Open Journal of
Clinical & Medical
Case Reports

Volume 9 (2023)
Issue 26

Case Report ISSN: 2379-1039

Covert and fatal intra-gastric hemorrhage from a duodenal ulcer in a background of a cirrhotic liver and carcinoma of the gall bladder: A postmortem finding

Innocent Emmanuel*; Barka Vandi Kwaghe; Philip Ojile Akpa; Benjamin Otene; Samuel Abaniwo; Bankole Adedeji; Bamnan Collins Dallang; Patrick Nzekwe; Christiana Nwanneka Ibeanu; Othman Agahu; Nanisi Zorto Bahaushe; Jacob Anzaku Emmanuel; Ben James; David Nyam; Pantong Mark Davwar; Jummai Sa'a Winnie; Titus Nansok Fadok; Danjuma Salihu

***Corresponding Author: Innocent Emmanuel**
Department of Anatomic Pathology and Forensic Medicine, Jos University Teaching Hospital, and Department of Pathology, University of Jos, Nigeria.
Email: kinapele58@yahoo.com

Abstract

Mortality of patients with Peptic Ulcer Bleeding (PUB) and other Upper Gastrointestinal Bleeding (UGIB) in a background of liver cirrhosis is seen in clinical practice and the literature. This is a case of covert and fatal intragastric hemorrhage from a duodenal ulcer in a background of a cirrhotic liver and carcinoma of the gall bladder which was revealed after a postmortem examination. This report concludes that covert blood loss should be considered in patients with hypotension especially when this is sudden and rapidly evolving. Also, for patients with covert/overt UGIB, investigations for comorbidities and management of such could be life-saving.

Keywords: Peptic ulcer bleeding; Liver cirrhosis; Hemorrhagic shock; Mortality.

Introduction

Mortality of patients with Peptic Ulcer Bleeding (PUB) and other Upper Gastrointestinal Bleeding (UGIB) in a background of liver cirrhosis is seen in clinical practice and the literature [1-4]. In cirrhotic patients, UGIB is the deadliest complication [3]. Fifteen percent (15%) of these cirrhotic patients die within 6 weeks of symptoms [5]. It has been reported that over 70% of patients with PUB are asymptomatic though with severe decompensated cirrhosis [6]. This is a case of covert and fatal intragastric hemorrhage from a prepyloric ulcer in a background of a cirrhotic liver and carcinoma of the gall bladder which was revealed after a postmortem examination.

The Case

Clinical History

The patient presented with weakness, nausea, anorexia, weighed loss, bilateral leg and abdominal swellings, severe jaundice and pruritus, and severe generalized abdominal pain. He was a known chronic liver disease, hypertensive, and diabetic patient. On examination, he was fully conscious, chronically ill looking, afebrile, mildly pale, deeply icteric with blood pressure of 124/78 mmHg (Pulse rate 88 beats/ minutes) and normal heart sounds. He had moderate ascites with normal liver span and a tender right hypochondrium. The investigations showed markedly elevated liver enzymes, fasting blood sugar 6.8 mmol/litre (HBA1C 6.1). Abdominal ultrasound revealed a gall bladder mass of indeterminate nature.

A diagnosis of obstructive jaundice secondary to a gall bladder mass was made in a background of chronic liver disease (not cirrhosis), controlled diabetes and hypertension. He was placed on intravenous (IV) fluids 1 litre 8 hourly with IV B-complex vitamin 3 mils/litre of IV fluid; Slow I.V Plasil 10 mg tds; Tablets Spironolactone 25 mg b.d and Tablets Frusemide 40 mg daily; Intramuscular (I.M) Vitamin K 10 mg/ml daily; I.V Cefuroxime 750 mg tds; Tabs Silibinin140 mg tds; Tabs Esomeprazole 40 mg daily; Tab Gilclazide 30 mg dly; Tab DF-118 30 mg b.d. Four days on admission Cholestyramine 8 g tds was introduced and IV Plasil stopped.

He was place on strict monitoring of vital signs as well as that of input and output and prepared for exploratory laparotomy and cholecystectomy in 7days. He was showing marked improvement of symptoms and essentially stable. On the 5th day on admission, the patient suddenly developed severe weakness, restlessness and rapidly progressing hypotension that persisted and progressed to gasping despite active resuscitation. He was certified death afterwards.

Gross postmortem findings

The external appearance showed a middled age black man with severe conjunctival pallor, jaundice and bilateral pitting pedal edema. The internal organs on opening were in their normal anatomic position with no excess or abnormal fluid in third spaces. The heart is of normal weight (300 g) with enlarged left ventricular wall thickness (2.0 cm). The stomach appears pale and markedly distended. Cut open reveals about 1.5 liters of clotted blood and a gastric outlet obstruction (Figures 1 and 2). There is a pyloric ulcer with clotted blood at its base. The duodenal mucosa is thickened. The pale and enlarged liver weighed 2400 g with multiple cirrhotic micro and macro nodules on its surface (Figures 2, 3 and 4). Cut surface is gritty and shows a pale and greenish discoloration with nodularity. The gallbladder is enlarged and measures 8 x 4 x 1 cm, containing a soft irregular greyish white pedunculated mass attached to the wall and measuring 6 x 3 x 1.5 cm (Figures 3 and 4). The cut surface of the mass reveals a smooth brownish solid surface. The gall bladder mucosa is velvety and unremarkable. The extrahepatic biliary tree is patent and does not contain any calculi. The Right kidney and left kidneys weighed120 g each. The renal capsule strips with difficulty to reveals depressed cortical scars and fine granularity. Cut sections shows prominent corticomedullary differentiation. The lungs are sub crepitant to touch with the right lung weighing 400 g and the left weighing

500 g. The bronchi are of normal caliber. Cut sections of both lungs shows pale parenchyma, with cut bits and pieces of lung tissue floating on water. The brain weighed 1400 g with relatively narrowed sulci and widened gyri, and poor demarcation of grey and white matter on serial sections. All other tissues and organs were essentially normal.

Postmortem Histology

Prepyloric ulcer: Shows an ulcerated foveolar to villous type mucinous columnar mucosa overlying the submucosa, muscularis propria and serosa. Duodenal submucosal Brunner glands, marked hypertrophy of muscularis propria and mixed inflammatory cells infiltrates with lymphoid follicles are also seen. Histologic diagnosis, Ulcer Edge with Muscular Hypertrophy.

Liver: Shows nodules of hepatocytes separated by fibrous bands with Porto portal and Porto central pattern. Nests of neoplastic epithelial cells with abundant eosinophilic cytoplasm, vesicular nuclei, prominent nucleoli were seen. Moderate interface hepatitis, ductular reaction and necrosis are also seen. Histologic diagnosis, Metastatic Carcinoma in a background of Liver Cirrhosis (Grade 3, Stage 4) (Figure 5).

Gall bladder: Shows neoplastic epithelial cells disposed in sheets acini and cribriform pattern. These cells have abundant eosinophilic, vesicular nuclei, prominent nucleoli, they also exhibit pleomorphism and increased nucleocytoplasmic ratio. Also noted are Tumor giant cells, mixed inflammatory cell infiltrate, necrosis, lymphovascular and perineural invasion. Histologic diagnosis, Adenocarcinoma (Hepatoid Variant) (Figure 6).

Kidneys: Shows renal parenchyma with most of the tubules lined by coagulative necrotic epithelial cells with foci of detachment. Congested blood vessels and few mixed inflammatory cell infiltrates are also seen. Histologic diagnosis, Acute Tubular Necrosis.

Lungs: Shows alveolar duct and sacs separated by alveolar septa within which are seen dilated congested capillaries. Siderophages and alveolar macrophages are seen within alveolar space. Histologic diagnosis, Congested/Edematous lungs.

Brain: Shows a loose fibrillary background consisting of enlarged astrocytes. There are seen neurons with increasing eosinophilic cytoplasm, shrinkage of cell body, nuclear pyknosis and karyorrhesxis. Histologic diagnosis, Diffuse ischemic injury.

Cause of death: Hemorrhagic shock secondary to bleeding gastric ulcer with a background liver cirrhosis and metastatic gall bladder adenocarcinoma (1a. Hemorrhagic shock b. bleeding gastric ulcer. 2a. Gall bladder adenocarcinoma b. Liver cirrhosis with metastatic adenocarcinoma c. Hypertensive heart disease).

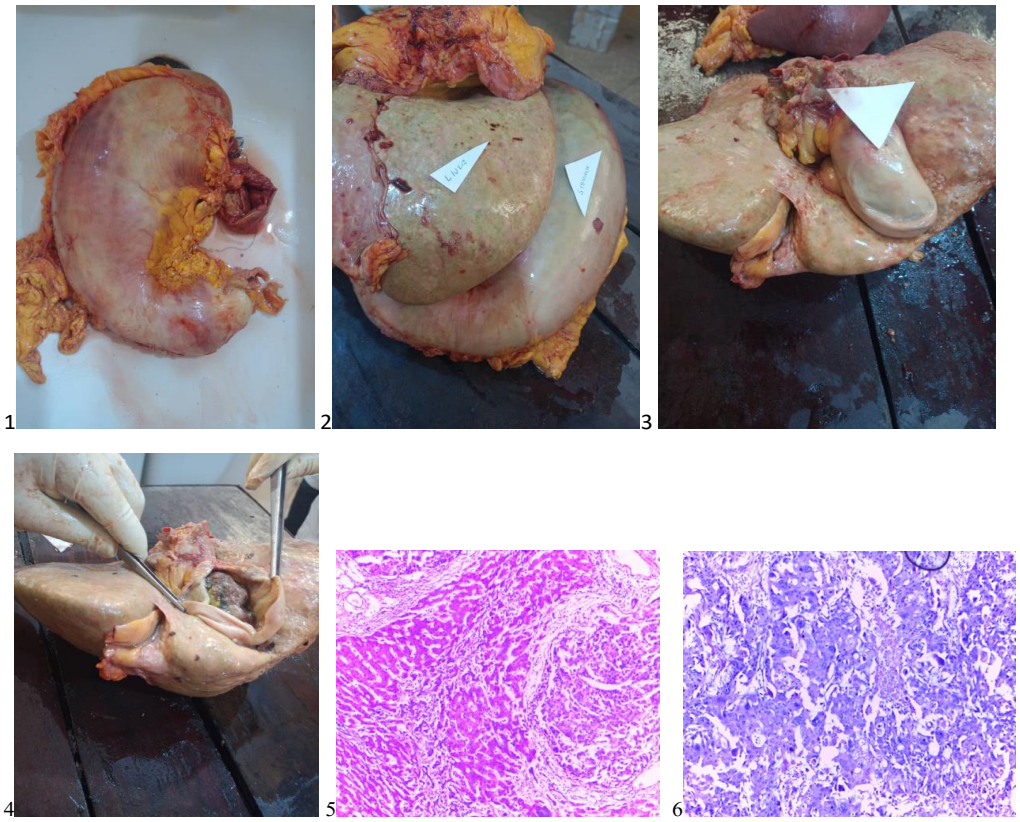

Figure 1: 1. Macroscopic image of stomach distended with clotted blood with a gastric outlet obstruction.
2. Macroscopic image of stomach as in figure 1, in continuity with a cirrhotic liver.
3. Macroscopic image of gall bladder distended with an enclosed mass in a background of a cirrhotic liver.
4. Macroscopic image of gall bladder distended as in figure 3, cut open to expose the in-situ mass in a background of a cirrhotic liver.
5. Histologic section of a cirrhotic liver showing broad bands encircling regenerating hepatocytes nodules. Metastatic malignant epithelial cells are seen in cell nest mostly within fibrous septae.
6. Histologic section of gall bladder adenocarcinoma showing malignant epithelial cells disposed in cell nests, and anastomosing trabeculae and cords.

Discussion

The association between peptic ulcer and cirrhosis has been reported. The index case falls within the subset of patients that succumbed to this association. Ardevol et al, in a study where 790 patients with UGIB were included, had 144 cases with peptic ulcer bleed and 646 cases with esophageal variceal bleeding. It was found that cirrhosis substantially increases mortality in both sets of patients [1]. After strict inclusion criteria, Yang et al mobilized 15,575 inpatients diagnosed and discharged with PUB of which 737 had cirrhosis and 1044 chronic hepatitis. These patients were carefully compared with a propensity-score matched controls at a ratio of 1:1 and subjected to a long term follow up [2]. Cirrhosis patients had the highest mortality (cirrhosis vs. matched controls: 24.15% *vs*. 19.16%, P=0.0225; chronic hepatitis vs. matched controls: 15.72% vs. 12.89%, P=0.0672) [2].

The patient in this study died of hemorrhagic shock although this is not the commonest cause of death amongst PUB patients with cirrhosis [3]. Lu et al sub-grouped 650 cirrhotic patients with endoscopically confirmed UGIB patients, into PUB (248 cases) and Esophageal and gastric variceal bleeding (402 cases) classes. Multiple systemic organ failure was the commonest cause of death in the former (50.00% of

12cases), while hemorrhagic shock in the later (69.97% of 20cases) [3].

One of the common and severe complication of peptic ulcer is bleeding.[3] In cirrhotic patients, bleeding is exacerbated by coagulation irregularities [2,3]. All the phases in coagulation which include: platelet plug initiation and formation; clotting process propagation by coagulation cascade; clotting termination by antithrombotic mechanisms; clot removal by fibrinolysis, are compromised in cirrhotic patients as the liver plays a critical role through its' inter-alia synthetic function to strike a balance between pro- and anti-coagulant factors [7].

The index case is a middle age man who falls within the age group of younger patients who die with peptic ulcer bleeding in a background of cirrhosis. Christensen et al concluded in a study that advancing age in perforated and bleeding peptic ulcer patients is a strong predictor of poor outcome independent of comorbidities [4]. The study reported rates of 4.3% and 33% respectively in 7,232 peptic ulcer patients aged younger than 65years and 80 years and above [4].

A significant finding in this patient was that he was relatively apparently asymptomatic of peptic ulcer, peptic ulcer bleeding, or cirrhosis and was not diagnosed or managed of these. The patient is therefore one amongst the over 2/3rd of patients that are asymptomatic of PUB despite a decompensated liver cirrhosis [6]. The absence of hemoptysis and hematochezia did not raise the suspicion of UGIB as the patient fatally and rapidly deteriorated with hypotension. The presence of a functional gastric outlet obstruction owing to hypertrophied tissues and for unknown reasons, the lack of vomiting masked the blood loss. Ardevol et al concluded in a background of current management protocols that for patients with PUB and variceal bleeding, only a small proportion die of uncontrolled hemorrhage, but rather due to comorbidities, hepatic decompensation, or bleeding complications [1].

Literature search for the coexistence of liver cirrhosis and gall bladder carcinoma did not yield positive results. Therefore, in the index case, it is difficult to relate a causal role of one on the pathogenesis of the other or to conclude a mere coincidence has occurred. These two pathologies develop gradually overtime [8-10]. Despite the near absent concurrent existence of these two pathologies in a patient, it will be worthwhile for future research to be conducted to seek for or exclude a casual role.

Conclusion

In conclusion, covert blood loss should be considered in patients with hypotension especially when this is sudden and rapidly evolving. Also, for patients with covert/overt UGIB, thorough investigations for comorbidities and management of such could be life-saving. Liver cirrhosis a common proximal comorbidity and distant pathologies such as gall bladder carcinoma should be considered in this regard.

Conflict of interest: Nil.

Consent: Informed consent of family was obtained.

References

1. Ardevol A, Ibañez-Sanz G, Profitos J, Aracil C, Castellvi JM, et al. Survival of patients with cirrhosis and acute peptic ulcer bleeding compared with variceal bleeding using current first-line therapies. Hepatology. 2018; 67: 1458-71.

2. Yang SC, Hsu CN, Liang C-M, Tai W-C, Wu C-K, et al. Risk of Rebleeding and Mortality in Cirrhotic Patients with Peptic Ulcer Bleeding: A 12-Year Nationwide Cohort Study. PLoS ONE. 2017; 12: e0168918.

3. Lu Z, Sun X, Han J, Jin B, Zhang W, et al. Characteristics of peptic ulcer bleeding in cirrhotic patients with esophageal and gastric varices. Sci Rep. 2020; 10: 20068.

4. Christensen S, Riis, A, Nørgaard M, Sørensen HT, Thomsen RW. Short-term mortality after perforated or bleeding peptic ulcer among elderly patients: a population-based cohort study. BMC Geriatr. 2007; 7: 1-8.

5. D'Amico G, De Franchis R. Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators. Hepatology. 2003; 38: 599-612.

6. Siringo S, Burroughs AK, Bolondi L, Muia A, Di Febo G, et al. Peptic ulcer and its course in cirrhosis: An endoscopic and clinical prospective study. J. Hepatol. 1995; 22: 633-41.

7. Muciño-Bermejo J, Carrillo-Esper R, Uribe M, Méndez-Sánchez N. Coagulation abnormalities in the cirrhotic patient. Ann Hepatol. 2013; 12: 713-24.

8. Hazeldine S, Hydes T, Sheron N. Alcoholic liver disease-the extent of the problem and what you can do about it. Clin Med (Lond). 2015; 15: 179-85.

9. Guan R, Lui HF. Treatment of hepatitis B in decompensated liver cirrhosis. Int J Hepatol. 2011: 918017.

10. Hundal R, Shaffer EA. Gallbladder cancer: Epidemiology and outcome. Clin Epidemiol. 2014; 6: 99-109.

Manuscript Information: Received: June 21, 2023; Accepted: August 04, 2023; Published: August 08, 2023

Authors Information: Innocent Emmanuel[1,2]*; Barka Vandi Kwaghe[1]; Philip Ojile Akpa[1,2]; Benjamin Otene[1]; Samuel Abaniwo[1]; Bankole Adedeji[1]; Bamnan Collins Dallang[1]; Patrick Nzekwe[1]; Christiana Nwanneka Ibeanu[1]; Othman Agahu[1]; Nanisi Zorto Bahaushe[1]; Jacob Anzaku Emmanuel[1]; Ben James[1]; David Nyam[3]; Pantong Mark Davwar[3]; Jummai Sa'a Winnie[4]; Titus Nansok Fadok[5]; Danjuma Salihu[6]

[1]Department of Anatomic Pathology and Forensic Medicine, Jos University Teaching Hospital, Nigeria.

[2]Department of Pathology, University of Jos, Nigeria.

[3]Department of Medicine, Jos University Teaching Hospital, Nigeria.

[4]Department of Nursing Science, Babcock University, Nigeria.

[5]Department of Histopathology, Federal Medical Center Jalingo, Nigeria.

[6]Department of Family Medicine, Jos University Teaching Hospital, Nigeria.

Citation: Emmanuel I, Kwaghe BV, Philip OA, Benjamin O, Samuel A, Adedeji B, Dallang BC, et al. Covert and fatal intra-gastric hemorrhage from a duodenal ulcer in a background of a cirrhotic liver and carcinoma of the gall bladder: A postmortem finding. Open J Clin Med Case Rep. 2023; 2087.

About the Journal: Open Journal of Clinical and Medical Case Reports is an international, open access, peer reviewed Journal focusing exclusively on case reports covering all areas of clinical & medical sciences.

Visit the journal website at www.jclinmedcasereports.com

For reprints and other information, contact info@jclinmedcasereports.com