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RESEARCH ARTICLE

ACUTE LIVER FAILURE ASSOCIATED WITH COVID-19 IN A 2-YEAR-OLD BOY

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Abstract

Almost 2 years after the first cases of the SARS-CoV-2 pandemic were reported, we are still learning about it till today. We are now more familiar with SARS-CoV-2 and it's role in causing COVID-19 and that the effects of the disease is mostly a respiratory illness (1).The gastro-intestinal/hepatic systems are the next most commonly affected after the respiratory system (2). Symptoms typically include nausea, vomiting, diarrhoea and abnormal liver enzymes (2). Acute liver failure secondary to covid-19 is rare and usually occurs later in the course of the disease (2). Some studies have demonstrated that SARS-CoV or MERS caused liver injuries with elevated liver enzymes and bilirubin levels, although these changes were mild (4). Studies of COVID-19 infection have shown the incidence of liver injury to be between 14.8 to 53%, mainly presentingwith abnormal alanine transaminase/aspartate aminotransferase (ALT/AST)(4).However alkaline phosphatase remains in the normal range in both mild and severe cases. Liver injury is significantly higher in patients with severe COVID-19.The effects of COVID-19 and it sequelae in children is still not widely reported. We describe a unique case of acute liver failure in a 2 year old boy most likely due to COVID-19in Saudi Arabia.

Introduction:-

Case Report

A 2 year old previously healthy boy presented to a primary health care center in Al-Nomaishospitalwith flu-like symptoms. He was pyrexic, with temperaturesranging between 39-40^{o}Crelieved by use of anti-pyretics.Initial diagnosis of pharyngitis was made and the patient was treated with co-amoxiclav antibiotics.Despite the treatment the child continued to spike high temperatures.He also displayed a poor activities.

On next day, the parents noticed that he became less responsive and difficult to arouse with increased lethargy.He was urgently brought to the emergency department, During evaluation He was drowsy with a disturbed level of consciousness, his temperature was38.5^{o}C, heart rate of 130 beats per minute and blood pressure was 80/40 mmHg. On examination the child was pale with mild jaundice, multiple ecchymosis with bleeding from the mouth were seen.Abdominal examination showed non-distended abdomen with hepatomegaly3 cm below the costal margin.Other systemic examination were unremarkable.

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After admission, the child became unresponsive to pain or voice with more deterioration in conscious level with frequent episodesof generalized tonic clonic seizure, lasting more than 5 minutes. A diagnosis of status epilepticus was made and a decision to intubate the patient was taken. However extensive bleeding from the nose and mouth made attempts at intubation extremely difficult. The child easily bled from sites of IV cannulation as well. This made it difficult for the child to be managed at a primary health care center. Hence he was transferred from Al-NomasHospital to AbhaGeneral Hospital for further treatment.

The child had no previous medical history of note. No coagulopathy or tendency to bleed or bruise. No history of metabolic disease or rheumatological disease. There was no history of any types of hepatitis. There was no family history of liver disease or bleeding conditions. Normal development , with all vaccinations received in a timely manner. No previous hospital admissions. He was born at term via spontaneous vaginal delivery to a 24 year old mother gravida 2, para 2. He required no NICU admission and all ante-natal checks were normal.The family confirmed no use of any herbal medication or any other medicationsapart fromparacetamol within safe limits.

Table 1:- Liver enzymes during the period of admission.

urea	**81.54**	**Normal range**
CRE	0.63	0.84-1.25
k	3.50	3.5-5.1
Na	136	136-146
ALP	342	0,00-50.00
TBILC	3.73	0.30-1.20
DBILC	2.32	0.00-0.20
TP	5.2	6.60-8.30
ALB	3.61	3.5-5.20

Investigations

On admission the child tested for covid 19. It was confirmed there was no recent travel history or contact with any covid positive individuals. Admission bloods showed a white cell count of 4.13^10^3/uL, Haemoglobin 11.2(gm/dl), Creatinine 0.63(mg/dl), Urea 8.54(mg/dl), alanine transaminase 2734(U/L), aspartate aminotransferase2947(U/L), alkaline phosphatase 342(U/L), total bilirubin 3.73(mg/dL), Direct bilirubin 2.32(mg/dL), Ammonia 109(units), Acetaminophen level <3(units). During the admission the ASTrose to 4995(U/L) with ALT 5005(U/L), this then gradually improved during admission. Table 1 ,shows series of live function tests during admission.Auto immune liver screen was negative for P.ANCA, Anti-liver-kidney microsome antibody, anti-smooth muscle antibody, ANA, Anti-liver cytosol antibody. Viral screen was also negative (including EBV, Hepatitis A, B, C)

A Chest x-ray done on admission (Figure 1) showed (enter report).Owing to the deranged liver enzyme an ultrasound of the abdomen was carried out which showed ‘a mildyenlarged levier otherwise a normal scan’. A full body computed tomography (CT) was carried out which revealed Bilateral lung consolidation and bilateral pleural effusions, normal abdominal and brain findings(Figure 2)

Table 2:- Liver function at time of admission.

Day of admission	**AST**	**ALT**
1th	2947	2734
2th	4995	5005

3th	1140	
4th	816	
5th	363	472
6th		708
7th	170	2368
8th	133	1247
9th	102	

Figure 1:-

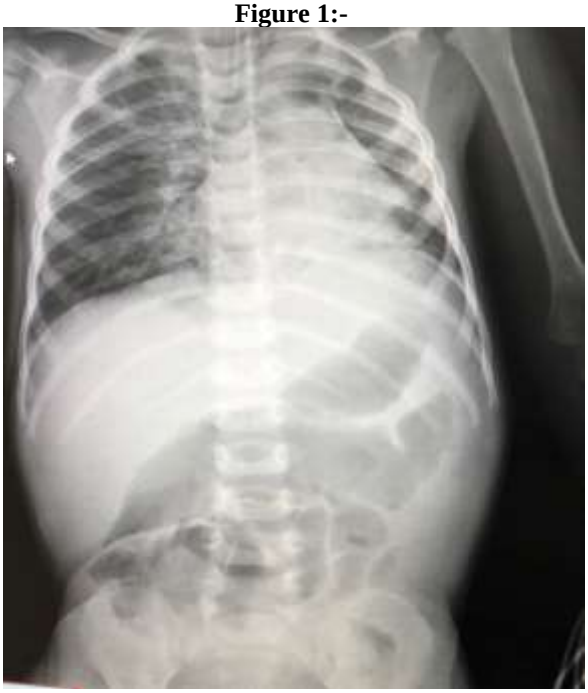
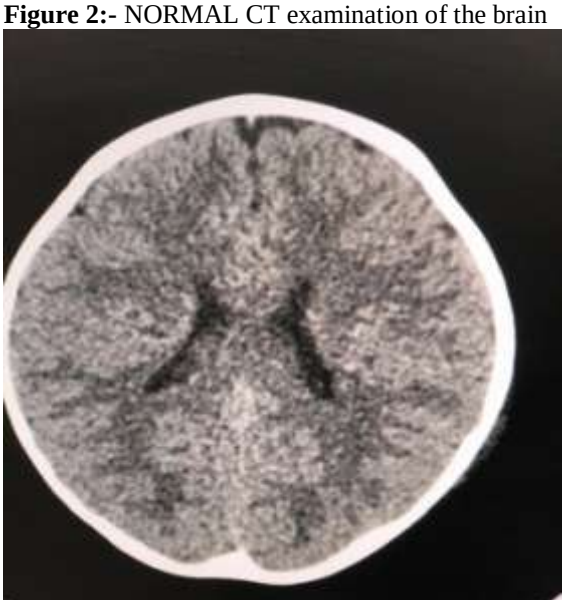


Figure 2:- NORMAL CT examination of the brain



Treatment

The child was treated as per the local Covid 19 treatment protocol (Remdesivir 200 mg IV load , then 100 mg IV q24h for 10 days) Treatment included methylprednisolone, Levetiracetam, vitamins K, Fresh Frozen Plasma, Metronidazole, Vancomycin, Meropenem, and IVIG

Outcome

The child was admitteddirectly to PICU for seven days and intubated for 5 days then shifted to high flow O_2.The child was eventually transferred to a regular pediatric ward once he did not require anymore oxygen therapy, his coagulopathy was corrected, there was no seizure activity and he was tolerating oral intake. The child gradually regained his energy and enthusiasm and stayed in hospital for a total of 12 days .He was discharged in stable condition withclosefollow up.

Discussion:-

This case report presents a unique case of a COVID-19 causing acute liver injury in a 2 year old child. Currently, studies on the exact pathophysiology of liver injury in these patients are limited, but it is believed either to be a direct effect of virus or immune-mediated inflammatory response (5).The respiratory effects of COVID-19 are well documented however cases of acute liver failure in children is very rare (6). This has been reported in the past but in much older children (7). For our case the treatment was merely supportive. The regenerative ability of liver cells may be attributed to the swift recovery in liver enzymes and coagulation. A learning point from this case report is the importance of early diagnosis of liver failure and transfer of patient from a smaller district hospital to a larger teaching hospital with PICU facilities. The potential of late or misdiagnosis is clear to see and may be inevitable.

Conclusion:-

This case report presents a unique case of a COVID-19 causing acute liver injury in a 2 year old child. Coverage and uptake of the covid-19 vaccinations varies throughout the world and is still not offered to children in many countries. Acute liver failure can be a feature of COVID-19 and should always be kept in mind when seeing acutely ill patients. Most importantly, timely diagnosis and having the facilities to provide care is imperative, especially for children.

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