over 20times the upper limit of normal during first 72hrs after return of spontaneous circulation (ROSC). Most of the cases reported are out-of hospital cardiac arrest(OHCA) with poor neurological outcomes. We are reporting a case of HH following ROSC, in hospital witnessed cardiac arrest (IHCA) with full neurological recovery. A 61year old female was scheduled for excision of meningioma between D1-D3 vertebrae under general anaesthesia. Induction and maintenance of anaesthesia was done as per routine protocol. Throughout the surgery, haemodynamics were maintained stable. But while transfusing 2nd unit of blood, patient developed sudden episode of hypotension(Blood Pressure-70/36mmHg) and bradycardia followed by asystole. Cardio-pulmonary resuscitation(CPR) was started in prone position immediately. On removing the drapes, red wheal rashes were observed all over the back. The ongoing blood transfusion was stopped. The patient was made supine and CPR was continued as per ACLS protocol for 45minutes after which ROSC was achieved. Patient was then shifted to Intensive Care Unit(ICU) for post-resuscitation care. During the recovery phase in ICU, significant increase in transaminases(more than 20folds) were observed in first 24hours, which got normalized over 5days along with full neurological recovery of the patient. HH is not a complication which we as an anaesthesiologists usually encounter but its not a rare one. Our case highlights that HH is potentially reversible and may not always be associated with poor neurological outcome.

Introduction:-

Hypoxic hepatitis (HH) is an acute liver injury resulting from liver hypoxia. Hypoxic Hepatitis is also known as ischemic hepatitis or shock liver. This disorder is noted in only 2-2.5% of patients admitted in ICU but it's not a rare complication after ROSC post CPR outside the hospital which is 11% [1]. It is rarely seen in inhospital cardiac arrest (IHCA) patients post CPR. HH is defined as an elevation of aminotransferase over 20 times the upper limit of normal during first 72 hrs. after ROSC. The most common predisposing factor is cardiac failure followed by circulatory failure[2]. HH has multifactorial pathophysiology which often involves hepatic congestion from right heart failure with reduced hepatic blood flow, total body hypoxemia, reduced oxygen uptake by hepatocytes or reperfusion injury due to ischemia. Diagnosis of HH relies on three criteria: clinical setting resulting in reduced oxygen delivery or utilization by the liver, a significant and often transient rise in serum aminotransferases level, and

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exclusion of other potential cause of liver injury like drug and viral hepatitis. A clinical states that predisposes Hypoxic Hepatitis include, cardiac failure, toxic shock states and respiratory failure[2].

Case-

A 61yr old female weighing 45kgs, scheduled for D1-D3 laminectomy with excision of meningioma as an elective case. She presented with the history of weakness and numbness in bilateral lower limbs since 1 year. No other history of co-omorbities or any surgery in past was present.

On examination patient was conscious, oriented with stable vitals, and was afebrile. Respiratory and cardiovascular system was within normal limit. Power in bilateral upper limb-5/5, in lower limbs-4/5. Patient had normal airway. Blood Investigations, electrocardiography (ECG) and chest x-ray were normal. MRI whole spine showed intradural extramedullary lesion (meningioma) in spinal cord at the level of D2 vertebral body. After all routine checkup, patient was considered fit for surgery from anaesthesia side and was advised Nil Per Oral for 8hrs and Tab. Pantoprazole 40mg in the morning of surgery.

In the operation theatre, all routine monitors were attached (ECG- sinus rhythm of 72beats/min, NIBP-130/78mmHg, SPO2-100%). Two IV cannulas were secured. Preoxygenation done with 100% oxygen and patient was induced with Inj. Fentanyl 100mcg IV and Inj. Thiopentone 250mg IV and muscle paralysis was achieved with Inj. Vecuronium 5mg IV. Endotracheal intubation was done with 7.0mm Internal diameter endotracheal tube. As patient was to be positioned prone, all parameters were taken care off, like- throat packing was done, bite block inserted and eyes were padded. Patient than made prone and all pressure points were padded with cotton. Intraoperatively, IV antibiotic Inj. Monocef 2gm IV, Inj Paracetamol 1gm IV, Inj Methylprednisolone 1gm IV given. Surgery lasted for one and a half hour. 2litre crystalloid, 500ml colloid and 1 unit PCV was given. Blood loss was approximately 900 ml. During the closure of surgical site, 2nd unit of blood started, and after 10min of which patient developed sudden episode of hypotension (BP-70/36mmHg). IV fluids were rushed and Inj. Mephenteramine 3mg IV stat given. No response was seen and was followed by bradycardia HR-52/min. Inj. Atropine 0.6mg IV stat given. Again there was no response to the drugs. HR- 30/min, BP-58/22mmhg. Inj Atropine 0.6mg IV stat again repeated, but patient went into asystole. Immediately CPR in prone position started, Inj. Adrenaline 1mg IV stat given. Simultaneously, the drapes were removed and red wheals noted all over the body. Hematuria was also noted, the ongoing blood transfusion was immediately stopped. After giving 2 cycles of CPR in prone position, patient was made supine without wasting much time and CPR was continued as per ACLS protocol. Inj. Hydrocortisone 100mg IV and Inj. Dexamethasone 8mg IV was given. Inj. Adrenaline 1mg IV boluses were repeated thrice. After 2 cycles of CPR in supine position, ECG showed Ventricular tachycardia and pulse was also absent, DC Shock with 200J was given and continued the CPR. After giving DC shock ECG showed Ventricular tachycardia with pulse 130/min, carotid became palpable, BP was recorded as 152/96mmHg. Loading dose of Inj. Amiodarone 150mg IV was given which was followed by infusion of 1mg/minute for 6 hours. Return of spontaneous circulation achieved, HR-120/min, BP- 80/50mmhg, Inj. Noradrenaline at 5mcg/min and Inj. Dopamine 10mcg/kg/min IV infusion was started via peripheral line and titrated according to BP. Serial ABG's were done and Inj. Sodabicarbonate 150meq IV was given, Inj. Calcium 10ml of 10% over 10min. IV was also given. Under all aseptic precautions, Right femoral Central venous pressure line was secured simultaneously and ionotropes were then connected to it. Over the duration of 1hr, patient monitored on operation table and meanwhile ionotropes were tapered off. Vitals were HR-98/min, BP- 138/77mmHg, Spo2- 100%, pupils- bilateral normal size and reactive. Patient was then shifted to Neuro ICU on Bain's circuit with oxygen cylinder for mechanical ventilation and advised for cross matching of blood product again, 12 lead ECG, Chest X-ray, all routine blood investigations, ABG, Cardiology opinion, monitoring of GCS and vitals.

During the stay in ICU, IV Antibiotics and all supportive treatment was started as per ICU protocol. Chest Xray shows homogenous opacities in upper and mid zone of right side of chest. Respiratory opinion was taken and nebulization and bronchodilators were started as advised. ECG was done and it was normal. Post resuscitation care was given. Symptomatic treatment and antibiotics were given. All the blood investigations in the sequence shown in **table 1** were ordered. There was significant increase in liver function tests (LFT) in first 24-36hrs which later started decreasing from Post op day-2 (POD-2). Similarly kidney function tests (KFT) were also high in Post op day 0 &1 which later starts normalizing. Patient's GCS improved and she became E4VtM6 on post op day-1(POD-1) only. Patient was maintaining vitals on noradr infusion on POD 1, which then tapered off on Post op day 2. Patient was extubated on Post op day 4. Patient was shifted to ward on Post op day 5, and discharged on the next day. Patient was fully recovered with no neurological deficit.

Immunoglobulin Profile-
We also advised to get Immunoglobulins as the marker of blood reaction- IgG- 1060mg/dl (700-1600), IgA – 320mg/dl (70-400), IgM – 47mg/dl (40-230), IgE – 1143 IU/ml (<150). IgE was raised but it is not specific test to blood transfusion reaction.

ECG- Figure-1 and Figure-2, showing changes in ECG postoperatively and after recovery in icu, respectively.

Chest X-RAY- Figure-3, showing improvement in chest x-ray during recovery phase in icu from day 1 of postoperative to day 4 of postoperative.

Figure Caption-
ECG-

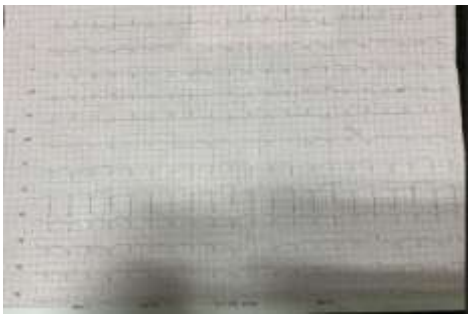

Figure 1:- ECG taken post-surgery.

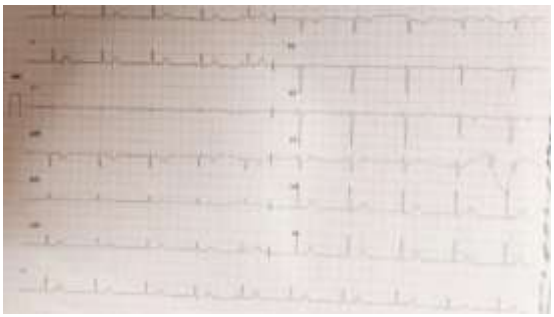

Figure 2:- ECG in ICU post recovery.

Chest x-ray-

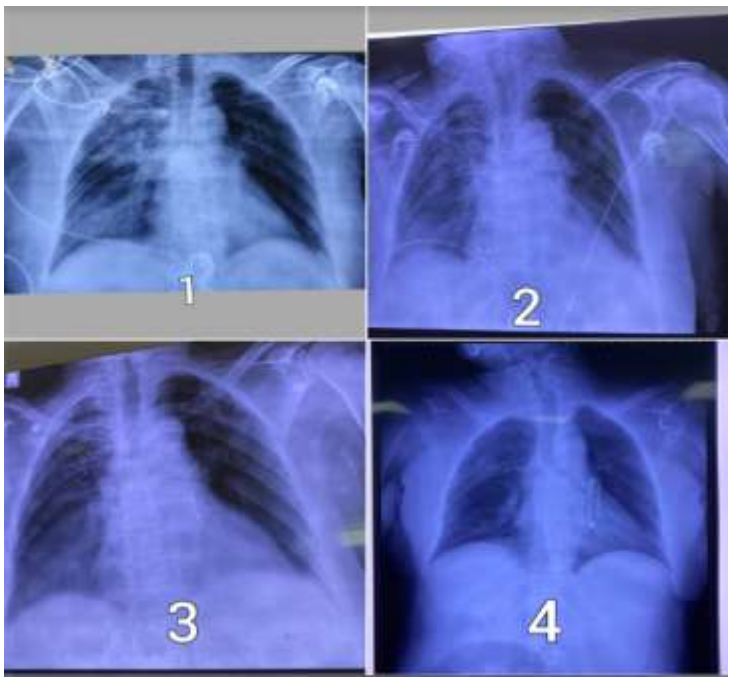

Figure-3, CXR showing improvement on subsequent days post-operatively (POD 1,2,3,4 respectively).

Table-

Table 1:- Showing all blood investigations in sequence from the day of surgery to the post-operative day 5.

Parameters	POD0	POD1	POD2	POD3	POD4	POD5
Hb(gm/dl)	11.5	11.5	9.1	10.9	12.5	11.1
WBC(10*3)	27	62	42.2	22.1	14.8	11.3
Platelets(lakh)	1.4	1.2	90	1.6	1,75	1.95
Na/K(mmol/l)	149/3.1	152/3.5	153/3.8	150/2.6	143/2.8	139/3
Urea/Creat(mg/dl)	38/0.6	74/1.1	74/1.4	76/1.3	68/1.2	40/0.5
Bilirubin(mg/dl)	0.8	0.6	1	1.2	1.1	1.6
AST/ALT/ALP(u/l)	2732/2095/107	1893/1729/88	537/931/86	171/690/93	55/523/170	71/90/83
PT/INR	12.3/1.06	19.3/1.41				
ABG-pH	7.197	7.32	7.429	7.446	7.45	
pCO2	39.3	38	39.9	41.9	40.4	
pO2	101	61	83.4	91.6	98.6	
HCO3	14.7	19.3	26	28.4	24.2	
Lactate	9.9	6.1	1.6	1.3	1.4	
SO2	96.8	99	97.2	98.4	97.6	
input/output	1450/530	4100/2410	3600/5250	2250/3400		

Discussion:-

Hypoxic Hepatitis is a condition which is rarely seen in a postoperative period. HH is an elevation of aminotransferase over 20 times the upper limit of normal during first 72hrs after Return of spontaneous circulation.[2] There is a similar condition called as Acute Liver Failure which can be confused with hypoxic hepatitis. HH and acute liver failure (ALF) are not rare complication after cardiac arrest but ALF is more common than HH and is mainly triggered by the duration of resuscitation and is associated with increased mortality[3]. The definitive diagnosis of HH is histopathological examination, however, it can be diagnosed based on marked elevation of liver enzymes level and underlying cardiopulmonary disease. Hence, HH is diagnosed largely clinically using the three criteria's which we discussed earlier. In our case, there was no history of exposure to hepatotoxic drugs or any viral hepatitis, neither any hepatotoxic medication were administered during general anaesthesia. Therefore, our patient met with three criteria of HH. In our case, drastic fall in BP, may have reduced hepatic blood flow, resulting in inadequate oxygenation of liver. Also the systemic hypotension causes hepatic artery constriction. A systolic blood pressure of less than 90mmHg for 15 minutes may lead to development of HH. HH usually resolves within 7-10days. Treatment of Hypoxic Hepatitis is a paradigm of post ROSC care. The definitive treatment of HH involves management including stable hemodynamics, appropriate blood oxygenation and monitoring for potential complications like hypoglycemia, hyperammonemia, and hepatopulmonary syndrome. Treatment should be directed towards the predisposing conditions like optimization of body circulation, maintenance of mean arterial pressure, and preservation of liver microcirculation and oxygenation[2]. Early recognition of disease and its underlying pathology is crucial to its management. While Acute liver failure(ALF) is considered when serum total bilirubin is >1.2mg/dl in the absence of chronic liver disease and an INR >1.5. Among Acute liver failure and Hypoxic Hepatitis, Hypoxic Hepatitis is significantly associated with poor neurological outcome.[4] uninterrupted and effective CPR. Our case highlights that it can happen in at risk patients but is potentially reversible and may not always be associated with poor neurological outcome.

Conclusion:-

Hypoxic Hepatitis is a rare entity occuring in IHCA and is usually with poor neurological outcome. HH usually occurs as a consequence of cardiac, circulatory or respiratory failure, leading to in-hospital stay and ultimately increasing morbidity and mortality. Prompt recognition and early actions is vital, as delay in diagnosis in medical as well as in post surgery care can worsen the outcomes. The only known treatment is to correct predisposing conditions and treat symptomatically. Our patient however fully recovered and was without any neurological deficit. This case report shows how crucial was the attempt of prone CPR as the circulation immediately started and permanent damaged was avoided. The survival of patient following cardiac arrest consistently depends on the duration of hypoxia. Prolong hypoxia results in decrease survival rate. HH is not a complication which we as an anaesthetists usually encounters but its not the rare one and can happen in any patient who gained ROSC postCPR postsurgery. The fundamental concept of HH is that the hypoxic liver injury is caused not only by decreased BP, but also by arterial hypoxemia. Elevated liver function tests, any coagulopathies, blood transfusion reactions, hypoxia

pre/intra/postop, preexisting liver disease and type of surgery should be taken in account before taking patient to Operation theatre. Our case highlights an important clinical finding in anaesthesia field that requires a high index of clinical suspicion to diagnosis. Anaesthesiologist must minimize even transient fluctuations in BP, and we should optimize oxygenation during peri & intraoperative period.

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