

A Compendious Account on the Significance of Gallstone Ileus in Amyotrophic Lateral Sclerosis

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Received 09 June 2021; **Accepted** 23 June 2021; **Published** 30 June 2021

Abstract

Gallstone Ileus (GI) is an uncommon pathology generally affecting elderly females with a history of untreated cholelithiasis. The development of GI is a sequela from a fistulous connection resulting from a chronic inflammatory state between the gallbladder and intestinal tract; most commonly the second (descending) segment of the duodenum. However, individuals suffering from a neurodegenerative disorder, such as Amyotrophic Lateral Sclerosis (ALS) causing autonomic dysfunction with resultant gut dysmotility and predisposition to gallstone formation may be at a significantly higher risk of morbidity in this patient population in regards to the development of GI.

Keywords: Gallstoneileus • Amyotrophic Lateral Sclerosis (ALS) • Dysautonomia

Description

GI cases are rare incidences, occurring in less than 1% of patients diagnosed with cholelithiasis. The term "ileus" is inaccurate to the gross pathology of the disease; because it is merely an obstruction in the gastro-intestinal tract (usually occurring at the terminal ileum), rather than a termination of

motility. The abnormal connection between the gallbladder and adjacent surrounding structures is most commonly a cholecystoduodenal fistula, however, fistulous connections have occurred with the colon, stomach, and biliary tree. The chronic inflammatory state that predisposes many patients to the development of GI can be a result of chronic or untreated inflammatory bowel disease, perforation from endoscopic procedures, and failure to retrieve intra-abdominal gallstones from a cholecystectomy. ALS is also an uncommon diagnosis in patients complaining of symptoms related to motor dysfunction. It is a rapidly progressive disorder that classically presents as muscular weakness with preservation of one's cognitive ability. While ALS typically targets neuronal cells of the anterior horn and corticospinal tracts, clinical symptoms related to dysautonomia have been reported; in particular affecting gastrointestinal homeostasis. The development of ALS is believed to be associated with several genetic and environmental factors; although the pathophysiology still remains quite unclear. However, ALS has been previously documented to reduce gastro-intestinal peristalsis. This lack of normal peristaltic function may result in sub-optimal gallbladder contractility; predisposing patients to biliary stasis and sludge formation. Furthermore, a hyper-stimulatory state of the adrenergic nervous tissue may contribute to hypo-perfusion to the viscera of internal organs leading to irregular contractile force of the gallbladder resulting in stasis, sludge, and gallstone formation. A significant reduction in gastric emptying and colonic transit times in patients suffering from ALS has been identified when compared against healthy age-matched controlled. When the lack of adequate gastric emptying and intestinal movement is taken into account, it greatly increases the risk of obstruction related to GI. It is important to consider and determine the degree of progression of one's neurodegenerative state when assessing patients with known ALS complaining of gastro-intestinal symptoms.

Conclusion

A multifactorial workup should always be completed when evaluating this patient population; taking into account the nutritional status/fiber content in the diet, age-related deconditioning/inactivity, surgical and past medical history. It is certainly relevant to consider a causal relationship between GI and ALS. Early and accurate identification of cholelithiasis with appropriate medical and surgical intervention is imperative in this patient population to prevent significant morbidity and mortality if left untreated. In the event of GI, the treatment options remain surgical intervention with either a one-stage procedure performing an enterolithotomy, cholecystectomy and fistula closure or a two-stage procedure with an initial enterolithotomy followed by a delayed cholecystectomy.