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RESEARCH ARTICLE

PRIMITIVE MESENTERIC CLAMP SYNDROME : UNUSUAL CAUSE OF UPPER GASTROINTESTINAL OBSTRUCTION

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Abstract

The syndrome of the mesenteric clamp or Wilkie's syndrome is defined by a compression of the third duodenum between the superior mesenteric artery, and the aorta . The symptoms are variable and non specific. Management is based first on medical treatment with recourse most often to surgery if failure. We report a review of the literature by reporting an observation of a case.

Observation: This is a 27-year-old patient with a history of intermittent chronic vomiting since the age of 16, unexplored, who for the past 1 year has been worsening vomiting becoming persistent with epigastralgia. gravity, evolving in a context of deterioration of the general state and slimming. A high gastrointestinal endoscopy that showed significant gastric stasis, injected abdominal CT found a disparity in caliber against D3. An upper gastrointestinal fluoroscopy showed gastric stasis with a stomach reaching the pelvis. The management consisted in a surgical treatment after failure of the medical treatment with good evolution. The mesenteric forceps syndrome is a rare and benign condition. Positive diagnosis is hard on the scanner. The treatment is medical in the first place but the use of surgery is common.

Introduction:-

Wilkie syndrome or mesenteric clamp syndrome is a rare, benign pathology that is defined by the extrinsic compression of the third portion of the duodenum, between the superior mesenteric artery in front and the aorta behind [1,2]. An obstacle sitting at the level of the 3rd duodenum is a weighty argument towards aorto-mesenteric forceps. The pathophysiology is multifactorial [3], and the diagnosis is radiological based on duodenal opacification [4]. An inadequate loading can lead to serious consequences hence the interest to update the condition by this observation, with review of the literature.

Observation:-

This is the case of a 27-years-old patient with a notion of intermittent vomiting since the age of 16, unexplored, who had for 1 year a worsening of vomiting becoming chronic, late postprandial with permanent epigastralgia type of gravity aggravated by food intake and relieved by late postprandial vomiting without the notion of stopping of materials and gases or gastrointestinal bleeding. On admission, the patient is malnourished with a BMI of 18 kg / m². Her clinical examination noted an epigastric sensitivity with fasting lapping, a dehydration skin fold, and a slit of the

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adipose panicle without impaction or abdominal mass. The impact bioassay revealed a disrupted ionogram made of a hyponatremia with functional renal failure, a deficit report made of cholesterol, albumin and protein in the blood did not notice any abnormality. An etiologic assessment made of a high endoscopic fibroscopy revealed an aspect of antro-fundic pangastritis, with a large gastric stasis, a duodenal mucosa was a normal appearance with preserved folds. Biopsies were made did not show signs of malignancy but rather concluded to a discrete nonspecific interstitial duodenitis associated with chronic gastritis active to HP. An abdominal CT scan reports extensive stasis with gastric distension and a zone of disparity of caliber at the level of D3 with a flat appearance at the level of the jejunal cuffs (Figure 1). The upper gastrointestinal fluoroscopy showed a stasis stomach descending to the pelvis (Figure 2). The management consisted of a medical treatment based on a correction of the hydro-electric disorders associated with the placement of a suction catheter with postural treatment and a stop of feeding. Faced with non-improvement under medical treatment, surgery was indicated. He had a median laparotomy, Intraoperatively, gastroduodenal dilatation was confirmed secondary to extrinsic compression of the third duodenum between superior mesenteric artery and aorta (Figure 3). A gastro-entero-anastomosis was performed without incident. The postoperative course was simple, the feeding was resumed on day 3 postoperatively; the patient is declared outgoing on D7 with simple postoperative follow-up and disappearance of vomiting with a weight gain of 5 kg in 5 months.

Discussion:-

Many names have been used over the years for Wilkie syndrome [5]: mesenteric forceps syndrome, mesenteric clamp syndrome, superior mesenteric artery syndrome, duodenal arterio-mesenteric compression syndrome. It was described in 1861 for the first time by Rokitanski during an autopsy [4]. Pathophysiology was described by Wilkie based on a series of 75 patients in 1927 [6]. It is characterized by the presence of a reduced mesenteric aorta-arterial distance, less than 8 mm at the height of D3 associated with an aorto-mesenteric angle thus becoming less than 20 [7]. It is a syndrome that affects young people between 17-39 years of age [5], its prevalence is from 0.013% to 0.78% [4], several clinical forms have been reported in the literature [8] : acute or chronic, postoperative, infant, anorexia nervosa, spine surgery, resuscitation.

Some contributing factors reported in many series were responsible for its occurrence: anatomical abnormalities such as brevity or hypertrophy of the Treitz ligament, a low origin of the superior mesenteric artery on the aorta. Other situations may promote duodenal compression including rapid weight loss, spinal deformity (spinal hyperlordosis, trauma or surgery of the spine, correction of scoliosis); certain autoimmune diseases (scleroderma), an intervention on the aorta; cerebral palsy; the presence of mesenteric lymphadenopathy; aneurysm of the abdominal aorta; an ileoanal anastomosis. Pediatric forms have been described incriminating the genetic factor in the occurrence of mesenteric forceps syndrome [2,3].

In our observation, none of these anomalies were found. It may be a primitive form that may be related to anatomical factors or incriminate significant weight loss at the onset of the condition. The revealing symptoms are variable and nonspecific dominated mainly by vomiting [4]: it may be an acute or more often chronic picture in 90% of cases [4] as the case of our observation with resonance on its ionogram. A digestive occlusion can be indicative of this syndrome with all its complications in terms of biological as well as respiratory and digestive and thus achieves the acute form. It may be associated with compression of the left renal vein responsible for proteinuria, hematuria and arterial hypertension [7]. In addition, the chronic form is less noisy and rarely involves a life-threatening condition. It may be intermittent postprandial epigastralgia, a sensation of gastric fullness, nausea, vomiting, or stasis-secondary reflux [2]. Indeed in our observation, the clinical diagnosis is made in front of a chronic symptomatology, made of repeated chronic vomiting associated with abdominal cramps with sagging of the condition. The positive diagnosis is evoked in front of direct signs on the abdominal CT injected in 95% of cases, it is about a duodenal dilation upstream of the obstacle and thus makes it possible to exclude other causes of obstruction and to calculate the angle and the duodenal crossing. The abdomen without preparation remains desirable and may show gastric dilation with two liquid levels, gastric and duodenal, achieving a double bull appearance [4]. As for upper gastrointestinal endoscopy has no diagnostic value. The upper gastrointestinal fluoroscopy is a key examination that may show indirect signs such as duodenal dilatation with linear stopping of the contrast medium at D3, a delay of 4-6 hours in the upper gastrointestinal fluoroscopy with a baryte passage in the supine jejunum ventral or lateral left. Doppler-coupled ultrasound may show dilatation of the second duodenum with respect to peristalsis, sometimes a reduction in the space between the superior mesenteric artery and the aorta [4]. Both acute and chronic forms require initial medical management with the placement of a nasogastric tube left in soft suction, nutrition with frequent meals diluted or enteral nutrition by weighted jejunal tube placed beyond the compression zone. Postural maneuvers in the left lateral decubitus or procubitus in parallel with the correction of

hydroelectrolytic disorders have an efficiency of the order of 30 to 50% in the acute forms [4]. Improvement of symptomatology depends mainly on the presence of an obvious and correctable promoting factor, otherwise it leads to failure in other more complex cases (cerebral palsy, spinal pathology) [2,3]. Surgery is considered only after failure of medical treatment and indicated in 75% of cases in disabling chronic forms with signs of maldigestion, weight loss and pronounced duodenal dilatation, as the case of our observation [4]. Its success rate is of the order of 90% [3,8]. Several alternatives have been described for surgical management: Strong's intervention which is based on the section of the ligament of Treitz in order to lower D4 and thus the crossing distance between D3 and the superior mesenteric artery. This technique is used in pediatric forms. Other surgical procedures indicated for the adult forms: the retro mesenteric translation of the duodeno-jejunum and the gastroentero-anastomosis and in the end the duodenojejunostomy laterolateral between the duodenum and the first jejunal loop which remains the most frequently performed without duodenal resection or uncrossing [9]. In our patient, we opted for laparotomy duodeno-jejunostomy due to the nutritional status of the patient. The prognosis is generally favorable if the diagnosis and the decision does not load are precocious as illustrated our case.

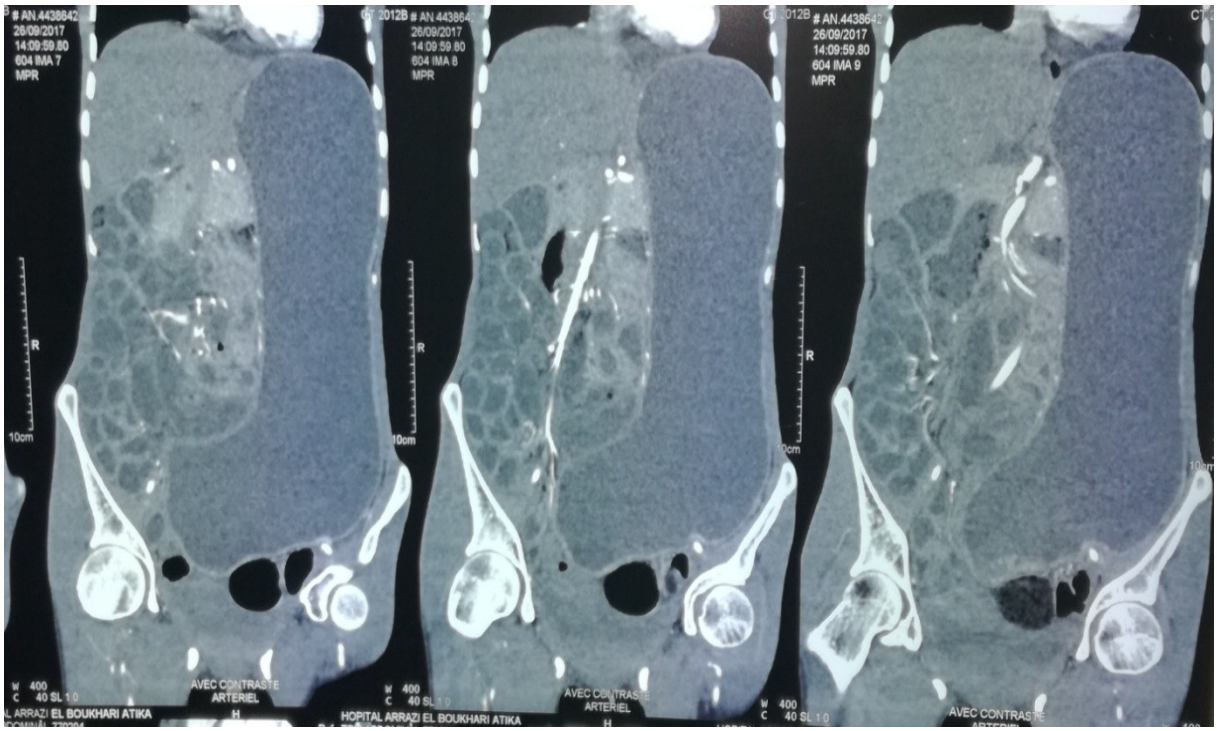

Figure 1:- abdominal CT: Disparity in caliber at D3 with flat aspect of jejunal loops downstream related to mesenteric forceps syndrome associated with extensive stasis and gastric distension.

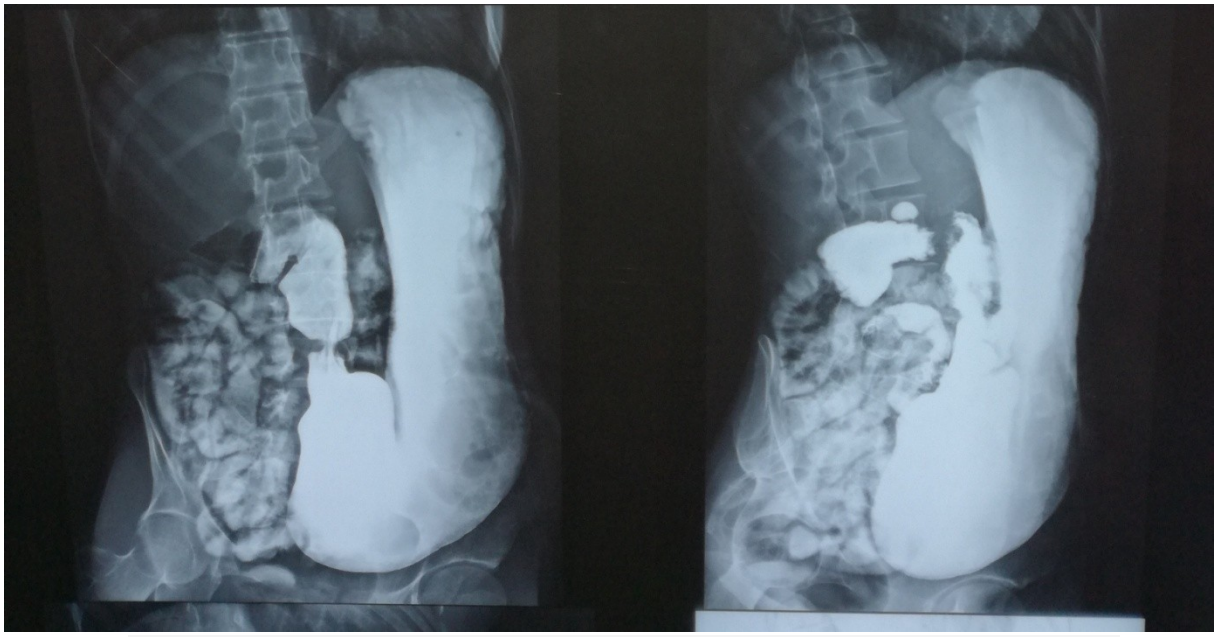

Figure 2:- upper gastrointestinal fluoroscopy : pelvic stomach in gastric and duodenal stasis.

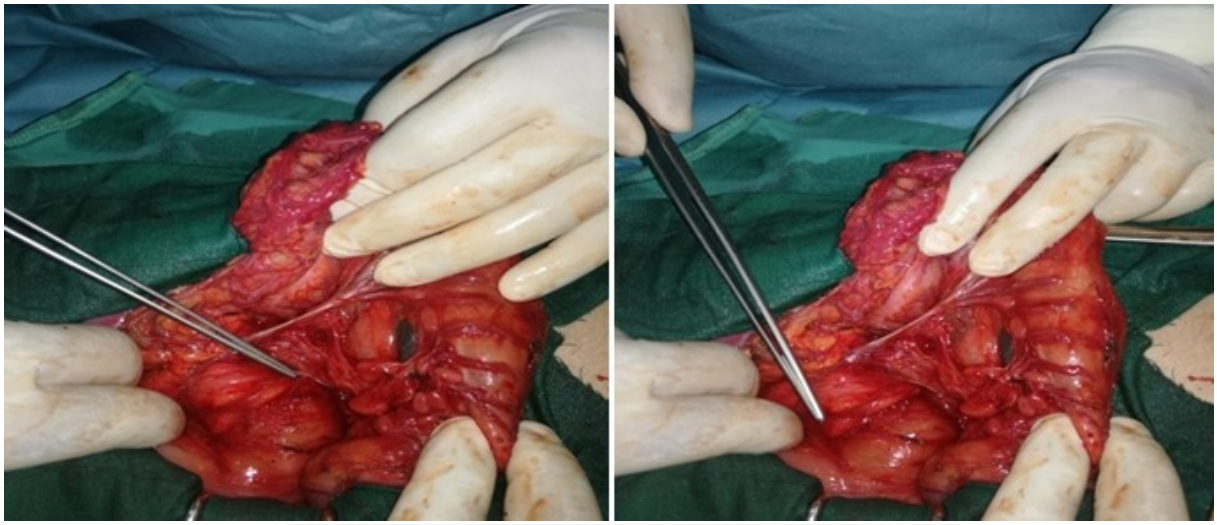

Figure 3:- intraoperative overview showing the dilation of D3.

Conclusion:-

The syndrome of the mesenteric forceps is a rare and benign affection which can have serious consequences if the care is made late. The clinic is often nonspecific. The injected abdominal CT scan is of vital interest to make the diagnosis in more than 90% of cases. Treatment is medical in the majority of cases and relies on nasogastric aspiration and postural maneuver. The use of surgery is becoming more common.

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