2. Sarpel U, Jacob BP, Steinhagen RM: Reduction of a Large Incarcerated Rectal Prolapse By Use of an Elastic Compression Wrap. Dis Colon Rectum, 2005; 48: 1320–22
3. Ris F, Colin J-F, Chilcott M, Remue C, Jamart J, Kartheuser A. Altemeier's procedure for rectal prolapse: analysis of long-term outcome in 60 patients. Colorectal Dis. 2012;14(9):1106–11.
4. Cernuda RB, Ángel JP, Fernández NT, Sánchez-Farpón JH, Pérez JAÁ. Perineal Rectosigmoidectomy (Altemeier Procedure) as Treatment of Strangulated Rectal Prolapse. J Gastrointest Surg. 2016;20(12):2102–3.
5. Brisighelli G, Di Cesare A, Morandi A, Paraboschi I, Canazza L, Consonni D, *et al*. Classification and management of rectal prolapse after anorectoplasty for anorectal malformations. Pediatr Surg Int. 2014;30(8):783–9.

Acute Hepatitis-A with COVID-19: Is it coinfection or Mere a Coincidence

Anil Tambe[1]
Pankaj Nawghare[2]
Padmaja Biradar[3]
Pravin M. Rathi[2]
Surbhi Rathi[3]

[1]Department of Gastroenterology, Jagjivan Ram Hospital, Mumbai. [2]Department of Gastroenterology, [3]Department of Pediatrics, B.Y.L. Nair Charitable Hospital & T.N. Medical College, Mumbai.

***Corresponding Author:** Dr Pankaj Nawghare*
Email: pankaj9ghare21@gmail.com

In December 2019, a cluster of pneumonia cases caused by Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), a novel virus, emerged in Wuhan, China.[1] The disease showed rapid spread globally and is recognized as a global pandemic by the World Health Organization. Coronavirus typically presents with symptoms of viral pneumonia and primarily manifests as a pulmonary disease. However, increasing data suggests the involvement of multiple organ systems, including the gastrointestinal tract and liver, with more than 60% of patients presenting with GI symptoms and significant proportion of cases presenting with deranged liver function tests.[2]

Case Report

A 6 years old boy presented to the hospital with complaints of yellowish discoloration of eyes since 10 days, and high colored urine. On enquiry, there was a history of fever; present since, 10-12 days, mild to moderate grade, intermittent, and pain in abdomen since, 5 days. There was no history of cough, sore throat, breathlessness, vomiting, loose stools and no h/o jaundice in the past.

On presentation, he was vitally stable, temperature was 37.2 °C. Mild pallor and icterus was present. Abdominal examination was suggestive of right hypochondriac tenderness, with hepatomegaly, 4 cm palpable below right coastal margin, soft in consistency. There was no splenomegaly or ascites. Respiratory system examination was normal. Other systemic examinations were unremarkable.

Laboratory results are depicted in **table 1**. An abdominal sonogram revealed a mildly enlarged liver with normal echotexture. Serological tests were performed, Hepatitis A IgM was positive. In view of persistent fever during hospital stay, further evaluation was done. Urine routine/microscopy and blood culture were negative, Chest x-ray was normal. However, nasopharyngeal swab taken for the SARS CoV PCR test was positive.

The patient was managed conservatively with symptomatic treatment. He improved during the hospital stay, and fever subsided. Repeat laboratory investigation was suggestive of a decreasing trend of the liver enzymes and serum bilirubin levels. Repeat nasopharyngeal swab for SARS CoV-2 PCR test, after 10 days was negative. The patient was discharged with home quarantine for 7 days and follow-up on OPD basis. During the follow-up visit he did not have any complaints and repeat LFT was normal.

Table 1: Laboratory results

Date	03/6/20	08/6/20	15/6/20	02/09/20
Hb / TLC / PLT		8.4/6900/400000		
Bilirubin T/D		7.4 / 3.4	3.4 / 1.2	0.6/0.1
AST / ALT	186/280	222 / 204	87 / 121	18/23
Alkaline Phosp		471	404	382
Total Protein /Albumin		8.0 / 3.5	7.8 / 3.7	7.9/4.0
INR		1.56	1.2	0.9
SARS CoV-2 PCR		Positive	Negative	
Hepatitis A IgM	Positive			

Discussion

Studies about the relationship between underlying mechanisms of COVID-19 and liver dysfunction are evolving. COVID-19 uses the angiotensin-converting enzyme 2 (ACE2) as the binding site to enter the host cell[3] and Chai *et al*, found that both liver cells and bile duct cells express ACE2.[4] The spectrum of liver injury ranges from asymptomatic abnormalities in liver biochemistry to acute liver failures in rare cases. The purported mechanisms for hepatic manifestations include, the possibility of direct cytopathic effect of the virus, liver injury related to accentuated immune response (cytokine storm) and immune mediated damage, drug toxicity, and ischemic hepatitis.[5]

Liver injury in the setting of COVID-19 presents a unique dilemma to the clinician. There is often an uncertainty regarding a preexisting undiagnosed liver disease. Many drugs used to treat moderate to severe COVID-19 cases have their own profiles of liver toxicity. Multiple cases of underlying chronic liver disease (Hepatitis B, Hepatitis C, NAFLD, Alcoholic cirrhosis) with COVID 19 and its clinical implications have been described. The incidence of liver injury in COVID 19 patients ranged from 58% to 78%, mainly indicated by elevated AST, ALT, and total bilirubin levels accompanied by slightly decreased albumin levels, with a higher proportion being present in severe COVID-19 cases.[6] The range of AST and ALT elevation is usually mild (ie, <5 times the upper limit of normal); however, higher aminotransferase levels and severe acute hepatitis have also been reported.[7] The pattern of elevation is often AST greater than ALT. The presence of liver injury during the COVID 19 infection has been associated with increased complication and high risk of mortality. When comparison of severe with non-severe COVID-19 cases was done, liver function abnormalities like hypoalbuminemia, GGT, and aminotransferase and bilirubin elevations were more frequent in those with severe disease[5].

Hepatitis A virus (HAV) infection is a widespread disease, accounting for 1.4 million cases annually worldwide. In high endemic areas the reported incidence of HAV may reach 150 per 100,000 per year[8]. It has a feco-oral mode of transmission. Clinical manifestations of symptomatic HAV infection vary from mild illness to fulminant hepatitis[8]. Although co-infection of Hepatitis A with other viruses is known, co-infection with COVID 19 has not been described.

Children are more likely to have asymptomatic SARS CoV 2 infections and experience mild symptoms.[9] A Meta-analysis by Cheung KS, *et al*, of >4000 East Asian patients with COVID-19 described up to 20% had GI symptoms and viral RNA was detected in the stool of almost 50% of patients.[10] Recent reports further suggest persistence of fecal viral shedding after symptom resolution in children.[11] Study by Santos VS, *et al* showed that children with COVID-19 had persistent fecal viral RNA shedding, even after RT-PCR tests of respiratory tract had become negative. The duration of SARS CoV-2 RNA shedding was longer in fecal than respiratory samples with a mean difference of 8.6 days (95% CI 1.7 to 15.4).[12]

Currently, many trails and investigations indicate the strong relationship between other viruses, bacteria,

fungi, and SARS CoV-2. On review of limited literature to date, our case appears to be the first reported case of COVID-19 infection presenting with coinfection with hepatitis A virus. Common route of infection (feco-oral) may be a possible explanation for coinfection of COVID-19 and hepatitis A. Although elevated liver biochemistries are commonly seen in hospitalized patients with COVID-19, it should not be assumed that these findings are a manifestation of COVID-19. So, it is important to rule out coinfection with hepatitis virus as it may impact management and clinical outcome of COVID-19 patients. Such patients should be isolated from other patients to prevent transmission of infection. We also encourage patients to be vaccinated against hepatitis A to reduce the risk of coinfection.

References

1. Chen N, Zhou M, Dong X, *et al*. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020;395:507-513.
2. Redd WD, Zhou JC, Hathorn KE, *et al*. Prevalence and Characteristics of Gastrointestinal Symptoms in Patients With Severe Acute Respiratory Syndrome Coronavirus 2 Infection in the United States: A Multicenter Cohort Study. Gastroenterology. 2020;159(2):765-767.e2. doi:10.1053/j.gastro.2020.04.045.
3. Hoffmann M, Kleine-Weber H, Krueger N, Mueller MA, Drosten C, Poehlmann S. The novel coronavirus 2019 (2019-nCoV) uses the SARS-coronavirus receptor ACE2 and the cellular protease TMPRSS2 for entry into target cells. bioRxiv. 2020.
4. Chai X, Hu L, Zhang Y, *et al*. Specific ACE2 expression in cholangiocytes may cause liver damage after 2019-nCoV Infection. bioRxiv. 2020.
5. Kumar-M P, Mishra S, Jha DK, *et al*. Coronavirus disease (COVID-19) and the liver: a comprehensive systematic review and meta-analysis [published online ahead of print, 2020 Jul 4]. Hepatol Int. 2020;1-12. doi:10.1007/s12072-020-10071-9.
6. Zhang B, Zhou X, Qiu Y, *et al*. Clinical characteristics of 82 death cases from COVID-19. PLoS ONE. 2020;15:e0235458.
7. Bertolini, A., van de Peppel, I.P., Bodewes, F.A., Moshage, H., Fantin, A., Farinati, F., Fiorotto, R., Jonker, J.W., Strazzabosco, M., Verkade, H.J. and Peserico, G. (2020), Abnormal liver function tests in COVID-19 patients: relevance and potential pathogenesis. Hepatology.
8. Kumar KJ, Kumar HC, Manjunath VG, Anitha C, Mamatha S. Hepatitis A in children- clinical course, complications and laboratory profile. Indian J Pediatr. 2014;81(1):15-19. doi:10.1007/s12098-013-1114-8.
9. Kelvin AA, Halperin S. COVID-19 in children: the link in the transmission chain. Lancet Infect Dis. March 2020. doi:10.1016/S1473-3099(20)30236-X.
10. Cheung KS, Hung IFN, Chan PPY, *et al*. Gastrointestinal Manifestations of SARS-CoV-2 Infection and Virus Load in Fecal Samples From a Hong Kong Cohort: Systematic Review and Meta-analysis. Gastroenterology. 2020;159(1):81-95. doi:10.1053/j.gastro.2020.03.065.
11. Xu Y, Li X, Zhu B, *et al*. Characteristics of pediatric SARSCoV- 2 infection and potential evidence for persistent fecal viral shedding. Nat Med. 2020;26(4):502-505. doi:10.1038/s41591-020-0817-4.
12. Santos VS, Gurgel RQ, Cuevas LE, Martins-Filho PR. Prolonged Fecal Shedding of SARS-CoV-2 in Pediatric Patients: A Quantitative Evidence Synthesis. J Pediatr Gastroenterol Nutr. 2020;71(2):150-152. doi:10.1097/MPG.0000000000002798.
13. Huang C, Wang Y, Li X, *et al*. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020. doi:10.1016/S0140 6736(20)30183-5.

Dizygotic Twins with Irritable Bowel Syndrome and Interleukin-6 Promoter Polymorphism (-174 G/C).

Saugata Acharyya
Kakoli Acharyya

Department of Paediatrics, Calcutta Medical Research Institute, 7/2 Diamond Harbour Road. Kolkata-700027. India.

Corresponding Author: *Dr Saugata Acharyya*
Email: acharyyasaugata@yahoo.com