ZOLLINGER-ELLISON SYNDROME AN OVERVIEW

Arati Machale[1*], Ajay sable[1], Ashwini Andhale[1], Santosh Waghmare[1], Hemant Kamble[1].

1. Loknete Shree Dadapatil pharate College of Pharmacy, Mandavgan Pharata, Shirur, Pune, 412211

Corresponding Author-
Author Name – Arati Machale

Address- Loknete Shree Dadapatil pharate College of Pharmacy, Mandavgan Pharata, Shirur, Pune, 412211

Abstract-
Zollinger and Ellison (1955) described a syndrome of recurrent gastric ulceration and gastric hypersecretion associated with pancreatic islet cell tumors. For many years, doctors and surgeons have agreed that a total gastrectomy is the best treatment. Cimetidine, a histamine H2 receptor antagonist, inhibits the basal stimulation of gastric acid secretion and promotes the healing of gastric and duodenal ulcers. ZES is a rare cause of gastric ulcers, accounting for only 0.1-1% of ulcers. . A perforated gastric ulcer was suspected and urgent surgical intervention was required. A tumor lesion in the head of the pancreas was then identified and repaired using the Whipple technique. Pathological results showed tumors indicative of neuroendocrine tumors. The incidence of ZES in the United States is 1-3 new cases per million people per year, making it a rare condition. The most common symptom of ZES is pelvic pain associated with peptic ulcer disease. More than 90% of gastrinoma patients develop upper gastrointestinal ulcers during the course of the disease. Similar to typical idiopathic ulcer disease, ZES is usually characterized by discrete ulcers, 75% of which occur in the first segment of the duodenum. Patients with ZES may also have recurrent, multiple, and atypical focal ulcers. In this surgery, a special endoscope called an endoscope uses ultrasound to treat the pancreas. The secretin stimulation test is the provocative test of choice, can diagnose ZES because it is sensitive, easy to use, and has no negative side effects.

Keywords- Zollinger-Ellison syndrome (ZES), peptic ulcer disease, gastrectomy, gastrinoma.

Introduction-
Zollinger and Ellison (1955) described a syndrome of recurrent gastric ulceration and gastric hypersecretion associated with pancreatic islet cell tumors. For many years, doctors and surgeons have agreed that a total gastrectomy is the best treatment. Cimetidine, a histamine H2 receptor antagonist, inhibits the basal and stimulation of gastric acid secretion and promotes healing of gastric and duodenal ulcers [1]. Successful treatment with cimetidine alone for 30 months has been reported. Zollinger-Ellison syndrome (ZES) is caused by ectopic hypersecretion of gastrin from neuroendocrine tumors (NETs) of the pancreas or duodenum, commonly called gastrinomas. Such high levels of gastrin cause an overproduction of gastric acid, resulting in typical symptoms, usually consisting of stomach ulcers and severe diarrhea.We present a typical case of ZES in a patient with multiple endocrine neoplasia [2].
Zollinger-Ellison syndrome (ZES) is characterized by marked hypersecretion of gastric acid associated with peptic ulcer disease, diarrhea, and gastrin-secreting non-beta-islet cell endocrine tumors (gastrinoma). The reported incidence of gastrinomas ranges from 0.5 to 4 cases per million people per year [3].

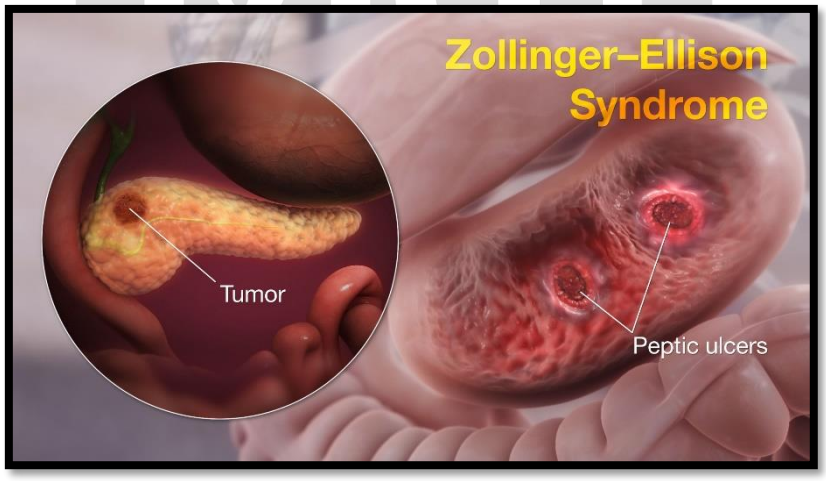

Fig no 01- Diagrammatic Representation of Zollinger ¬Ellison syndrome.
ZES is a rare cause of gastric ulcers, accounting for only 0.1-1% of ulcers. The median age of onset is 41 years, with a slightly higher prevalence of 3 in men than in women.2. Diagnosis of ZES is usually delayed by at least 5-7 years. Most gastrinomas occur as sporadic tumors, but approximately 20-25% of ZES patients have gastrinomas as part of the hereditary multiple endocrine neoplasia syndrome [4].

MEN-1 tumor suppressor gene on chromosome 11q13. Although the actual incidence and prevalence of ZES are unknown, it is estimated that 0.1-3 per million people develop gastrinoma each year. Incidence in the United States is 0.1-1% of patients with peptic ulcer disease. Gastrinoma is the most common functional pancreatic tumor in patients with multiple endocrine neoplasia type I (MEN-I). About 20% of ZES patients also have her MEN-I [5].

Patients with ZES range from he is 7 years old to he is 90 years old. The average age at onset is 50 years. Patients with both MEN-ZES and I present at an earlier age and usually develop peptic ulcer disease in the first 30 years of life. The ratio of males to females with ZES ranges from 2 to 2 [6].

Etiology & their Pathogenesis-

A 42-year-old man with a long history of peptic ulcer disease presented with persistent upper abdominal pain, melena, and signs of peritoneal irritation. A perforated gastric ulcer was suspected and urgent surgical intervention was required. Tumor lesions in the head of the pancreas were then recorded and repaired using the Whipple technique. Pathological results showed tumors indicative of neuroendocrine tumors. ZES incidence in the United States is 1–3 new cases per million people per year, making it a rare condition [7].

80% of gastrinomas are sporadic and 20% are associated with multiple endocrine neoplasia type 1 (MEN-1). ZES causes 0.1% to 1% of gastric ulcers. A man is slightly more likely that he will develop ZES. The median age at onset is 41 years. Patients with MEN-1-associated ZES may present at an early age. 30th year. ZES occurs in approximately 25% of MEN-1 patients. Patients with ZES most commonly present with symptoms related to hypersecretion of gastric acid. A gastrinoma's uncontrolled gastrin production binds to her CCK-2 receptors on enterochromaffin (ECL) cells, causing histamine release [8].

Histamine then binds to her H2 receptors on parietal cells, stimulating the release of acid. Furthermore, gastrin also has trophic effects on gastric epithelial cells and their ECL cells. Chronic hypergastrinemia increases parietal cell mass, further promoting acid hypersecretion. The enteroendocrine cells that makeup gastrinomas are well differentiated, and round, with small nuclei and prominent nucleoli. Neuroendocrine tumor markers such as chromogranin A, neuro-specific enolase, and synaptophysin are often included [9].

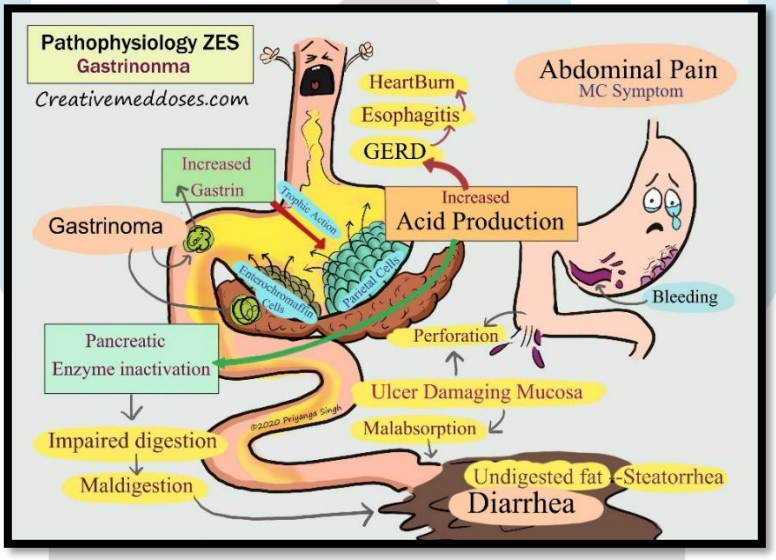

Fig no 02- Diagrammatic Representation of Pathogenesis of ZES

Presenting Symptoms-

The most common symptom of ZES is epigastralgia, consistent with peptic ulcer disease. More than 90% of gastrinoma patients develop upper gastrointestinal ulcers during the course of their illness. Similar to typical idiopathic ulcer disease, ZES is usually characterized by discrete ulcers, 75% of which occur in the first segment of the duodenum. These ulcers are often less than 1 cm in diameter, but can be more than 2 cm in diameter. Patients with ZES may also have recurrent, multiple, and atypical focal ulcers [10].

Distal duodenum (14%) or jejunum (11%). Recurrent ulcers are common after major types of gastric surgery for peptic ulcer disease, such as vestibulectomy and vagotomy. Perforated ulcers remain a common complication, with 7% of his patients having jejunal perforations. However, approximately 20% of ZES patients have no evidence of peptic ulcer disease at presentation. Persistent severe diarrhea occurs in one-third of patients and may be the sole symptom in 20% of patients. Diarrhea is caused by direct damage to the small intestinal mucosa, inactivation of pancreatic lipase, and precipitation of bile acids by low pH due to hypersecretion of gastric acid. Diarrhea is secondary to hypersecretion of gastric acid, as total gastrectomy or drug control of acid secretion controls diarrhea in all patients [11].

Up to 60% of ZES patients present with signs of dysphagia, esophagitis, and endoscopic esophageal abnormalities, including erosive inflammation, ulceration, stricture formation, and perforation. These esophageal complications can occur despite treatment with antisecretory drugs. Patients with ZES and esophageal disease should greatly reduce acid secretion [12].

Malabsorption of vitamin B12 may also occur in ZES patients due to low intestinal pH, which prevents intrinsic factor-mediated absorption of vitamin B12. Malabsorption cannot be modified by supplemental intrinsic factors [13].

Diagnosis-

Despite the availability of sensitive and specific radio immunoassays for detection of serum levels of gastrin, the duration of symptoms prior to the diagnosis of ZES remains long, ranging from 2 months to more than 30 years (mean 6 years). ZES should be suspected if a patient has one or more of the following symptoms recurrent or persistent ulcers despite treatment with the normal doses of omeprazole or H2-receptor antagonists; persistent diarrhea of unknown etiology, peptic ulcer disease associated with diarrhea, multiple ulcers in the upper gastrointestinal tract, ulcers distal to the first portion of the duodenum, a complication of a peptic ulcer such as perforation, penetration, or bleeding; a strong family history of peptic ulcers, peptic ulcers occurring at a young age, severe esophagitis or failure to obtain relief of acid reflux symptoms despite treatment with omeprazole or H2-receptor antagonists, or peptic ulceration with hyperparathyroidism and/or nephrolithiasis [14].

ZES should also be suspected if radiologic or endoscopic studies show gastric rugal hypertrophy, multiple ulcers, or ulceration of the distal duodenum and jejunum. A diagnosis of ZES should be excluded by measuring fasting serum levels of gastrin before performing an elective operation for duodenal ulcer disease, as ZES would alter the surgical approach and may increase the complication rate if the acid hypersecretion is not managed correctly [15].

Measurement of the fasting serum concentration of gastrin is the best single screening test for ZES, as more than 99% of patients with ZES will have abnormally elevated levels (>100 pg/ml). Ideally, all antisecretory drugs should be discontinued several days before the test, but this is not required for screening tests. However, if the serum gastrin concentration rises only slightly, the antisecretory drug should be discontinued for 3 days and the serum gastrin concentration should be measured again [16].

Serum gastrin concentrations vary from day to day, so if ZES is strongly suspected, the measurement should be repeated on another day. Another important criterion for the diagnosis of ZES is the presence of basal acid hypersecretion. This is defined as 15 mEq/h in patients without basal acid output (BAO) > 5 mEq/h in patients who have undergone previous acid-reducing surgery. The BAO >15 mEq/h requirement includes 66-99% of patients with ZES and excludes 90% of patients with idiopathic duodenal ulcer [17].

A simpler but less accurate indicator of gastric hypersecretion is gastric pH. A pH > 3 virtually eliminates the possibility of ZES, as most ZES patients have gastric pH values below .0. Various medical and surgical conditions other than ZES can cause hypergastrinemia, with or without gastric acid hypersecretion. The most common cause of elevated fasting serum gastrin levels is hypoxia associated with pernicious anemia or atrophic gastritis [18].

Serum gastrin concentrations in these patients can exceed 10,000 pg/mL, but measurement of gastric acid production allows for accurate diagnosis. Vagotomy, renal failure, or treatment with H2 receptor antagonists or omeprazole can also cause hypergastrinemia and low BAO. Hypergastrinemia with gastric hypersecretion occurs in patients who have recently undergone major small bowel resection, who have gastric outlet obstruction and massive gastric dilatation, or who have sinus G-cell hyperplasia or hyperfunction. increase. gain. is possible [19].

A condition that may mimic the serum gastrin and BAO levels seen in ZES patients is gastric sinus syndrome, in which the sinus lining is intact but altered after Billroth II gastrojejunostomy. There is no continuity with the acid-producing part of the stomach. However, any of the above conditions can be distinguished from ZES by a negative secretin challenge test (see below). Conversely, fasting serum gastrin concentrations >1000 pg/mL and BAO >15 m Eq/h are sufficient for diagnosis of ZES and do not require provocative testing. However, 68% of ZES patients have slightly elevated fasting serum gastrin (100-1000 pg/ml) and decreased gastric pH [20].

In such situations, a secretin provocation test is required for a definitive diagnosis. Various provocative diagnostic tests for his ZES using secretin, calcium, secretin calcium, glucagon, bombesin, or a protein diet have been proposed. The secretin stimulation test is recommended as the provocative test of choice because of its 85% reliability, simplicity, and sensitivity. In this study, a 2 U/kg bolus of secretin is administered intravenously and fasting serum levels of gastrin are measured at 0, 2, 5, 10, and 15 minutes. An elevated serum gastrin level of 200 pg/mL is consistent with a ZES diagnosis. Secretin testing is also the most sensitive indicator of recurrence or persistence [21].

Endoscopic ultrasound-

In this technique, the pancreas is ultrasound using a specialized endoscope called an end echoscope. A tiny ultrasonic probe integrated into the end echoscope bounces sound waves harmlessly and painlessly off organs to produce an image of their structure. A gastroenterologist carries out the procedure in a hospital or outpatient facility, and a radiologist decodes the images. The gastroenterologist carefully guides the end echoscope until it is close to the pancreas after feeding it down the esophagus, past the stomach, and into the duodenum. A liquid anesthetic may be sprayed or gargled on the back of the throat to an individual. A sedative aids in maintaining the person's comfort and ease. Pancreatic gastrinomas may be visible in the photos [22].

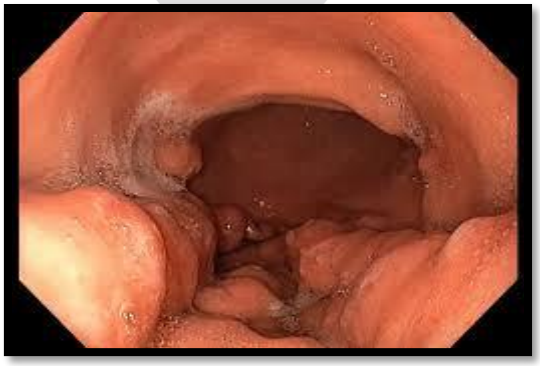

Fig no 03- Endoscopically view of ZES

Angiogram-
When an interventional radiologist, a radiologist with specialized training, inserts a thin, flexible tube known as a catheter into the large arteries, often starting from the groin to the artery of interest, the procedure is known as an angiogram. The radiologist injects contrast material through the catheter to improve the x-ray images. The interventional radiologist performs the procedure and examines the pictures in a hospital or an outpatient facility. No anesthesia is necessary; however, a light sedative may help someone feel less anxious while the procedure is being done. This test may be used to identify gastrinomas in the pancreas [23, 24].

Somatostatin receptor scintigraphy-
An x-ray technician at a hospital or an outpatient facility carries out this test, also known as an Octreo Scan, and a radiologist deciphers the results. Anesthesia is not required for a person. When injected into the bloodstream, a radioactive substance known as a radiotracer marks tumour cells only. When a gamma camera is used to scan the tagged cells, they illuminate. The examination can reveal gastrinomas in the pancreas, duodenum, and other areas of the body [25, 26].

Special Tests-
The secretin stimulation test, which is the provocative test of choice because to its great sensitivity, simplicity of administration, and lack of negative side effects, can establish the diagnosis of ZES. According to some research, secretin can directly interact with gastrinoma cell receptors to induce gastrin secretion. While the blood gastrin level declines or stays the same in individuals with other illnesses, intravenous secretin treatment causes an excessive serum gastrin increase in ZES patients. Prior to administering 2 units/kg of secretin (Secretin-Kabi) intravenously over the course of one minute, a baseline serum level of gastrin is assessed during the secretin stimulation test. Then, 2, 5, 10, 15, and 20 minutes later, repeated measurements of gastrin are taken [27].
A serum gastrin absolute rise of 200 pg/mL or more following secretin injection is a widely used indicator of a positive secretin stimulation test. With about 83% sensitivity and specificity, this finding can identify gastrinomas. However, Jensen and colleagues have proposed a new standard for the diagnosis of ZES as part of a sizable investigation that involved 293 ZES patients from the National Institutes of Health and 537 cases from the literature. The maximum sensitivity (94%) and specificity (100%) were seen at an absolute rise in gastrin greater than 120 pg/mL [28].
It is significant to highlight that another secretin formulation, produced by the Repligen Corporation in Waltham, Massachusetts, is currently also offered. This preparation is administered at a dose of 0.4 mcg/kg of body weight. The calcium infusion study is challenging to conduct and has a low diagnostic accuracy (sensitivity of 62%) Although it is also linked to significant side effects, it may be effective in treating a rare gastrinoma patient whose secretion stimulation results were mixed or negative. Unfortunately, the gastrin response during meal provocative testing overlaps with those reported in patients with antral disorders and is therefore mostly useless [29].
Although they are not widely available, studies of gastric acid secretion have historically helped in the diagnosis of ZES by determining the basal acid output (BAO) and pentagastrin-stimulated acid output (MAO) [30].
In patients who have previously undergone a vagotomy and a partial gastrectomy, hypergastrinemia with a BAO greater than 15 m Eq/h or larger than 5 mEq/h is suggestive of ZES. These studies on acid output are primarily used to track responses and establish the proper dosage of drugs that decrease acid production. Chromogranin A levels in the serum, which are typically increased in individuals with neuroendocrine tumors, including those with gastrinomas, may also be useful, according to recent research [31].

Upper Gastrointestinal Endoscopy-
Providers use upper gastrointestinal endoscopy to check for esophageal, gastric, and duodenal ulcers and esophagitis. A general term for inflammation or swelling of the esophagus. This procedure uses a small, flexible endoscope tube with a light to view the upper digestive tract, including the esophagus, stomach, and duodenum [32].
A gastroenterologist who specializes in gastrointestinal disorders does the test at a hospital or outpatient center. A gastroenterologist gently guides an endoscope down the esophagus and into the stomach and duodenum. Images from a small camera mounted on the endoscope are displayed on a monitor, enabling detailed examination of the intestinal mucosa. A liquid anesthetic may be gargled or sprayed into the back of the throat. A technician or nurse inserts an IV needle into a vein in your arm when anesthesia is administered [33].

Treatment
The main goals of treatment for patients with ZES are medical control of gastric hypersecretion (to reduce symptoms and complications) and attempted surgical cure (i.e., removal) in selected patients or control of tumor growth. Acid hypersecretion can be effectively treated both acutely and long-term with high-dose oral PPIs such as omeprazole, 60-120 mg daily. Since the development of these potent acid suppressants, gastrinoma patient survival has been highly dependent on the extent of tumor growth and disease involvement [34].
In all her ZES patients without MEN-1 syndrome and in the absence of liver or distant metastases, surgical examination with the possibility of radical resection is recommended. This includes sporadic ZES patients with localized resectable tumors, as well as patients with no tumor detected by imaging. Unfortunately, up to 30% of gastrinomas may be missed on preoperative imaging. Intraoperative methods such as duodenectomy, palpation, fluoroscopy of the duodenal wall, ultrasonography, or a combination of these techniques are very helpful in locating the gastrinoma. The goal of surgery for sporadic ZES is to cure the disease [35].
Experienced surgeons recommend extended Kochel technique and exploratory laparotomy with careful palpation of the nodal pancreas and duodenum. Reports suggest that duodenectomy is highly effective in identifying duodenal gastrinomas, which account for 60% of gastrinomas. Eligible patients should also undergo pancreatic head tumor resection or tail pancreatectomy for tail lesions, as well as duodenectomy and regional lymphadenopathy [36].

One study reported an immediate postoperative cure rate of 60%, followed by his 10-year cure rate of 34%. In another large study of 195 of her ZES patients followed 12 years after her diagnosis, surgical exploration to remove or cure the gastrinoma significantly prolonged patient survival [38].
Patients who underwent surgery had longer disease-related survival than those who did not, which was attributed to significantly reduced (residual) tumor growth and progression and liver metastases. The role of surgery in her ZES patients as part of the MEN-1 syndrome remains controversial as these gastrinomas are multifocal and rarely curable with surgery [39].
Therefore, an individualized approach should be considered for these patients. Several studies have shown that her MEN-1 patients with small gastrinomas (<2 cm) usually have an indolent clinical course and an excellent long-term prognosis. However, because the size of the primary tumor influences the development of subsequent liver metastasis, in her ZES patient with MEN 1 whose tumor exceeds 2–2.5 cm, surgical examination is recommended to reduce the risk of malignant metastasis. should be considered [40].
ZES patients with diffuse liver metastases, especially those with rapidly increasing size, have significantly reduced survival. Aggressive surgical resection or tumor reduction surgery can be considered for patients with advanced disease to improve prognosis. Embolization, chemoembolization, and cryoablation of liver lesions are alternative approaches for palliation. Somatostatin analogues are often used alone or in combination with interferon-alpha as first-line anticancer therapy [41].
Somatostatin analogues, such as octreotide and lanreotide, and the long-acting depot lanreotide ¬SR not only help control acid secretion, but also reduce tumor size and slow tumor growth. There is a possibility. Systemic chemotherapy with streptozotocin, doxorubicin, or 5-fluorouracil may reduce tumor size in a minority of patients, but does not appear to improve survival. These agents are also toxic and are reserved for patients with diffuse metastatic disease. Cytotoxic chemotherapy with temozolomide has also been reported to be effective in a small series of patients with neuroendocrine malignancies [42].
The role of liver transplantation is still under investigation, but may be considered for younger patients with metastases confined to the liver. Somatostatin analogs are often used alone or in combination with alpha-interferon as first-line anticancer therapy. These include long-acting somatostatin analogs such as octreotide-LAR and lanreotide-SR, or auto-gel formulations [43].
These not only help control acid secretion but may also reduce tumor size or slow tumor growth. Systemic chemotherapy with streptozotocin, doxorubicin, or 5-fluorouracil may reduce tumor size in a minority of patients, but does not appear to improve survival. These agents are also toxic and are reserved for patients with diffuse metastatic disease [44].

SURGICAL MANAGEMENT- CONTROVERSIES-

Although the above guidelines were developed through the last 50 years of research and treatment of ZES, some controversial aspects of treatment remain and some new controversies have emerged. Control of acid hypersecretion is no longer the focus of surgical management. However, the question remains whether acid-reduction procedures should be performed during laparotomy for tumor resection in ZES. Although vagotomy is not currently a standard part of the surgical procedure for ZES, the inclusion of parietal cell vagotomy could be useful for long-term acid-suppressing drug therapy in patients with persistent or recurrent disease [45].
It seems to lessen the need. This may be important as it can lead to undesirable consequences of long-term acid-suppressive therapy. Maybe connected. Furthermore, long-term use of PPIs in MEN-1 and ZES patients has led to the development of malignant carcinoid gastric tumors. For these reasons, vagotomy may be considered when performing laparotomy for gastrinoma. The use of pancreaticoduodenectomy in ZES is also described. Given the favorable patient prognosis, even with metastatic gastrinoma, procedures with high morbidity and mortality are withheld [46].
According to some authors, the 15-year survival rate for patients with distant metastases is still 52, and anatomical changes make Whipple less likely to resect or treat recurrent tumors. It will be more difficult to come back in the future because of future liver metastases with interventional modalities. Studies have shown that pancreaticoduodenectomy may be curative in patients with MEN1. However, these small studies did not consecutively use full biochemical evaluation to rule out recurrent disease [47, 48].
A recent study showed a cure rate of 77% for a patient with MEN1-ZES, but his median follow-up was less than 1 year. Others estimate that the incidence of duodenal gastrinoma is as high as 90% in patients with MEN and that pancreatoduodenectomy may be a more definitive procedure to prevent a recurrence. For these reasons, some authors now recommend Whipple as the first-line treatment for MEN1-ZES patients whose gastrin source is localized to the head of the pancreas by selective arterial secretin injection [49, 50].
I'm here. Our current recommendation is to limit Whipple surgery to young patients with large pancreatic head tumors that are not amenable to removal. Whipple resection is often non-curative, especially with conservative surgical procedures. -1 May increase cure rate in patients. Further studies should be performed to compare long-term survival and quality of life [51-55].
Although no controlled clinical trials exist, there appears to be a relative consensus regarding the surgical treatment of metastases. Although there is evidence that resection of liver metastases improves survival, it is difficult to affirm that the demonstrated survival differences depend on surgical treatment rather than the extent of the disease itself [56, 57].

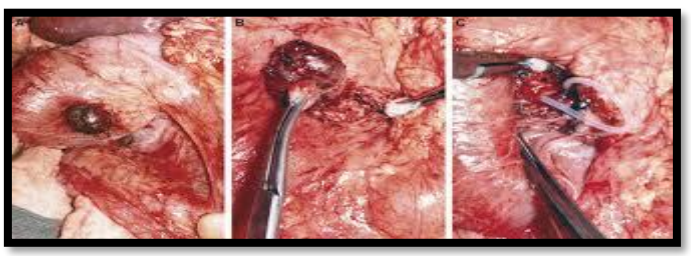

Fig no 04- Surgical View of ZES

A significant proportion of patients with metastatic gastrinoma can undergo hepatic resection, resulting in elimination of all known disease with a 5-year survival rate of up to 85%. In addition to potential survival benefits and surgical cure of metastatic disease, cytoreductive surgery may benefit amelioration of functional endocrine tumor syndromes. The current recommendation is to attempt liver resection if preoperative imaging allows resection of at least 90% of the tumor. Laparoscopy is increasingly being used in complex abdominal surgeries, with increasing experience with laparoscopy, especially for pancreatic resection. There are several reports of laparoscopic resection of pancreatic neuroendocrine tumors, especially insulinomas [58, 59].

Laparoscopic pancreatic tail resection is a promising surgery with a complication rate (fistula) equal to or lower than that of open surgery. Laparoscopic removal seems possible. However, laparoscopic treatment of gastrinoma has some significant challenges. More than half of gastrinomas occur outside the duodenum or pancreas. Gastrinomas tend to be larger than other neuroendocrine tumors and are more likely to metastasize [60, 61].

These factors make gastrinomas less susceptible to minimally invasive techniques, as evidenced by the higher conversion rates in the reported series. Because many gastrinomas are located in the duodenum, the feasibility of endoscopic resection has been debated. There are reports of sling polypectomy or band ligation. Some of these cases reported healing of ZES but also had at least one perforation [62, 63].

Lee et al61 reported treatment of his ZES with endoscopic band ligation. In this case, the patient refused surgery. Serum gastrin levels dropped from 647 to 100 pg/ml, and a postoperative biopsy near the banding site was negative for tumor. Although this is an interesting report, there are several factors that prevent endoscopic treatment from being considered equivalent to surgery in patients eligible for surgery. First, gastrinomas arise in submucosal sites, making them inaccessible and unsafe for endoscopic resection [64].

Moreover, they are often invasive beyond the submucosa. Second, banding does not remove lesions but rather peels them off. This makes the pathological examination imperfect. Therefore, it is difficult to assess margins. Third, in previous studies, endoscopy was inferior to duodenectomy for gastrinoma detection. A gastrinoma may be missed if detected only by endoscopy. Finally, up to 60% of gastrinomas are associated with lymph node metastasis at diagnosis, making endoscopic treatment of duodenal gastrinomas an imperfect oncological treatment [65].

Conclusion-

Only 0.1–1% of stomach ulcers are caused by ZES, making it a rare cause of ulcers. There was a suspicion of a perforated stomach ulcer, necessitating an immediate surgical procedure. ZES is a rare illness with an annual incidence of 1-3 new cases per million persons in the United States. Stomach ulcers and pelvic pain are the most typical symptoms of ZES. During the course of the illness, upper gastrointestinal ulcers appear in more than 90% of his gastrinoma patients. ZES is often characterised by distinct ulcers, 75% of which develop in the first segment of the duodenum, similar to typical idiopathic ulcer disease. 20% of gastrinomas are linked to multiple endocrine neoplasia type 1 and 80% are sporadic (MEN-1). ZES is somewhat more likely to affect men than women. He is 41 years old on average when he is diagnosed. Patients with ZES linked to MEN-1 may show symptoms at a young age. thirty years. Approximately 25% of MEN-1 individuals experience ZES. Jejunum (11%), or the distal duodenum (14%) Following major gastrointestinal procedures for peptic ulcer disease, like vestibulectomy and vagotomy, ulcer recurrence is typical. Jejunal perforation was observed in 7% of his patients, and perforated ulcers continue to be a common consequence.

Conflicts of interest-

There are no conflicts of interest and disclosures regarding the manuscript.

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