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RESEARCH ARTICLE

MULTIPLE LIVER AND SPLENIC ABSCESSES AS AN EARLY COMPLICATION AFTER COVID-19 INFECTION: CASE REPORT AND REVIEW OF LITERATURE

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Abstract

COVID-19 is a major health problem affects millions of people worldwide. The effect of COVID-19 infection is not only related to the cardiopulmonary insult, but rather affecting every system in the body. Many publications have reported common hepatosplenic insult during COVID-19 infection.

Here we describe a case ofmultiple liver and splenic abscesses post corona virus infection in a healthy 13-years old girl, highlighting the possible explanations of her condition in the context of literature review.

Background: Coronavirus disease 2019 (COVID-19) has been considered overwhelming heath crisis with reported death over 6 million people till now and over half billion confirmed cases according to WHO dashboard [(1).]

This raises a concern that this virus might have the capability of attacking not only the respiratory system but also almost every single organ system. Patients with the virus have presented with features of acute liver injury, gastrointestinal symptoms, endocrine problems, neurological damage, and cardiovascular disease, most of them recover completely with no sequalae, and others develop short- and long-termSide effects[(2,3)].

Recent research on COVID-19 have shown a high incidence of liver injury ranging from 14.8 to 53%, mainly indicated by elevated liver enzymes levels accompanied by slightly elevated bilirubin levels. The proportion of patients who develop liver injury in severe COVID-19 seems to be higher than in mild cases(4). SARS-CoV-2 uses angiotensin-converting enzyme 2 (ACE2) as its entry receptor, which is expressed in liver and bile duct cells (5,6), suggesting direct liver injury in patients with COVID-19.In contrary, isolated splenic affection usually passed unnoticed unless splenicthrombosis or infarction with superadded abscess formation occurred [(7)]. Here we describe apostcovid

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multiple hepatic and splenic abscesses without previous abnormalities shortly after COVID-19 infection in otherwise healthy 13 -years girl.

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Introduction:-

13 years old girl presented to the ED with severe upper abdominal pain of 10 days duration associated with vomiting, fever and bodyache. The patient has a recent history of COVID-19 infection 6 months back with no obvious sequala. Two months after clearance from COVID-19 infection, she received 2 doses of Pfizer vaccine. Three months later, she developed cough, rhinorrhea, sore throat. COVID-19 test turned positive with CRP 205mg/L, WBC 13.7x10^3/uL, platelet 695x10^3/uL. The patient clinically has high grade fever 40 C, with normal saturation on room air, no pneumonia on chest x ray, but lab revealed leukocytosis, thrombocytosis, iron deficiency anemia, elevated cardiac markers. No abdominal pain nor upper or lower gut symptoms. The patient admitted and started medical treatment ceftriaxone and enoxaparin and antiviral medications; Favipiravir 400 mg orally twice daily. 5 days later, fever subsided with improvement of labs; WBC 10.3x10^3/uLplatelet 751x10^3/uL, CRP 255 mg/L, D-dimer 0.6ug/ml FEU, procalcitonin 0.9ng/mL, and patient discharged home.

One week later the patient presented again to the ED with anorexia, repeated vomiting, abdominal pain and low-grade fever 37.3 C. leukocytosis 22.7x10^3/uLwith absolute neutrophilia, CRP 395 mg/L, D- dimer 1.25ug/ml FEU, Procalcitonin 1.4ng/mL. liver function revealed normal bilirubin with elevated alkaline phosphatase 198U/L. COVID-19 test was negative.

CT abdomen with contrast revealed showed multiple cystic lesions in the liver and spleen showing marginal enhancement the largest (1.1 x 1 cm), diffuse hepatic and splenic perfusion differences with multiple small portal venules and splenic segmental linear filling defects denoting thrombosis pointing to abscess formation and possible small portal and splenic venous thrombosis.The diagnosis of multiple hepatosplenic micro-abscesses was established as shown in Figure [1,2].

Patient was admitted for symptomatic treatment and IV antibiotics and for further workup and septic screening. Virology panel was done and was normal. Echocardiogram to exclude endocarditis was normal. After 3 days the symptoms subsided with improved laboratory finding:WBC 14x10^3/uL, D dimer 2.1ug/ml FEU, procalcitonin 4.3ng/mL, CRP 295 mg/L. Blood culture was positive for gram positive bacilliCutibacterium acnes contamination. The patient was discharged against medical advice)

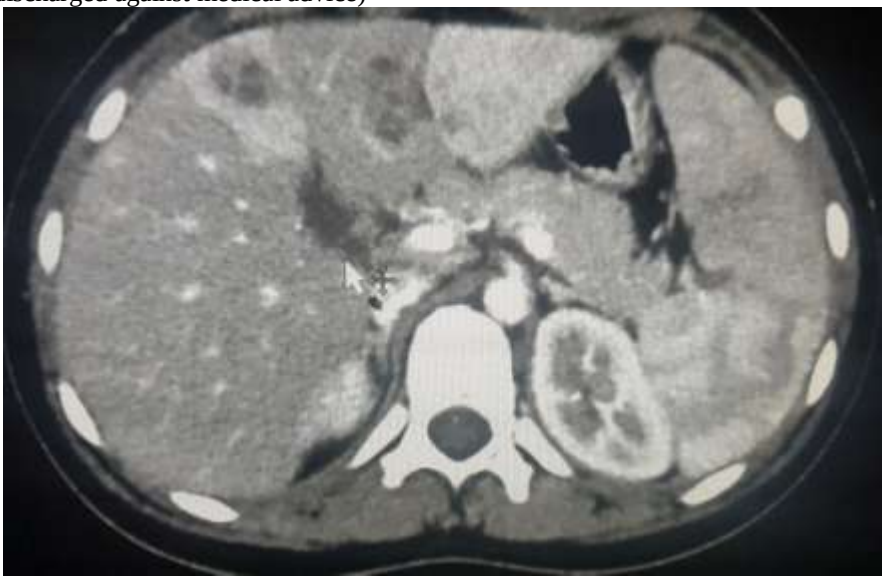

Figure 1:- Hepatic abscess with marginal enhancement.

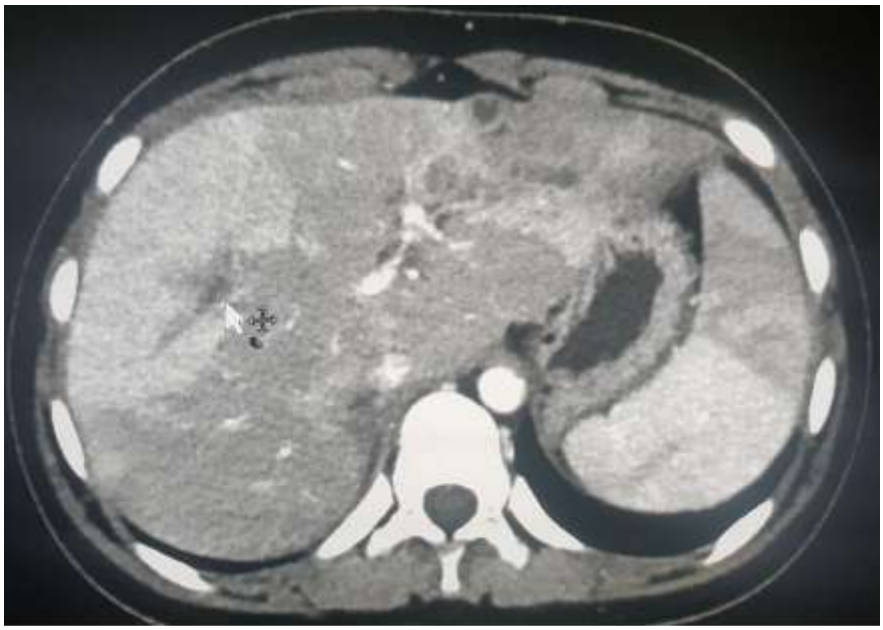

Figure 2:- Heterogenous perfusion with terminal portal and splenic venule thrombi.

One month later, she came to the family physician complaining of Post viral fatigue syndrome with no fever nor abdominal pain with complete resolution of the previous symptoms.

Discussion:-

Our case is an example of disseminatedcoagulopathy associated with COVID-19 infection which is well known in literature. While the most common organ affected is the pulmonary vasculature with its lethal sequalae, intra-abdominal vessels are less frequently appreciated during COVID-19 infection[8,9,10].

The intense cytokine and proinflammatory response to corona virus seems to be the mainculprit for such widespread hypercoagulable state[11].

Recent studies have illustrated that patients with venous thromboembolism are at great risk of arterial thrombotic complications than matched population. Therefore, it seems that the two vascular complications may be simultaneously triggered by biological stimuli responsible for activating coagulation and inflammatory pathways in both vascular system[12].

Apart from therespiratory system, the liver is the most frequently damaged organ in COVID-19. The mechanism of hepatic injury is not fully appreciated, but it seems to be multifactorial and attributable to direct viral effect that results in damage to cholangiocytes, immune related injury, and/or drug hepatotoxicity. The ACE2 receptorsarehighlyexpressed in the liver, particularly in cholangiocytes and this may be responsible for liver damage in covid-19 infection. Researchers have found that viral infection with COVID-19 impairs the barrier and bile acid transport functions of cholangiocytes through dysregulation of genes involved in tight junction formation and bileacid transportation. This could explain the accumulation of bile acids and resultant liver damage in patients [13]

Current data show that 14.8%– 53% of patients with COVID-19 have abnormal liver function tests [14].Also, approximately 50% of patients with COVID-19 had increased levels of γ-glutamyl transferase (GGT) denoting cholestasis [15].

In theliterature to date, hepatic manifestations of COVID-19 were mostly transient, although severe liver insult may occur. The proportion of liver injury was also higher in patients with severe COVID-19[16]. Periportal necrosis, lymphocytic infiltration of the sinusoids, dense portal infiltration by abnormally small lymphocytes, central vein thrombosis, and cirrhotic changes with thick fibrosis were all findings at liver autopsy[17].

Another potential cause of liver damage and elevated liver enzyme levels is microthrombosis within hepatic sinusoids, which likely is related to previously described generalized coagulopathy, as well as liver-induced coagulopathy (18). Hypoxic or ischemic hepatic injury, resulting from a coagulopathy-induced decrease in hepatic perfusion.

Our case demonstrated multiple hepatic and portal venule thrombosis with heterogenous area of perfusion which is most likely one of the factors pointing toward the formation of hepatic focal breakdown and subsequent infection. We assume that this is the primary reason for initiating the inflammatory process in our case.

Another contributing factor which may predispose to the persistent inflammatory reaction in our case can be explained by reported risk of hepatic steatosis in COVID -19 infection [17]. In fact, it has been reported that hepatic steatosis is an independent risk factor of severe disease [19]. Moreover, biliary stasis without obstructive cause were detected in 54% of patients with COVID-19, and it is important to note that acute cholecystitis can develop as a result of biliary stasis with sludge or mud formation predisposing to superadded infection or bacterial translocation[20].

Another leading cause depicting the occurrence of liver abscess in our case is the post-recovery state of immunosuppression that would enforce serious bacterial and fungal infections[21]. The virus-mediated immunosuppression enables opportunistic organisms to colonize vulnerable tissues including the liver and spleen in the affected patients[22].

In review of the previous reports, it seems that our case had the same potentials to have a complicated course post COVID-19 infection which has led to the development of liver and splenic abscesses. The detailed analysis of the history and the clinical course has clearly identified multiple factors for her sequala. We assume the direct effect of the viremia on cholangiocytes causing stasis of intrahepatic bile together with intense inflammatory reaction and microthrombi causing potential inflammatory foci that coalesce together forming small areas of tissue necrosis and liver parenchyma breakdown.This has been reinforced by the prolonged period of viremia and the immunosuppression. The positive blood culture in our case has revealed Cutibacterium acnes contamination which is a normal skin flora and known opportunistic organism. This is clearly supporting our hypothesis of immunosuppression and opportunistic infection over foci of tissue breakdown and cholestasis.

To the best of our knowledge, this is the first case to report multiple liver and splenic abscesses in the same patient. Previous reports detected either hepatic or splenic abscesses post COVID-19 infection.

Alhaddad et al, reported liver abscess evoked by COVID-19 infection and described the same pathophysiological pathways [23]. Other cases are also reported (24).

The cause of splenic abscess is mainly hematogenous, with infective endocarditis being the most common source[25]. In our case, we excluded the common causes of splenic abscess, which usually have Gram-negative bacillus as the most common culprit pathogen isolated. The blood culture in our case have revealed an opportunistic gram-positive bacillus which reinforce the same mechanism explained in liver affection.

The identical pathways described before it was reported by researchers regarding splenic abscesses in COVID-19 patients (26,27,28).AlZarooni et al described a case series of 3 patient developed splenic abscess alone duringCOVID-19 infection with different treatment strategies [29].

The optimal treatment strategy for splenic abscess remains controversial.To date, the traditional surgical approach involving splenectomy has been the treatment of choice[28]. currently, some experts consider antibiotics alone as an adequate treatment option, with success rates of 75%[30].

Regarding our case, we chose to start conservative treatment in the form of IV antibiotics and symptomatic treatment based on the small sizes of liver and splenic abscesses and underlying microthrombi, which goes with recommended treatment protocols in such cases.

Conclusion:-

COVID-19 infection may be a major risk for the development of hepatic and splenic abscess. The reason of which is multifactorial including, hypercoagulability,impaired immunityand superinfection. However, further studies are needed to ensure a clear pathophysiologyof COVID-19 related liver and splenic affection.Low threshold of suspicion about these conditions in management of COVID-19 patient is warranted.

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