axial skeleton bone marrow heterogeneous hypercaptation (Fig. 1C, D).

While making the assessment, liver function worsened. The patient experienced a deterioration of the liver biochemistry, with bilirubin 24.1 mg/dl and ALP 1065 U/L, and developed moderate ascites, with good response to diuretic treatment. Signs of acute liver failure were not seen during assessment.

Therefore, with the diagnosis of AL amyloidosis with exclusively liver involvement, chemotherapy treatment was initiated with bortezomib (adjusted-dose 0.7 mg/m^2, according to data sheet because of hyperbilirubinemia), cyclophosphamide and dexamethasone. Clinical and laboratory improvement was initially achieved, bringing bilirubin levels down to 10 mg/dl four weeks after initiation of treatment.

However, one month later the patient was readmitted to hospital due to hepatic decompensation with ascites, spontaneous bacterial peritonitis and bilateral pneumonia and finally died due to respiratory failure.

The clinical spectrum of hepatic amyloidosis can range from hepatomegaly (most frequently physical sign observed) and mild abnormal liver function tests, to more severe symptoms rarely observed, such as jaundice, intrahepatic cholestasis, portal hypertension, hepatic failure or spontaneous liver rupture.[1,3–5] Jaundice and intrahepatic cholestasis as the primary manifestation of the disease is very uncommon (<5% cases).[4]

Hyperbilirubinemia and marked elevation of serum alkaline phosphatase may indicate a poor prognosis of hepatic amyloidosis.[1,3–5]

Diagnosis of hepatic amyloidosis is based on a high clinical suspicion, exclusion of other infiltrative disorders of the liver (tuberculosis, sarcoidosis, malignancy and glycogen storage diseases) and tissue biopsy stained with Congo red demonstrating amyloid deposits with apple-green birefringence.[1,2,4,5]

Treatment of AL amyloidosis is based on chemotherapy to eradicate the underlying clone, usually combinations of bortezomib, cyclophosphamide and dexamethasone.[2,4] Patient who meet the criteria will be eligible for autologous hematopoietic cell transplantation.

Liver involvement in patients with amyloidosis is often an indicator of poor prognosis,[3] having been reported as 9 months the median survival of patients with primary hepatic amyloidosis.[1,3–5]

Conflicts of interest

There are no financial or other conflicts of interest regarding this article.

References

1. Park MA, Mueller PS, Kyle RA, Larson DR, Plevak MF, Gertz MA. Primary hepatic amyloidosis in 98 patients. Medicine (Baltimore). 2003;82.
2. Gertz MA. Immunoglobulin light chain amyloidosis: 2020 update on diagnosis prognosis, and treatment. Am. J. Hematol. 2020.
3. Zhang L, Shen K, Zhang C, Qiu Y, Miao H, Feng J, et al. Clinical presentation and prognostic analysis of Chinese patients with systemic light chain amyloidosis with liver involvement. Leuk. Res. 2019;86.
4. Dias T, Ferreira D, Moreira H, Nascimento T, Santos A, Carvalho A. A case of severe cholestasis due to hepatic AL amyloidosis. GE Port J Gastroenterol. 2019;26:425–9.
5. Syed U, Ching Companioni RA, Alkhawam H, Walfish A. Amyloidosis of the gastrointestinal tract and the liver: clinical context, diagnosis and management. Eur. J. Gastroenterol. Hepatol. 2016;28.

Diego Martínez-Acitores de la Mata[a,*], Silvia Bravo Meléndez[a], Candela Ceballos Bolaños[b], Irene Amat Villegas[c], María Carmen Mateos Rodriguez[b], Mara Pilar Huarte Muniesa[a], Susana Oquiñena Legaz[a], Montserrat Alvarellos Outerio[b], Sara Pérez Ricarte[d]

[a] *Department of Gastroenterology, Complejo Hospitalario de Navarra, Pamplona, Spain*
[b] *Department of Haematology, Complejo Hospitalario de Navarra, Pamplona, Spain*
[c] *Department of Pathology, Complejo Hospitalario de Navarra, Pamplona, Spain*
[d] *Department of Internal Medicine, Complejo Hospitalario de Navarra, Pamplona, Spain*

* Corresponding author.
E-mail address: Diegoacitores132@hotmail.com
(D. Martínez-Acitores de la Mata).

Dysentery as a rare GI symptom found in COVID-19 patients

La disentería como síntoma digestivo poco frecuente que se encuentra en los pacientes con COVID-19

Coronavirus Disease 2019 (COVID-19) caused by SARS-CoV-2 first started in Wuhan, China, and soon turned to a pandemic and still continue to spread across the world.[1,2] With an increasing number of COVID-19 cases outside of China, our clinics in Shahid Beheshti Hospital, Qom city, was faced with a large number of patients who were suspected of being infected with the SARS-CoV-2. Following the outbreak of COVID-19, the prevalence of gastrointestinal (GI) tract symptoms significantly goes up. This incidence in COVID-19 patients is well documented in literatures.[3] After February 20, 2020, the day that Iran reported as the start of COVID-19 outbreak, the number of patients referred to our GI clinic was unusually increased. The most important complaint of patients was the incidence of some unusual GI symptoms resistant to medication.[4]

Table 1 Serial laboratory results of 43-year-old woman with presentation of dysentery due to the infection with SARS-CoV-2.

Blood biochemistry			Serology, endocrinology and tumor marker			ESR 1 h, mm/h	21[a]	0–20
Test, unit	Result	Reference range	Test, unit	Result	Reference range			
FBS, mg/dL	109	70–99 Diabetic: >126	CRP, mg/L	8.4[a]	Up to 6	WBC, $\times 10\hat{3}$ /uL	7	4.1–10.5
Creatinine, mg/dL	1.54[a]	0.7–1.4	Ferritin, ng/mL	95.8	20–250	Neutrophil, $\times 10\hat{3}$ /uL	5.70	2–7.7
Direct bilirubin, mg/dL	0.36[a]	0–0.3	AFP (Clia), IU/mL	3.22	0–4	Lymphocyte, $\times 10\hat{3}$ /uL	3.61	1–2.7
						Monocyte, $\times 10\hat{3}$ /uL	1.04	0.3–0.7
SGOT (AST), U/L	30	11–37	CEA (Clia), IU/mL	2.02	Up to 4.7	Eosinophil, $\times 10\hat{3}$ /uL	0.32	0.2–0.6
SGPT (ALT), U/L	28	13–40	*Hematology*			Basophil, $\times 10\hat{3}$ /uL	0.03	0.01–0.3
Iron (Fe), μg/dL	89	40–120				RBC, $10\hat{6}$/μL	4.31	4.5–5.9
TIBC, μg/dL	419	230–440	*Test, Unit*	*Result*	*Reference range*	Hb, g/dL	13.80	13.5–17.5
Hb A1c, %	6.2	Non diabetic: 4–6 Diabetic: >6.5	LDH	400	Up to 300	Platelet, $10\hat{3}$/μL	92.6	80–100

Abbreviation: FBS: Fasting Blood Sugar; AST: Aspartate Amino Transferase; ALT: Alanine Transferase; TIBC: Total Iron-Binding Capacity; Hb A1c: Glycosylated Hemoglobin; CRP: C-Reactive Protein; AFP: Alpha-Fetoprotein; CEA: Embryonic Carcinoma Antigen; LDH: Lactate Dehydrogenase; WBC: White Blood Cell; RBC: Red Blood Cell; Hb: Hemoglobin.

[a] Higher in comparison to the reference value.

One of the questions that need to be answered promptly is that whether the incidence of rare GI symptoms is possible in confirmed COVID pneumonia patients. The answer to this question may help physicians take better diagnostic and therapeutic approaches for patients with this symptom. Currently, we have reported the clinical data in details as well as the result of chest CT of a COVID-19 patient with dysentery.

A 43-year-old woman was referred to our gastroenterology clinic, complaining from dysentery for one week. She did not mention any cough, dyspnea or respiratory disorders. Her past medical history was unremarkable and she ignored smoking, drinking or using any drugs and medications. Respiratory rate: 18 per minute and body temperature from oral root: 37.5–38 °C. In order to accurately evaluate the patient, laboratory test was performed. The results are summarized in (Table 1). The levels of CRP, LDH and ESR 1 h were higher than the normal range and were equal to 8.4, 400 and 21 respectively. Alkaline phosphatase, amylase serum and bilirubin values of the patient were in normal range. Medication with ciprofloxacine, 500 mg and metrodinazole, 500 mg was started after the first symptoms of dysentery, to reduce the volume of diarrhea and severe dehydration. Due to the persistence of diarrhea and the

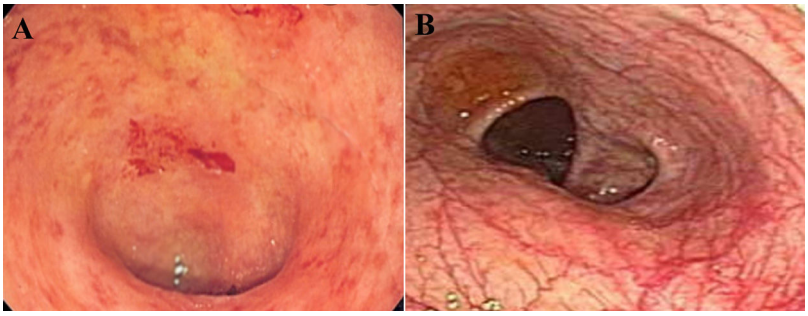

Figure 1 Colonoscopy finding of 43-year-old woman with presentation of dysentery which was resulted in patchy erythema.

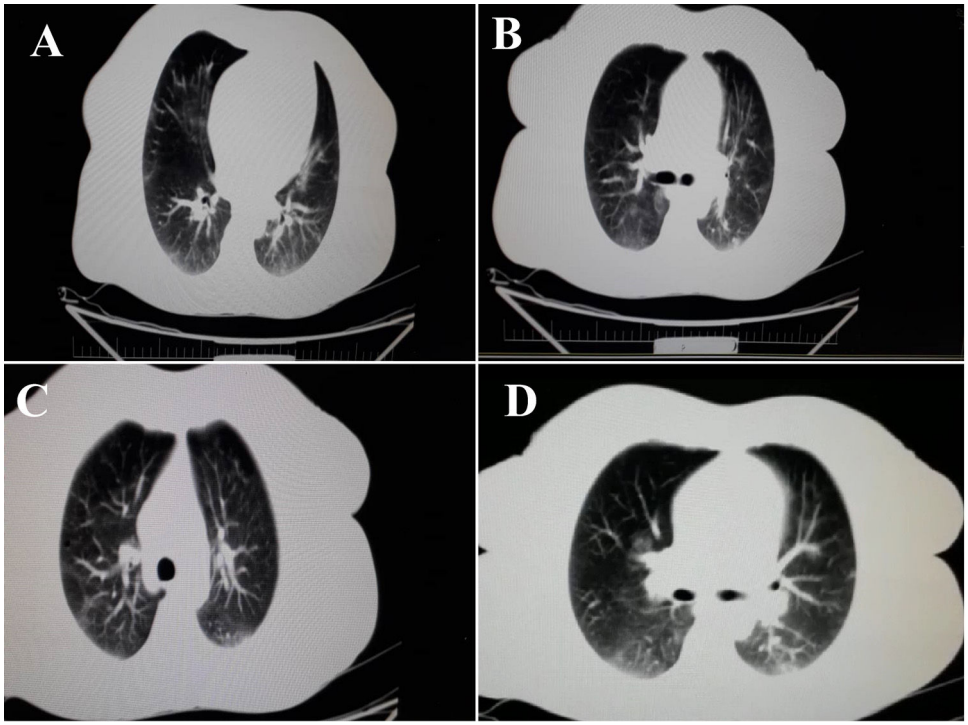

Figure 2 Chest CT scans (transverse plane) of 43-year-old woman with presentation of dysentery due to the infection with SARS-CoV-2. Chest CT resulted bilateral peripheral ground glass, crazy paving and small consolation opacities.

lack of therapeutic response to medications, as well as evaluation for the probability of inflammatory bowel disease (IBD) in the patient, colonoscopy was performed which was resulted in patchy erythema (Fig. 1). Also, pathological finding was associated with the infiltration of inflammation cells which made us suspect infectious (viral) colitis. Since the incidence of such GI symptoms coincided with the COVID-19 outbreak in Iran, and also more importantly since the patients reported to reside in high risk areas, we suspected patients to be infected by SARS-CoV-2. Therefore, for more validation, laboratory-confirmed COVID pneumonia was performed by using SARS-CoV-2 conventional polymerase chain reaction assay and sequencing of the polymerase chain reaction (PCR) amplicons, which was reported to be positive for the patient. After performing chest CT scan, we noticed the bilateral lung involvement, as revealed bilateral peripheral ground glass, crazy paving and small consolation opacities (Fig. 2). Patient was isolated in a negative pressure room for one week and underwent medications as mentioned above.

According to the findings of Zou et al., about the role of ACE-2 as the main host cell receptor for entrance of 2019-nCoV to cell and with notice to the high expression of ACE-2 receptor in the oral cavity, as well as in colon, intestine and gallbladder and enriching in epithelial cells, we supposed that this receptor may mediate some GI symptoms of infected patients.[5] Previously, epidemiologic finding of Lai et al., demonstrated that among adult patients, some common respiratory symptoms were followed by diarrhea.[3] Previously, we reported that infected patients with COVID-19 can present some unusual GI symptoms.[4] These symptoms were resistant to medications and in some cases without any medications, they subsided.

At the age of COVID-19 crisis, in some patients, GI physicians may face rare GI symptoms such as dysentery, hepatic involvement or other ones in patients. Therefore, it is necessary that all GI physicians should be aware of the possible occurrence of these symptoms as an important prognosis of COVID pneumonia. Although, in this letter we only documented one case of this issue, at the age of COVID-19, all rare GI symptoms should be exactly addressed in new referred patients to GI clinic. Recording and investigation of these symptoms may open a new window to help finding valuable information on the characterization of this mysterious disease.

Declarations

Ethics approval

Approval was obtained from the ethics committee of Qom University of Medical Sciences. The procedures used in this study adhere to the tenets of the declaration of Helsinki (Nu: IR.MUQ.REC.1399.044).

Consent to publish

Patients signed informed consent regarding publishing their data and photographs.

Availability of supporting data

All data and materials are available.

Authors' contribution

Ahmad Hormati, Writing of the report and therapeutic physician. Mohammad Reza Ghadir, Therapeutic physician. Reza Aminnejad & Fatemeh Khodadust, Review and edit of the manuscript. Mahboubeh Afifian, Collecting of the data. Sajjad Ahmadpour, Writing of the report and correspondence.

Funding

No funding was provided for this manuscript.

Conflict of interest

The authors declare that they have no conflict of interest.

Acknowledgments

The authors would like to thank Sanam Ahmadpour for her contribution to the edit of the present manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.gastrohep.2020.06.005.

References

1. Salzberger B, Glück T, Ehrenstein B. Successful containment of COVID-19: the WHO-Report on the COVID-19 outbreak in China. Infection. 2020;48:151–3.
2. Cheng ZJ, Shan J. 2019 Novel coronavirus: where we are and what we know. Infection. 2020;48:155–63.
3. Lai CC, Shih TP, Ko WC, Tang HJ, Hsueh PR. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and coronavirus disease-2019 (COVID-19): the epidemic and the challenges. Int J Antimicrob Agents. 2020;55:105924.
4. Hormati A, Shahhamzeh A, Afifian M, Khodadust F, Ahmadpour S. Can COVID-19 present unusual GI symptoms? J Microbiol Immunol Infect. 2020, http://dx.doi.org/10.1016/j.jmii.2020.03.020.
5. Zou X, Chen K, Zou J, Han P, Hao J, Han Z. Single-cell RNA-seq data analysis on the receptor ACE2 expression reveals the potential risk of different human organs vulnerable to 2019-nCoV infection. Front Med. 2020, http://dx.doi.org/10.1007/s11684-020-0754-0.

Ahmad Hormati[a,b], Mohammad Reza Ghadir[b], Mohammad Saeidi[c], Reza Aminnejad[c,d], Fatemeh Khodadust[e], Mahboubeh Afifian[f], Sajjad Ahmadpour[b,*]

[a] *Gastrointestinal and Liver Diseases Research Center, Iran University of Medical Sciences, Tehran, Iran*
[b] *Gastroenterology and Hepatology Diseases Research Center, Qom University of Medical Sciences, Qom, Iran*
[c] *Department of Anesthesiology and Critical Care, Qom University of Medical Sciences, Qom, Iran*
[d] *Department of Anesthesiology and Critical Care, Shahid Beheshti University of Medical Sciences, Tehran, Iran*
[e] *Department of Radiopharmacy, Faculty of Pharmacy, Mazandaran University of Medical Sciences, Sari, Iran*
[f] *Health Information Technology, Tehran University of Medical Sciences, Tehran, Iran*

[*] Corresponding author.
E-mail address: sajjadahmadpour@yahoo.com
(S. Ahmadpour).