Clinics in Surgery

Editorial
Published: 01 Nov, 2018

Long Term Effects of Endoscopic Sclerotherapy on the Oesophagus

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Editorial

Portal hypertension and its secondary effects significantly contribute to the morbidity and mortality in patients with advanced liver disease [1]. Due to a progressive rise in portal pressure, peri-esophageal portal- systemic circulatory pathways open up as a means to establish collateral circulation, which is essentially an attempt by the body to "shunt" portal blood which is at a higher pressure, into the systemic circulation in the region, which is at a lower pressure. This collateral portal-systemic circulation is represented by distal esophageal varices [2]. The oesophageal varices can be rapidly fatal from their ability to bleed. Over the years several modalities have been used to control the bleeding by decompression of the portal venous system by mesocaval or other shunts or by variceal sclerotherapy or ligation etc. Transjugular Intrahepatic Portal Systemic Shunting (TIPS) has been used but this treatment is plagued by shunt dysfunction arising in up to 50% of patients when bare metal stents are used and somewhat lower when covered stent graft is used [3]. However, use of small calibre TIPS may offer clinical benefits while at the same time delay the development of encephalopathy. Liver transplantation has also been used as a treatment option in selected patients. In a clinical situation however, where one is confronted with a severely ill patient who is bleeding from oesophageal varices and is unsuitable for anaesthetic and surgical intervention, endoscopic variceal ligation and Endoscopic Sclerotherapy (EST) can save the patient's life. Currently, endoscopic sclerotherapy should be reserved for high risk patients as this saves lives. There is a huge clinical experience in the use of EST in the treatment of oesophageal varices. Endoscopic sclerotherapy however has by far been the mainstay of management of these patients for many decades. Many of us, who have seen these patients for years, have also noticed the severe aftereffects of EST. Although several groups have earlier conducted manometric studies on the oesophagus following EST, our group conducted one of the first studies attempting to correlate histologic changes with manometric studies in patients who had been treated with EST earlier [4]. We shall review the current status of our knowledge in developing an understanding of the correlation between histologic changes and manometric abnormalities following EST.

Portal-systemic collaterals are predominantly intramucosal and in the submucosa of the distal oesophagus, gastroesophageal junction and even the gastric fundus [5,6]. Once the portal venous pressure surpasses 10 mmHg, predisposition to development of portal-systemic collaterals (varices), is high. However, vulnerability of varices to cause life threatening bleeding invariably exists at 12 mmHg and over. Variceal bleeding however is dependent upon location, size etc. Bleeding from variceal rupture combined with ascites, jaundice, deteriorating hepatic metabolic function and encephalopathy contribute to the significant morbidity and mortality of these very ill patients.

Management of acute variceal bleeding should be prioritized and if a risk group has been identified then prevention of bleeding by for example adjuvant therapy with Non-Selective Beta Blockers (NSBB), renin- angiotensin system inhibitors, statins etc. should be considered [8-10]. In acute bleeding however, small calibre TIPS can be useful as noted earlier, however its availability may be the limiting factor. The "turning off" of the bleeders endoscopically by ligation and/or sclerotherapy can save lives and helps buy time for more substantive measures like meso-caval shunting or liver transplantation etc. in the most severe cases. Often on entering the oesophagus with a forward looking scope, one can't see anything due to clots and altered and fresh blood. Meticulous evacuation of clots and other material is critical in establishing a clear enough view to endoscopically identify and sclerose the bleeding varices. It is essential that this is done by surgeons or in the presence of surgeons in order to develop an understanding of the clinical situation and reduce the transit to an operative option should all else fail. The use of NSBB in the prophylactic treatment of these "bleeders" to prevent rebleeds is beyond the scope of this paper. The patients who have been treated successfully with EST and go on to have several sessions and finally go into

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Received Date: *26 Sep 2018*
Accepted Date: *30 Oct 2018*
Published Date: *01 Nov 2018*

Citation:
Sharma PP, Sharma DK. Long Term Effects of Endoscopic Sclerotherapy on the Oesophagus. Clin Surg. 2018; 3: 2194.

remission or a compensated state, report the symptoms characteristic of the post- EST period.

In our original work published earlier, it was found that the severity of manometric and histologic changes could be correlated to the severity of esophageal dysmotility but was unrelated to the total amount of sclerosant used in any patient [4]. The severity of histologic and manometric changes was related to the number of sclerotherapy sessions that the patients had had. In fact, it would appear that the manometric changes after EST are not caused by the sclerosant itself. This is supported by the fact that the dysmotility and the symptoms attributable to it, do not appear in the immediate post-EST period. These appear several months after the repeated EST and in patients who have become asymptomatic from their original condition, whether by EST alone or some other definitive treatment. It is therefore reasonable to hypothesise that the chronic inflammatory changes seen in the distal oesophagus after repeated EST are the trigger for the dysmotility and may well lend themselves to treatment once we understand the correlation better. In similar study, Ghosal et al. [7] found that Lower Oesophageal Sphincter (LES) pressure was reduced in the post- sclerotherapy group compared to the pre-sclerotherapy group [7]. Their study was conducted using absolute alcohol while most of our work has been done using sclerosants like polidocanol. Their study concluded that oesophageal dysmotility is commoner after sclerotherapy. However, there was no correlation with histologic changes or any attempt to explain the changes if any. There have been no other significant studies on the correlation between histologic and manometric changes after repeated EST since our paper was published and opened up the new field which established a basis for the manometric changes observable after repeated EST and the accompanying histologic changes. It is our hope that further studies will be done with more patients to study this in greater detail. This will help us to devise treatment protocols for management of often severe oesophageal symptoms of chest pain, oesophagitis, dysphagia and even stricture formation by treating the cause rather than instituting symptomatic treatment.

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