Case report

Crit Care Shock (2024) 27:135-140

Early detection of portal hypertensive gastropathy in a cirrhotic patient using point of care ultrasound: A case report and review of the literature

Mohamed Gohar[1], Zouheir Bitar[1], Ossama Maadarani[1], Faisal Alshatti[2]

Abstract

Portal hypertensive gastropathy (PHG) is common in liver cirrhotic patients, usually manifesting as chronic gastrointestinal bleeding. It has been linked to gastroparesis and delayed gastric emptying. It is usually diagnosed by endoscopy.

Point-of-care ultrasound (POCUS) is a rapidly emerging bedside diagnostic tool that is becoming very popular in medical practice. It helps physicians reach early diagnoses in various situations.

In our case, POCUS was used for early diagnosis of PHG in a cirrhotic patient complaining of recurrent vomiting.

Key words: Portal hypertensive gastropathy, gastroparesis, point of care ultrasound.

Introduction

Point-of-care ultrasound (POCUS) is becoming very popular, as it is a readily accessible and easy-to-use tool for early diagnosis of different conditions. POCUS diagnoses cardiac, respiratory, abdominal, and traumatic conditions. Moreover, increased interest has recently risen in POCUS's use in diagnosing gastric-related diseases.

Portal hypertensive gastropathy (PHG) is a common complication of liver cirrhosis that usually occurs on top of portal hypertension. It usually causes chronic bleeding and anemia but is also linked to gastroparesis and delayed gastric emptying.

In our case, we used POCUS to raise the suspicion and diagnose PHG in a patient known to be cirrhotic and complaining of recurrent vomiting, suggesting gastroparesis.

Case

Our case was that of a man 79 years old. He had a known medical history of diabetes mellitus, hypertension, and ischemic heart disease, with a pacemaker inserted for intermittent atrioventricular heart block and decompensated liver. He was known to have liver cirrhosis on the backdrop of non-alcoholic fatty liver disease (NAFLD).

The patient had a history of recurrent admissions with refractory ascites necessitating therapeutic paracentesis and previous endoscopies showing esophageal varices and serial endoscopic band ligation.

The patient was seen in the Emergency Department with the main complaint of generalized weakness and constipation for a few days and denied any history of fever, cough or expectoration, chest pain, dysuria, or disturbed consciousness level.

On exam, the patient was conscious, alert, and well-oriented, with no signs of hepatic encephalopathy.

[1.] Critical Care Unit, Internal Medicine Department, Ahmadi Hospital-Kuwait Oil Company
[2.] Endoscopy Unit, Internal Medicine Department, Ahmadi Hospital-Kuwait Oil Company

Address for correspondence:
Mohamed Gohar
Email: mgohar@kockw.com

He was found to be overloaded with marked ascites, extensive bilateral lower limb edema, and bilateral pleural effusion.

He was admitted to our hospital due to decompensated liver disease representing ascites. Moreover, it was planned for therapeutic paracentesis.

During admission, the patient developed several episodes of vomiting that led to aspiration pneumonia and respiratory failure. The patient's level of consciousness became compromised, and he failed to maintain saturation on noninvasive ventilation, so he was intubated and connected to a mechanical ventilator. A nasogastric tube was inserted, and the patient kept nil per oral (NPO) with gastric drainage.

Bedside POCUS was performed focusing on the stomach, and it showed a significantly dilated stomach with stagnant food content and a markedly thickened wall (**Figure 1**), raising the suspicion of gastroparesis on top of portal hypertensive gastropathy, given his known cirrhotic history.

An endoscopy was then performed, confirming our suspicion as it showed the presence of severe hemorrhagic gastritis overlapping with portal hypertensive gastropathy with absent peristaltic movements in the stomach (**Figures 2,3**).

The patient was treated with multiple prokinetic agents (metoclopramide, neostigmine, and erythromycin), intravenous proton pump inhibitor, sucralfate, and diuretics.

The patient failed to respond to the medical treatment and was intolerant of any amount of oral feed. So, the decision was made to proceed to fully transparent nutrition through a peripherally inserted central catheter (PICC).

Unfortunately, thirty days later, the patient died from multiorgan failure due to aspiration pneumonia and septic shock.

Discussion

PHG occurs in around 20-98% of cirrhotic patients. This high variability may be related to the study of different populations. In 2006, Fontana et al. conducted a study on 1016 cirrhotic patients and found the prevalence of PHG to be around 37%. (1) The incidence of PHG seemed to increase in patients with more advanced liver disease, patients with esophageal varices, or who have a history of prior managing varices by endoscopic band ligation or sclerotherapy. (2)

PHG is caused by both congestion and hyperemia of the stomach, as evidenced by increased gastric mucosal blood flow. (3) Other factors, such as mucosal ischemia and increased intrinsic oxide synthase activity, may also play a role in the development of PHG. (4)

PHG is usually asymptomatic. The most common symptom in symptomatic patients is bleeding from the friable gastric mucosa. Bleeding is mostly chronic and leads to chronic iron deficiency anemia, yet in rare instances, acute bleeding does occur. (5)

Many studies have suggested a relationship between liver cirrhosis, PHG gastroparesis, and delayed gastric emptying. In 2000, Güler et al. concluded that liver cirrhosis and PHG were associated with delayed gastric emptying. (6) Another study in Egypt by El Bokl et al. in 2001 also found a strong relation between liver cirrhosis and PHG and gastroparesis and delayed gastric emptying. (7)

The gold standard for diagnosing PHG is gastroduodenoscopy, which shows the stomach wall with a fine white reticular pattern intervening with areas of pinkish mucosa. (8)

POCUS uses ultrasound imaging devices by the attending physician at the patient's bedside. (9) The increasing usage as a diagnostic tool, especially in emergencies, is very helpful in situations where a formal radiological investigation would delay diagnosis and management. It also decreases the burden on radiologists, who are already overwhelmed by the demand for diagnostic and interventional radiological procedures. (10) An added benefit to incorporating POCUS in daily practice is the integration of imaging with history and clinical findings in one large picture. (11)

Even though POCUS is becoming more popular, there are no definite estimates of how much it is being used in daily practice among different institutes. (12) In 2016, a study by Mengel-Jorgnesen et al. showed a highly variable usage percentage among European countries, from 52% in France to less than 1% in Austria. (13) Another study, however, in China by Ahn et al. showed that around 82.7% of physicians who were involved in the POCUS survey used it in their daily practice. (14)

The use of POCUS traverses different fields and specialties. Cardiology has shown equal or even higher accuracy in diagnosing pulmonary edema by detecting B lines in chest ultrasound compared to pro-brain natriuretic peptide. (15,16) In pulmonology and during the coronavirus disease 2019 (COVID-19) outbreak, a study by Bitar et al. showed that POCUS played an important role in the bedside diagnosis, hemodynamic assessment, and management of patients with acute hypoxic respiratory and circulatory failure in COVID-19 pneumonia. (17) Another study by Bitar et al. showed that the number of subpleural consolidations correlated well with disease severity in COVID-19. (18)

POCUS is widely used in abdominal emergencies.

It can diagnose small bowel obstruction with abnormal peristalsis, dilatation, intraperitoneal free fluid, and small bowel edema. (19) Of course, it can also detect variable liver conditions, such as ascites. (20) In our case, the presence of a grossly dilated stomach with the presence of air bubbles and stagnant food particles denoted a form of gastric outflow obstruction; the presence of a markedly thickened gastric wall that signified wall edema (**Figure 1**) and the background history of decompensated liver cirrhosis and esophageal varices with multiple sessions of endoscopic band ligation. All these factors drove us to the early suspicion of portal hypertensive gastropathy as being the cause of gastroparesis in our patient, which was confirmed using the golden standard tool of gastroduodenoscopy (**Figures 2,3**).

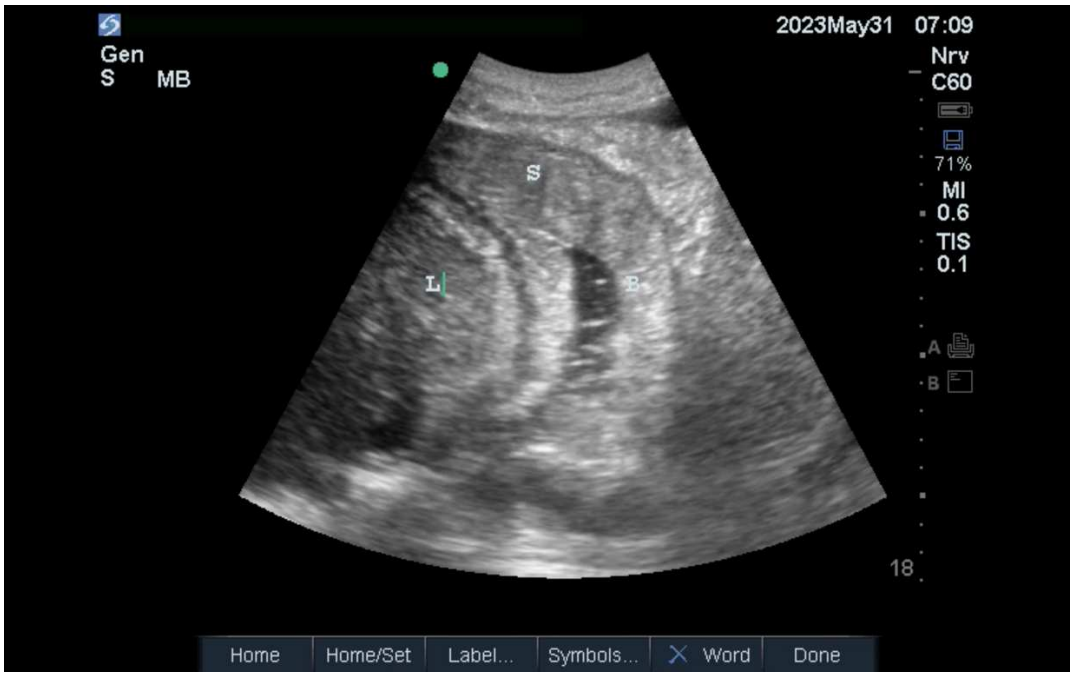
Conclusion

Point-of-care ultrasound could be used as an efficient tool for the early diagnosis of portal hypertensive gastropathy in patients with known liver cirrhosis presenting with a picture of gastroparesis.

Limitations

This is only a case report. Further studies should be undertaken to define the role of POCUS in diagnosing PHG clearly.

Figure 1. The first ultrasound scan done on admission shows the significant stomach (S) dilated with a markedly thickened wall with food particles and bubbles in the stomach cavity



Figures 2 and 3. Endoscopy images showing portal hypertensive gastropathy and markedly thickened mucosal folds

References

1. Fontana RJ, Sanyal AJ, Mehta S, Doherty MC, Neuschwander-Tetri BA, Everson GT, et al. Portal hypertensive gastropathy in chronic hepatitis C patients with bridging fibrosis and compensated cirrhosis: results from the HALT-C trial. Am J Gastroenterol 2006;101:983-92.
2. Merli M, Nicolini G, Angeloni S, Gentili F, Attili AF, Riggio O. The natural history of portal hypertensive gastropathy in patients with liver cirrhosis and mild portal hypertension. Am J Gastroenterol 2004;99:1959-65.
3. Ohta M, Hashizume M, Higashi H, Ueno K, Tomikawa M, Kishihara F, et al. Portal and gastric mucosal hemodynamics in cirrhotic patients with portal-hypertensive gastropathy. Hepatology 1994;20:1432-6.
4. El-Newihi HM, Kanji VK, Mihas AA. Activity of gastric mucosal nitric oxide synthase in portal hypertensive gastropathy. Am J Gastroenterol 1996;91:535-8.
5. Urrunaga NH, Rockey DC. Portal hypertensive gastropathy and colopathy. Clin Liver Dis 2014;18:389-406.
6. Güler I, Ortapamuk H, Üner E, Üstündağ Y, Köseoğlu T, Çetin F, et al. Evidence for gastroparesis in patients with portal gastropathy and liver cirrhosis and discussion of possible pathophysiologic theories. Turk J Gastroenterol 2000;11:277-82.
7. El Bokl M, Badawy HMM, El Maltawy MAF, Abdul Mageed KH, Ghali SM. Effect Of Portal Hypertensive Gastropathy On Electrogastrographic changes & Gastric Emptying Time. Egypt J Hosp Med 2001;3:107-25.
8. Wollenman CS, Chason R, Reisch JS, Rockey DC. Impact of ethnicity in upper gastrointestinal hemorrhage. J Clin Gastroenterol 2014;48:343-50.
9. Moore CL, Copel JA. Point-of-care ultrasonography. N Engl J Med 2011;364:749-57.
10. Zwank MD, Gordon BD, Truman SM. Refining the wild wild west of point-of-care ultrasound at an academic community hospital. J Am Coll Radiol 2017;14:1574-7.
11. Kwee TC, Kwee RM. Point-of-care ultrasound (POCUS): an opportunity for radiologists to improve patient care? Eur J Radiol 2021;139:109690.
12. Doniger SJ, Kornblith A. Point-of-Care ultrasound integrated into a staged diagnostic algorithm for pediatric appendicitis. Pediatr Emerg Care 2018;34:109-15.
13. Mengel-Jorgensen T, Jensen MB. Variation in the use of point-of-care ultrasound in general practice in various European countries. Results of a survey among experts. Eur J Gen Pract 2016;22:274-7.
14. Ahn C, Kim C, Kang BS, Choi HJ, Cho JH. Variation of availability and frequency of emergency physician-performed ultrasonography between adult and pediatric patients in the academic emergency department in Korea. Clin Exp Emerg Med 2015;2:16-23.
15. Prosen G, Klemen P, Strnad M, Grmec S. Combination of lung ultrasound (a comet-tail sign) and N-terminal pro-brain natriuretic peptide in differentiating acute heart failure from chronic obstructive pulmonary disease and asthma as cause of acute dyspnea in prehospital emergency setting. Crit Care 2011;15:R114.
16. Bitar Z, Maadarani O, Almerri K. Sonographic chest B-lines anticipate elevated B-type natriuretic peptide level, irrespective of ejection fraction. Ann Intensive Care 2015;5:56.
17. Bitar ZI, Shamsah M, Bamasood OM, Maadarani OS, Alfoudri H. Point-of-Care Ultrasound for COVID-19 Pneumonia Patients in the ICU. J Cardiovasc Imaging 2021;29:60-8.
18. Bitar ZI, Shamsah M, Maadarani OS, Bamasood OM, Bitar AZ, Alfoudri H. Lung Ultrasound and Sonographic Subpleural Consolidation in COVID-19 Pneumonia Correlate with Disease Severity. Crit Care Res Pract 2021;2021:6695033.
19. Becker BA, Lahham S, Gonzales MA, Nomura JT, Bui MK, Truong TA, et al. A prospective, multicenter evaluation of point-of-care ultrasound for small-bowel obstruction in the emergency department. Acad Emerg Med 2019;26:921-30.
20. Lindgaard K, Riisgaard L. Validation of ultrasound examinations performed by general practitioners. Scand J Prim Health Care 2017;35:256-61.