Severe hypoalbuminemia and steatohepatitis leading to death in a young vegetarian female, 8 months after mini gastric bypass: A case report

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\textbf{A B S T R A C T}

\textbf{BACKGROUND:} Hypoalbuminemia is an important complication after Mini Gastric Bypass (MGB) and is more frequent in vegetarians, diabetic nephropathy, and alcoholic and liver disease patients. The patients must be followed in regular intervals and serum albumin must be checked in every visit after MGB. Hypoalbuminemia must be prevented by good protein regimes.

\textbf{CASE SUMMARY:} A 29 years old female was admitted 8 month after Laparoscopic Mini Gastric Bypass with malaise, dyspnea, icter, nausea, vomiting, diarrhea and edema of extremities from 2 weeks before admission. She had become vegetarian autonomously and had not participated in routine postop follow up, and also discontinued her high protein regimen. In para clinic test results, she had severe hypoalbuminemia, anemia, elevated liver enzymes and direct bilirubinemia, metabolic acidosis in Arterial Blood Gas (ABG), and in Core Needle Biopsy (CNB) marked Steatohepatitis was shown. Unfortunately, the patient did not respond to medical care and died.

\textbf{CONCLUSION:} Regular follow up after Mini Gastric Bypass is very important for many reasons as early diagnosis and treatment of hypoalbuminemia.

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1. Introduction

The Mini Gastric Bypass (MGB) was invented by Rutledge as an effective form of bariatric surgery. Previous studies have documented very good outcome and low complications in the MGB [1]. Severe nutrition complications such as hypoalbuminemia after Mini Gastric Bypass were poorly described. Hypoalbuminemia is a rare complication, especially after MGB and it usually responds to good nutritional support in the form of high-protein diet and conservative management [1,2].

One case series study suggests that MGB can expose the patients to risk of severe nutritional complications that need to vigorous nutrition care [2]. Some studies show that sometimes it may be necessary to reverse the MGB to correct the hypoalbuminemia [3].

We reported a case of severe hypoalbuminemia after MGB that was admitted in our academic hospital, based on Surgical Case Report (SCARE) Guidelines [4].

2. Presentation of case

The case of this study was a 29 years old Caucasian female that was brought into to emergency department by ambulance and had undergone Laparoscopic Mini Gastric Bypass with a 200 cm bil-pancreatic limb, 8 months before second admission, with preop BMI 55.7 and 55 kg EWL and had remission of diabetes mellitus. The patient had malaise, dyspnea, icter, nausea, vomiting, diarrhea and edema of extremities from 2 weeks before coming back to hospital without any intervention for her complains. She had not participated in routine postop follow up, discontinued her high protein regimen and had become vegetarian autonomously for more weight loss.

She had a history of hypothyroidism, hyperlipidemia and diabetes mellitus before LMBG. She had normal Thyroid Function Test (TFT), Liver Function Test (LFT) and albumin before bariatric surgery. In drug history, she was using tab pantoprazole...
Table 1

Patient’s some lab findings at admission.

| Lab Data | Value | Reference value |
|----------|-------|-----------------|
| Hb       | 7 g/dl| 12–16(f)        |
| WBC      | 8200/mm3| 4000–10,000     |
| Platelet | 195,000/mm3| 140,000–440,000 |
| SGOT(AST)| 128 IU/L| 5–40            |
| SGPT(ALT)| 22 IU/L| 5–40            |
| ALK.P    | 342 IU/L| 64–306          |
| Direct Bili| 4.8 mg/dl| 0–0.4           |
| Total Bili| 7.7 mg/dl| 0.2–1.2         |
| Albumin  | 2.3 g/dl| 3.5–5.5         |
| Total TSH| 30 nmol/L| 58–161         |
| TSH      | 22.1 mIU/L| 0.5–4.70       |

Hb: hemoglobin, WBC: white blood cell, SGOT: serum glutamic oxaloacetate transaminase, SGPT: serum glutamic-pyruvic transaminase, Alk.p: Alkaline phosphatase, T4: thyroxine, TSH: thyroid stimulating hormone.

400 mg/daily and tab Levothyroxine 100 mg/daily. She was non-smoker and had no family history of any specific genetic disorders. In physical examination, she was febrile (T: 38.1°C), PR: 110, BP: 100/85, RR: 20 and was pale and icterus and had mild abdominal tenderness and lower extremity pitting edema (2+), with normal size and non-tender thyroid.

In laboratory testing, she had normal BS (Blood Sugar), normal amylase and lipase, severe hypoalbuminemia, anemia, hypothyroidism, high serum ferritin and elevated liver enzymes, and direct bilirubinemia (Table 1). She had a metabolic acidosis in ABG (PH: 7.15, PCO2:44.1 mm Hg, PO2:47.9 mmHg, HCO3:20.5 mEq/L, BE:−8.5). Viral markers for hepatoviruses and ANA were negative. Urinary copper excretion and serum copper concentration were normal. Electrolytes were in normal ranges. In abdominal sonography, she had a course and high echo liver.

The patient underwent conservative management with high protein nutritional supplementation (high protein meal and serum aminofusion 500cc 10% IV/q12 h and vial albumin 20%/IV/q 12 h), Central Vein Pressure (CVP) monitoring, hepatobiliary, endocrine, hematology and cardiology consultation. A core needle liver biopsy was also done.

In CNB, pathologist reported marked steatohepatitis associated with intracellular cholestasis and multifocal lobular and periportal fibrosis (grade3/3 and stage2/4).

The LFT persisted high (SGOT:128 to 134 IU/L, SGPT:22 to 40 IU/L, and total bilirubin rose from total 7.7 to 16.8 mg/dl and direct bilirubin elevated from 4.8 to 12 mg/dl) and Albumin increased by TPN but not reached to normal levels (maximum to 3.4 mg/dl) in admission in 15 days.

Metabolic acidosis in patient was persistent and had only partial response to resuscitation and medical management.

For her anemia, after oncologic consult and analyzing the peripheral blood smear and no evidence of hemolysis, we performed the cross-matched packed cell transfusion to reach the Hb: 9.

The patient was candidate for liver transplant but in acute and severe course we tried to make the patient hemodynamically stable. Unfortunately, the patient got worsen after 2 weeks and did not respond to medical care and died.

3. Discussion

Mini Gastric Bypass is a good and effective bariatric and metabolic surgery that is recommended by IFSO (International Federation for Surgery of Obesity) and it has some complications such as hypoalbuminemia that is more reported in vegetarians, and alcoholic and nonalcoholic liver disease patients [1]. Steatohepatitis and hepatic failure are well known complications of jejunoileal bypass an abundant type of bariatric surgery and also are reported after biliopancreatic diversion [5]. Early recognition and appropriate management may prevent more serious complications. However, in some cases, the condition progresses to acute hepatic failure in patients with no previous liver disease [6].

Hypoalbuminemia can also take part in pathogenesis of NASH at initial stage. The possible mechanism can be portal pressure increasing which lead to reduction of small bowel absorptive capacity and progressing to malnutrition and hypoalbuminemia [7].

Hypoalbuminemia is classified as mild, moderate and severe. Mild hypoalbuminemia (2.5–3.5 g/dl) may occur along with fatigue and weakness. Severe hypoalbuminemia (<2.5 g/dl) may present with edema of ankle, with or without ascites. There is an increased incidence of mild hypoalbuminemia in MGB with limb length >230 cm [1].

Hypoalbuminemia usually responds to good nutritional support of high-protein diet [8], but in our case we had a decompensated patient that did not pass any regular follow up and had come so late with severe hypoalbuminemia and acute liver disease with direct bilirubinemia. We performed all available para clinic tests to diagnose, monitor the course of disease and rule out other differential diagnosis like as viral hepatitis, Wilson and other autoimmune liver disease, but we had not success to stabilize the patient hemodynamically for liver transplantation. In our case, we could not say that hypoalbuminemia is a cause for steatohepatitis and hepatic failure or it is result of steatohepatitis and hepatic failure. It is possible that hypoalbuminemia in vegetarians patients has worse outcome and more complicated situation with faster deterioration that we think that needs more studies. Hypoalbuminemia can be very dangerous with bad outcome, especially in vegetarian patients and must be prevented with precise and regular follow up by checking albumin, Liver function test (LFT), and ferritin every 3 month for first year, every 6 month for second year, and then annually for early diagnosis and treatment of hypoalbuminemia after Mini Gastric Bypass in order to have appropriate nutritional support.

4. Conclusion

It seems that vegetarian can aggravate hypoalbuminemia and liver failure after Mini Gastric Bypass and this group need more punctual follow up.

Conflict of interest statement

There is no competing interest in this article.

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Ethical approval

Ethics committee of the Minimally Invasive Surgery Research Center have approved the study by the code number: IR.IUMS.MISRC-101.REC.

Consent

The patient’s spouse signed an informed consent letting us to use her document information to write this case report.

Author contributions

Mohammad Kermansaravi: data collection, first drafting, final approval of the manuscript.
Mohammad Reza Abdolhosseini: idea, data collection, commenting on the manuscript, final approval of the manuscript.
Ali Kabir: idea, first drafting, final approval of the manuscript.
Abdolreza Pazouki: idea, commenting on the manuscript, final approval of the manuscript.

Guarantor
Mohammad Kermansaravi, Mohammad Reza Abdolhosseini, Ali Kabir, Abdolreza Pazouki.

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