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RESEARCH ARTICLE

CHYLOUS ASCITES MISTAKEN AS PEG TUBE LEAKAGE IN PATIENT WITH CREUTZFELDT-JAKOB DISEASE IN VEGETATIVE STATE, A DIFFICULT MANAGEMENT SITUATION

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Introduction:-

Chylous ascites is a rare condition characterized by an intra-abdominal accumulation of a milky-appearing fluid that contains high amount of triglycerides[1]. The incidence of chylous ascites is scarce with only one case reported in 20,000 cases admitted in a hospital[1]. To the best of our knowledge the literature is lacking any reported cases in adult from our country and that is due rarity of the condition and probably underreporting.

We report this case as it is the first case we have encountered in our hospital which is a large secondary/tertiary hospital over the last 15 years and because of difficulty in reaching diagnosis and difficulty in management.

Case Report:-

A 61-year-old Saudi male known case of diabetes mellitus, hypertension, Creutzfeldt - Jakob disease (diagnosed in USA in 2012) and dementia. He's bedridden on PEG tube and tracheostomy for the last 4 years. He was brought to the emergency department by his family with history of undetectable low BP by their home electronic BP meter. He also, had fever and loose watery stool for 2 days prior to admission.

He had no drug allergies. He was a non-smoker, non-alcoholic and has no history of liver or heart diseases.

He was admitted to ICU on Nov 15th as a case of sepsis/septic shock secondary to urinary tract infection/gastroenteritis, to Rule out MERS-CoV (Middle East respiratory syndrome Coronavirus) and H1N1.He developed acute kidney injury, respiratory failure put on mechanical ventilation. Later on, he developed Multi-organ dysfunction syndrome on the 27th of Nov which was managed accordingly with broad spectrum antibiotics and vasopressors. He improved gradually.

During the course of his illness the gastroenterologist was consulted for a possible leak from the PEG tube site. On examination the day of consultation, vitals were: Temp 36.5, Pulse 92, BP 123/49, RR 23 on mechanical ventilation through tracheostomy, O2 sat 97%. Generally, the patient was contracted, opening eyes but not responsive not communicating. His baseline GCS was 4/15 for the last 4 years.

No jaundice, pallor, or lymphadenopathy. Chest was clear bilateral vesicular breathing. Cardiovascular examination normal. No LL edema and no signs of DVT.

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Abdomen was Soft, lax and distended, with no scars or dilated veins. There was large ascites.

PEG tube Ex: The tube site was above and left to the umbilicus, there is erythema around it with greenish-yellowish discharge coming freely and with pressure. There was no pus. The PEG tube moves freely to the inside and out and to right and left.

Ascetic tapping was performed and 6 litters of ascetic fluid were drained initially, then the GI consultant was again contacted during the night by ICU. They thought that any feeding given to the patient through the PEG tube was coming out through the ascetic tube drain. The gastroenterologist came to assess the case and possibility of migration of the PEG tube to the peritoneal cavity .Gastrografin die given through the tube confirmed its presence in the stomach with no leak, also Methylene blue give through the tube did not appear in the ascetic fluid drain that support tube is intact with no leak.

Lab Test:-

Complete Blood Count (CBC):-
WBCs 9.79x103/μL, HGB 9 g/dL, Platelet Count 242x103/μL, MCV 79.5 fL, MCH 25.2 pg.

Basic Screen:-
NA 149 mmol/L, K 4.3 mmol/L, CL 120mmol/L, HCO3 19 mmol/L, BUN 13.4 mg/dL, CR 94 mmol//L, Random Glucose 15.4 mmol//L.

Renal Profile:-
GFR 76 mL/min, Adjusted Calcium 2.21 mmol/L, Phosphate 1.11 mg/dL, Magnesium .98 mEq/L, Uric Acid 317 μmol/L.

Liver Profile:-
Total Bilirubin 9.9 umol/L, Total Protein 42 g/L, Alkaline Phosphatase 82 IU/L, AST 15 IU/L, ALT 14 IU/L, ALB 27 g/L.

Coagulation Profile:-
PT 14.4 sec, INR 1.5, PTT 39.9 sec.

Coronary Risk Profile:-
Cholesterol 1.33 mmol/L, Triglyceride 0.70 mmol/L, HDL 0.35 mmol/L, LDL 0.57 mmol/L.

Ascetic Fluid:-
appearance was turbid, milky .The analysis showed RBCs 111x10, WBCs 3889x10, lymphocyte 2%, monocyte 3%, Segs 95%, LDH 647 U/L, Triglyceride 9.58 mmol/L (848mg /dl), albumin 18 g/L, total protein 35 g/L, amylase 42 U/L, cholesterol 1.17mmol/L and culture revealed no organisms.

Cytology report: Negative for malignant cells. TB PCR was negative. Adenosine deaminase was negative. TB culture negative.

Echo:Showed EF of 55-60%, RVSP 52 mmHg, normal biventricular systolic function, Mild LVH with impaired relaxation, trivial mitral regurgitation. Mild to moderate tricuspid regurgitation and trivialpericardial effusion.

Abdominal and pelvic CT scan showed no definitive contrast leakage at the gastrostomy site and no pneumoperitoneum. Bowel wall thickening involving part of the jejunum and the leftrecto-sigmoid area with mild abdominal ascites. In addition it showed small left pleural effusion and diffuse bony osteopenia.

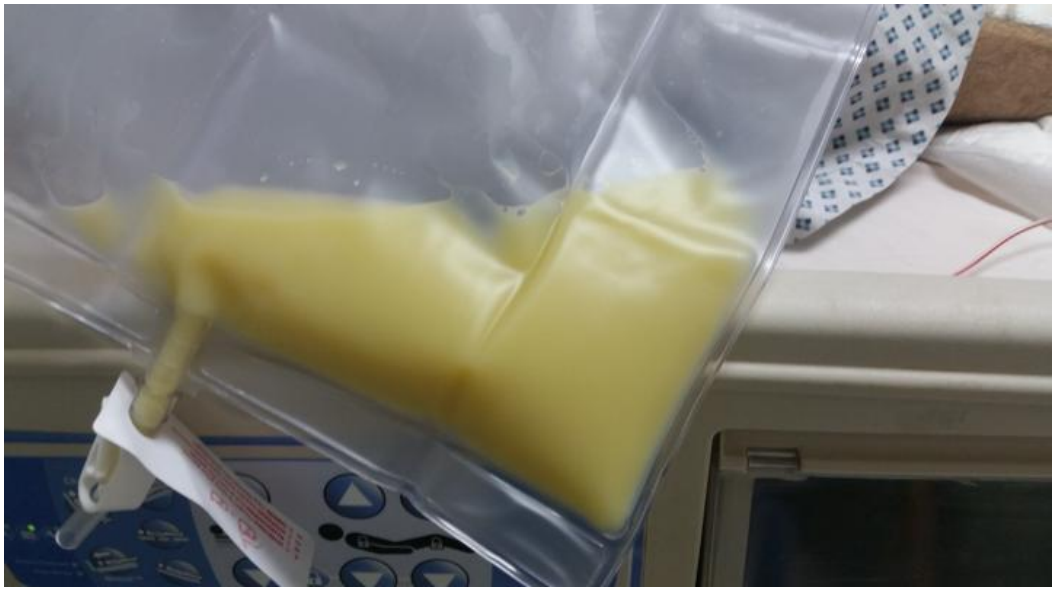
Colonoscopy: Showed large defuse ulcerations at the recto- sigmoid area.Rest of the colon was normal. Multiple biopsies and cultures taken.
The biopsies showed large bowel-type mucosa with benign ulceration, with no evidence ofgranulomas, dysplasia or malignancy.

With the above investigations there's no clear identified cause for the chylous ascites. It was decided to perform a

diagnostic laparoscopy and peritoneal biopsy to Rule out tuberculosis and malignancy but because of the patient's general condition and vegetative state the surgeon and family did not agree for the procedure. The patient was managed conservatively and started on octreotide 100 mcg subQ every 8 hrs,plus trail of Orlistat and medium chain fatty acid diet with repeated paracentesis in case of increase abdominal distension. It was noticed that re - accumulation of fluid is becoming less.

Patient stayed in the ICU for 6 weeks receiving broad spectrum antibiotic and supportive care.

He improved gradually, weaned of ventilator and kept on O2 through tracheostomy tube. His general condition stabilized and return to the base line status transferred to step down ward. Finally he was taken home to be cared-of by his special nurse with tracheostomy and O2,PEG tube and Foley catheter to be followed by home health care team accordingly.



Discussion:-

Chylous ascites is a rare condition defined as an intra-abdominal accumulation of a milky-appearing fluid[1]. It was first identified by Morton in 1691[2]. Thereafter the first acute case was admitted by Renner in 1910[3].The characteristic feature of chylous ascites is the presence of high fat (triglyceride) content in the ascetic fluid, usually greater than 110 mg/dl[1] . The incidence of chylous ascites is scarce with only one case reported in 20,000 cases admitted in a hospital[1]. However, the incidence of this condition is increasing with time due to prolonged lifespan of patient with malignancies and due to vigorous cardiothoracic and abdominal procedures[2].Regardless, there have not been many cases reported until this given day.

From our country there has been no reported case in adult. We have seen one case in patient with liver cirrhosis (not reported). There has been one report of infantile intestinal lymphangiectasia and chylousascites[4]. We think the discovered cases are underreported.

There are a wide variety of conditions that can lead to chylous ascites. Causes of chylous ascites can be categorized into two main groups traumatic and non-traumatic. Traumatic causes include surgically-induced chylous ascites for example, retroperitoneal lymphadenectomy, preritoneal dialysis catheter placement, Nissen fundoplication, and pancreaticoduodenectomy[5]. Abdominal aortic aneurysm repair procedure accounts for more than 80% of the post-operative Chylous ascites cases[6]. And non-surgically induced chylous ascites like in case of radiotherapy[7].
Non-traumatic causes comprise some congenital conditions like Kippel-Trenaunay syndrome, primary lymphatic hypoplasia and lymphangioma[8]. According to a recent reported case, appendicitis can be a cause for the development of Chylousascites[9].

Liver diseases like cirrhosis can frequently give rise to chylous ascites. Furthermore, cardiac diseases like congestive heart failure, gastrointestinal conditions like small bowel volvulus, intestinal malrotation and celiac disease can contribute to chylous ascites. Tuberculosis and filariasis are the commonest infectious causes. Inflammatory disorders like pancreatitis, fibrosingmesenteritis, sarcoidosis, systemic lupus erythematosus and nephrotic syndrome can induce chylous ascites as well[8].

According to a recent study, abdominal malignancies, liver cirrhosis and tuberculosis are the most common encountered contributors for this condition[8].

Our patient's clinical condition did not allow extensive investigations partly due to patient's factors since he has poor general condition, bedridden in vegetative state for 4 years and partly due to family issues as they don't like to expose the patient to any more invasive procedures like laparoscopy and lymphangiography. However the patient has almost satisfactory investigations to rule out cardiac, liver, malignant or infectious causes. There has been no history of trauma since he did not undergo any invasive procedure.

Filariasis is very rare in our region and there hasn't been any cases reported. Patient didn't have any risk factors to get filariasis.

People with this condition present with progressive, painless abdominal distention which occurs over weeks to months, according to the primary cause. A non-specific pain could be the only presenting complaint[8]. Ascites can cause dyspnea owing to the high intra-abdominal pressure. Other clinical features include edema, weight gain, anorexia, weakness, lymphadenopathy, night sweats, early satiety, fever and other characteristic of the underlying disease. Physical examination could reveal ascites with positive fluid wave and/or shifting dullness, pleural effusions, lower extremity edema, lymphadenopathy, cachexia, temporal wasting, abdominal masses, hernias and other stigmata of the underlying illness[1,10].
Since our patient was in vegetative state and bed ridden, the ascites was noticed late when it became large.

The diagnosis is mainly based on the analysis of ascetic fluid and paracentesis. Chylous ascites is characterized by its odorless milk-like appearance, alkali bacteriostatic nature, specific gravity of more than (1.012), sterile culture and resisting putrefaction, positive fat staining and ether test[11]. The serum to ascites albumin gradient should be calculated to determine if the ascites is related to portal hypertension or other causes[12].The triglycerides level in chyle is greater than that in plasma, typically 2-8 times the plasmalevel[13].

Table 1:-Characteristics of Chyle

Chyle content	2.5-7 g/dl
Triglycerides	>200 mg/dl
Cholesterol	Ascites: serum <1
Total solids	>4%
Cell count	>500 (predominantly lymphocytes)
SAAG	<1.1

SAAG: Serum ascetic fluid gradient

Gram staining, lactate dehydrogenase, amylase, lipase cytology, culture, glucose and ascetic fluid for cell count should be performed as well[3,14].

In addition Adenosine deaminase activity (ADA), culture and tuberculosis smear when Tuberculosis is suspected[15].
In our case the tuberculosis PCR, culture and adenosine deaminase all were negative that make abdominal tuberculosis unlikely .Cytology was also negative in two occasions.

The initial ascetic tap result with high neutrophils count suggested superadded acute bacterial peritonitis which has been treated. The culture was negative because the patient had been on broad spectrum antibiotics prior to the tapping.

Other diagnostic tools include laparotomy, lymphangiography, lymphoscintigraphy and computed tomography[1].
In our case we were unable to procced for invasive diagnostic procedures because of the patient's clinical condition, in addition the surgeon and family reservations.

Looking at the major causes of chylous ascites we think that our patient has idiopathic chylous ascites since no obvious cause could be identified after doing most of the necessary investigations.

The main stay of therapy is treating the underlying cause, especially in infectious, malignant or inflammatory causes. Regardless,conservative management is usually effective for most patients. It consists of stopping the patient's oral nutrition, administering somatostatin analog, diuretic and diet therapy with medium-chain triglycerides (MCTs)and administration of total parenteralnutrition (TPN), alone or in combination[16]. These measures are directed to decrease chyle formation and enhance nutritional states[17]. Paracentesis is both diagnostic and therapeutic method in chylous ascites management. In the face of various certain disadvantages and complications, among the non-operative management plan, a frequently done paracentesis is routinely included to lessen abdominal distention[18]. Urgent exploration is compulsory in some acute presentations[19]. Peritone-venous shunting could be the ultimate choice if the case is unqualified for surgery and not responding to non-operative options[20].

In the present case a definite cause has not been identified therefore we opted to treat him conservatively, initially he has been tried on octreotide and Orlistat .Later on medium chain fatty acid diet was introduced .He showed some improvement but upon transfer from ICU to step down the medications were mistakenly not continued therefore, the ascetic fluid recollected. However the initial effect has been satisfactory. The actual effect of the treatment will need to be observed and follow up for a while.

Summery:-

We are reporting a rare case of chylous ascites that had initially been mistaken as a leakage of PEG tube into the peritoneum. There was no identifiable cause in this case after performing almost most possible investigations thatcould make it idiopathic.

This casedemonstratesthatin occasions there are uncontrollable factors which lead to major limitations in reaching the diagnosis and completing ideal management for such cases.

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