cost effective. Therefore, it is important to identify accompanying additional risk factors which assist in selection of a high-risk group for further investigation. We also confirmed that higher AST and CAP values, lower platelet, and hypertension were independently factors for increased liver stiffness in our T2DM patient. Beside there are limitations of our demonstration, it includes a cross-sectional nature and absence of histological data using Transient Elastography (TE, Fibro Scan), a simple and fast modality, to evaluate hepatic steatosis and fibrosis in this cross-sectional report. But this single TE result in our cross-sectional report may not be a true reflection of patient's fibrosis status as it can be confounded by several factors such as obesity, alcohol/food consumption, or patient dependence. Nevertheless, TE has been endorsed as an alternative to liver biopsy by international guidelines in guiding clinical management of chronic liver disease, including NAFLD. Therefore, TE may be a valid and accurate modality in assessing NAFLD fibrosis in a community-based population where liver biopsy is not practical for all.

Conclusion

NAFLD was highly prevalent in our T2DM patients. In particular, patients with higher AST and CAP values, lower platelet count or comorbidity of hypertension have higher risk for increased liver stiffness and should be considered for further assessment. The clinical burden from NAFLD-related complications is expected to be considerable, because T2DM is known to be an accelerating factor for NAFLD progression and associated with increased mortality. NAFLD is largely an asymptomatic condition and only manifests at late stage of liver disease. Indeed, the general population may not be familiar with the risks associated with NAFLD, particularly in disabled subjects. In addition, the perceived lack of treatment for NAFLD by the patients may add a further barrier to timely diagnosis and intervention. Consequently, improving public awareness of NAFLD and education of risk recognition and pre-empirical treatment in selective populations such as T2DM patients may alleviate the onslaught of the NAFLD epidemic.

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PSEUDOMEMBRANOUS COLITIS

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Relevance

Pseudomembranous colitis is an inflammatory condition of the colon characterized by elevated yellow-white plaques that coalesce to form pseudomembranes on the mucosa, inflammation (swelling, irritation) of the large intestine. Patients with the condition commonly present with abdominal pain, diarrhea, fever, and leukocytosis. In many cases, it occurs after taking antibiotics. Using antibiotics can cause the bacterium *Clostridium difficile* (*C. difficile*) to grow and infect the lining of the intestine, which produces the inflammation. Certain antibiotics, like penicillin, clindamycin, the cephalosporins and the fluoroquinolones, make *C. difficile* overgrowth more likely because pseudomembranous colitis is often associated with

C. difficile infection, stool testing and empiric antibiotic treatment should be initiated when suspected. When results of *C. difficile* testing are negative and symptoms persist despite escalating empiric treatment, early gastroenterology consultation and lower endoscopy would be the next step in the appropriate clinical setting. If pseudomembranous colitis is confirmed endoscopically, colonic biopsies should be obtained, as histology can offer helpful clues to the underlying diagnosis. The less common non- *C. difficile* causes of pseudomembranous colitis should be entertained, as a number of etiologies can result in this condition. Examples include Behcet's disease, collagenous colitis, inflammatory bowel disease, ischemic colitis, other infections organisms (e.g. bacteria, parasites, viruses), and a handful of drugs and toxins. Pinpointing the correct underlying etiology would better direct patient care and disease management. Surgical specialists and gastroenterologists would be most helpful in colonic perforation, gangrenous colon, or severe disease [1, 3, 4].

Purpose of the study

To identify the etiology pseudomembranous colitis, review the evaluation of a patient with pseudomembranous colitis, summarize the treatment and management options available for pseudomembranous colitis and describe interprofessional team strategies for improving care coordination and outcomes in patients with pseudomembranous colitis.

Material and methods

The literature, case reports and statistical data on recent studies were analyzed.

Results and discussion

Epidemiology: *C. difficile* infections have increased over the last 20 years with almost 500,000 episodes and 29,000 associated deaths reported annually in the United States, which makes it among the most common nosocomial infections. *C. difficile* colitis in patients without antibiotic and healthcare exposure has led to the recognition of community-associated *C difficile* infection. The most common cause of infectious diarrhea in healthcare settings, *C. difficile* causes substantial morbidity and can potentially result in mortality in vulnerable inpatient populations. *C. difficile* colonization occurs in 13 % of hospitalized patients with stays of 2 weeks and up to 50 % of patients with stays more than 4 weeks. *C. difficile* can colonize the human colon; 2 to 5 % of the healthy outpatient community will be colonized and will not manifest signs of infection. [6] Among those who are admitted to hospital, it occurs in between 4–8 people per 1,000 according to recent studies [7].

Pathophysiology: The administration of antibiotics, chemotherapeutic drugs, or immunosuppressive therapy disrupts the normal colonic biome, which allows for *C. difficile* colonization. *C. difficile* induces colitis via exotoxin production, toxin A, and toxin B. These toxins generate inflammation, colonic cell cytoskeleton disruption, and cellular death. The pseudomembranes of pseudomembranous colitis form as these toxins pathologically hyperstimulate the native immune system by drawing neutrophils to invade the colonic mucosa. The complications include: Dehydration: Severe diarrhea can lead to a significant loss of fluids and electrolytes. This condition can cause blood pressure to drop to dangerously low levels, Kidney failure: In some cases, dehydration can occur so quickly that kidney function rapidly deteriorates (kidney failure). Toxic megacolon: In this rare condition, colon is unable to expel gas and stool, causing it to become greatly distended (megacolon). Left untreated, colon may rupture, causing bacteria from the colon to enter the abdominal cavity. An enlarged or ruptured colon requires emergency surgery and this may be fatal. Bowel perforation: This is rare and results from extensive damage to the lining of the large intestine or after toxic megacolon. A perforated bowel can spill bacteria from the intestine into abdominal cavity, leading to a life-threatening infection (peritonitis) [2, 5, 8].

Case report

A 35-year-old woman admitted to hospital due to fever, abdominal pain, diarrhea after extraction of teeth 2 weeks ago and using single dose amoxicillin 500 mg

for prevention infective endocarditis [9, 10]. She had tenderness to palpation in the bilateral lower quadrants of abdomen, right greater than left. Her white blood cell (WBC) count was 25.6 × 10³ cells/µL. Ultrasonography of the abdomen and pelvis showed marked edema and inflammation of the cecum and ascending colon as well as an enlarged appendix with surrounding inflammatory changes with a small amount of free fluid in the right paracolic gutter. *C. difficile* stool polymerase chain reaction was positive. Oral vancomycin was started. The patient markedly improved with medical *management alone and was subsequently discharged on oral vancomycin.*

Diagnosis

Complete blood count (CBC): The white blood cell count is markedly high in case of *Clostridium difficile* infection.

Initiate stool testing for *C. difficile* when patients have positive guaiac diarrhea in combination with radiographic evidence of toxic megacolon or the presence of *C. difficile* risk factors such as hospitalization, antibiotic or chemotherapy exposure, and marked leukemoid reactions. Screening for *C. difficile* as the causative agent for pseudomembranous colitis begins with screening for the *C. difficile* toxins.

A common lab algorithm utilizes an enzyme immunoassay (EIA) to screen for glutamate dehydrogenase antigen, present in most *C. difficile* isolates followed by EIA sampling for *C. difficile* toxin A and toxin B.

Nucleic acid amplification tests, PCR (polymerase chain reaction), loop-mediated isothermal amplification for *C. difficile* have excellent sensitivity and specificity with high positive likelihood ratios; utilization of these assays aids in clarifying discrepancies between glutamate dehydrogenase and EIA toxin results. Nucleic acid amplification tests results should be interpreted with caution because they do not distinguish *C. difficile* colonization from active infection unless testing for concurrent toxin production [2].

CT scan: CT scan of the abdomen shows thickening in the intestinal wall indicating inflammation.

Treatment

Multiple studies document the effect of metronidazole with oral vancomycin for the initial treatment of mild to moderate *C. difficile* colitis. Because of superior cure rates for severe disease, administer oral vancomycin 1000–2000mg per day in 4 equal doses (every 6 hours) with Metronidazole 500 mg for 10 days as first-line treatment of *C. difficile* colitis. With recurrent *C. difficile* colitis after oral vancomycin therapy, consider either fidaxomicin or rifaximin oral administration. For fulminant *C. difficile* pseudomembranous colitis refractory to pharmacological therapy or with complications such as megacolon or colonic perforation, surgical intervention may be warranted, including hemicolectomy. Recurrent *C. difficile* colitis commonly occurs following initial pharmacologic therapy; usually, the initial antibiotic therapy is reinstituted unless evidence exists of worsening disease severity. With three or more disease recurrences despite therapy with oral vancomycin, fecal microbiota transplant has shown benefit in small trials.

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