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Research Article

DIAGNOSTIC AND THERAPEUTIC IN THE INTESTINAL DUPLICATION

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ABSTRACT

Pollution to the environment by heavy metals is mainly due to human activities. The presence of heavy metals even in traces is toxic to both plants and animals. The excess of cobalt can affect the plant's physiological functions. In order to control cobalt heavy metal soil pollution by a low cost technique an attempt was made employing biosorption using *Dictyota dichotoma*. In this study, the seedlings of (cowpea) *Vigna unguiculata* (L) were treated with various concentrations of cobalt chloride and its impact on the morphometric, biochemical and enzymatic characteristics were studied. Eight days after treatment with different concentrations of cobalt chloride (2mM, 4mM, 6mM, 8mM, & 10 mM), the growth parameters such as leaf area, fresh weight, dry weight, shoot length, root length were found decreased than in the control. Biochemical parameters such as soluble sugar and protein content were decreased with the increase in the concentrations of cobalt chloride. On the contrary the contents of free amino acid, proline and leaf nitrate were increased with increase in the concentrations of cobalt chloride. The activities of enzymes such as catalase and peroxidase barring nitrate reductase were found increased with the increase in the concentration of cobalt chloride. Application of 6mM cobalt chloride solution treated with various concentrations of *Dictyota dichotoma* such as 2gm/L, 4gm/L & 6gm/L & 8gm/L on the experimental plants has brought about changes in the suppressed characteristics showing relief from stress due to cobalt chloride. Atomic Absorption Spectroscopy (AAS) technique was employed to confirm the presence of cobalt in the treated and control plants. Comparison of the values of treated plants with control reveals that cobalt chloride has seriously affected the (cowpea) *Vigna unguiculata* (L) plants and *Dictyota dichotoma* is effectively biosorbed the cobalt heavy metal.

INTRODUCTION

The midgut (mid gut) is the primitive gut portion embryologically is open ventrally in the yolk sac and is vascularized by the superior mesenteric artery. In The fifth and sixth week, the middle intestine (bowel loop) expands more rapidly than the embryonic body; this discrepancy, also accentuated by the liver growth, induces a whole series of movements up to reaching the end position of the small intestine and colon; these successive movements can be divided into three stages: First stage: herniation. It forms an umbilical hernia saline. A gang mesenteric fixing the proximal portion of the duodenum and prevents its entry into the umbilical cord (herniation). This structure maintains its anatomical entities in the adult form of the ligament of Treitz. Second stage: the return of the herniated loops in the abdomen during the tenth week the herniated intestinal loops begin to fall into the abdominal cavity, Third stage: setting process Around the twentieth week of gestation takes the final positioning of the colon and mesentery. This fixing process can cause stoppage anomalies at various points. By the term "digestive duplications" means hollow formations in cystic or tubular morphology that are in intimate contact with the various segments of the alimentary canal Ladd in 1937 proposed the term "intestinal duplication" in an attempt to group under a single diction All Definitions used previously as "cysts enterogene", "enteric cysts", "dual ileum", "giant diverticula", "abnormal diverticula of Meckel" digestive duplications are rare and include the 0.1- 0.3% of all malformations congenital. (1,2,3) Those in Jejunoileal localization are the most frequent (more than 50% of all duplications) numerous are the hypotheses proposed to explain the embryogenesis of the intestinal duplications. One of these is based on a endodermal defect that follows to an altered during the separation of the notochord pre somitico stage of embryonic development. This theory may explain the mediastinal duplication, but not those intra-abdominal. Other authors suggest a failure to regression of these diverticula embryonic. (Stomach, duodenum and ileum) that are regularly present during the early stages of intrauterine development: with this theory does not explain the

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formation of the long tubular duplication. Other AA. give blame to a bad intestinal recanalization for an error in the vascularization process during the 6th - 7th week of fetal life, in which the spaces formed by multiple cystic vacuoles fuse with each other, but do not come together with the main lumen: even this theory has some inconsistencies, such as diverticular since it does not explain why the duplications are always localized between the sheets of mesentery, while the vacuolization process is in all sides of the intestinal lumen. In rare cases, the cystic dilatation have a extra mesenteric headquarters, free in the peritoneal cavity and the intestines connected only by a thin vascular pedicle. (4,5,6) The purpose of the study in conjunction with the clinical observation and to expose current diagnostic and therapeutic guidelines for the treatment of this disease.

MATERIALS AND METHODS

Paz 7 years female is received to our attention in May 2016 at the Surgical Clinic II of the University of Catania. AUO Polyclinic. The clinical history of the small patient showed the presence of left flank pain with palpable abdominal mass. and distension, vomiting do not digestive blood / melena, you will peritonitis diarrhea Performed the instrumental diagnosis showed:

Ultrasound fig 1What shows no alterations in the liver, pancreas, spleen and kidneys. At below the lower pole of the kidney, posteriorly and inferiorly to the expansive formation stomach weakly vascularized well circumscribed and encapsulated, to echo inhomogeneous structure and most of about 7 cm axis. The presence of liquid in the pelvic cavity.

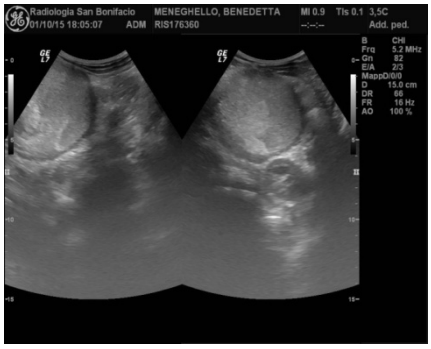

Fig 1 ultrasound expansive training

low-dose CT without contrast medium for two reasons radioprotezionistici.fig is confirmed in the left flank expansive training well circumscribed encapsulated approximately 7 x 8 cm, with uneven fat content and in the sloping content corpuscular liquid density (mucin?). Non-invasive characteristics.

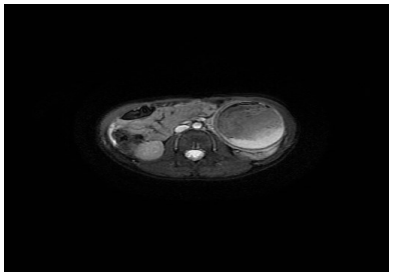

Fig 2 TC capsulated trainin

RM: 3 fig 4 the left side confirms expansive formation with rounded morphology, with more than 7 cm axis, bordered by coarse device capsule. The content of the lesion is uneven, almost completely fluid (hyper-intensity on T2-dependent sequences), with "image layer" in which the sloping portion appreciate blood components or alternatively mucus / protein (hyper-intensity on T1 sequences employees). Apparently appreciable along the medial profile of the pedicle training with the mesentery. After administration of contrast medium (Dotarem, 4 ml) not significant enhancement. What is described is not unambiguous interpretation of being able to be attributable to mesenteric cyst or, alternatively, to intestinal duplication

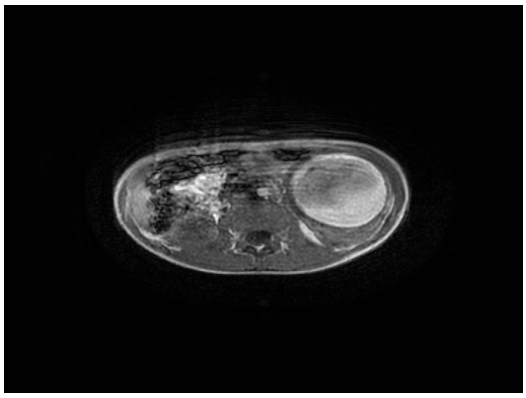

Fig 3 RMN formation fluid

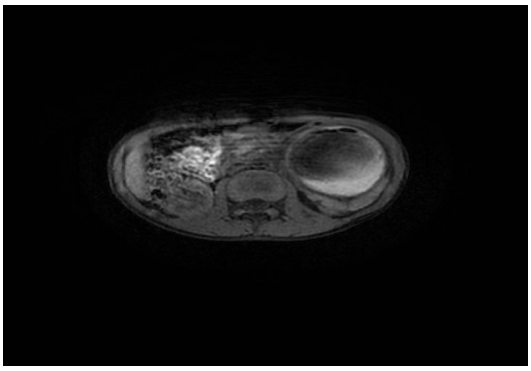

Fig 4 RMN pedicle mesentery

RESULTS

instrumental diagnostics, especially for cystic forms, is based on the survey neonatal ultrasound in the presence of specific symptoms or as noted in the patient when the clinical picture is presented in all its relevance (sub occlusive crisis). The ultrasound examination detects a quarry training with muscle walls, adjacent to a segment of the digestive tract. Figure 1. Figure 2 The TAC and RM figure 3-4 are useful to improve the topographical study, the seat and in particular its relationship and the content of the training as well as detect the mesenteric pedicle. Are obsolete both the direct abdomen which stressed, the characteristic pattern "double bubble" with the presence of air in the stomach and the first portion of the duodenum. As well as (Rx digestive system) or barium enema X-ray to rule out an intestinal malrotation. Not have been necessary in the patient under study in the bowel scintigraphy with labeled red blood cells used to detect duplication or heterotopic gastric mucosa contained in it, nor the endoscopic examination because current diagnostic tests accurately indicate topographical aspects of the lesion. The surgical therapy of

choice in cystic or tubular duplications, not very extended, is the total excision of the duplication with the intestinal segment resection contiguous and subsequent anastomoses Fig 5-. In extended tubular duplication of the small intestine you can be performed total excision of the duplication by extra mucosal dissection or "stripping" of the mucosa. The mucosa in the tubular duplication is in fact weakly cohesive to the muscle wall. Stripping of the mucosa can also be performed with multiple incisions in the muscle wall of the duplication.

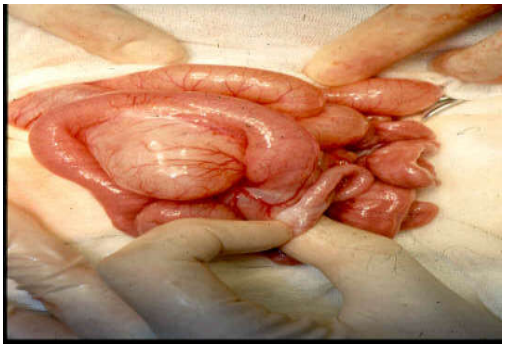

Fig 5 Anatomy intestinal duplication

DISCUSSION

Digestive duplications in cystic or spherical morphology as that observed account for 90% of all digestive duplications. They are located on the side mesenteric and are found mainly in the small intestine-mind, particularly in the terminal ileum.(7.8.9,10) Cystic duplications placed in the vicinity of the ileocecal junction can cause intestinal obstruction during the neonatal period or may remain asymptomatic and be a potential point of intussusception, such as a common Meckel's diverticulum. The mucosa which lines the duplication is equal to that of adjacent normal bowel and possess the coalescence of the muscle layers in the point in which they are in contact, thereby preventing to find a cleavage plane, and therefore is difficult to dissect and separate the duplication of the adjacent intestinal segment. Furthermore, also the vascularization is in common; the veins and the arteries that supply blood to the intestine adjacent shall run usually on the surface of the duplication, for which the only attempt to resect the duplication can fail for the normal vascular compression of the loop. Cystic duplications, in 80% of cases, do not communicate with the lumen contiguous. They contain a clear mucoid substance secreted by the mucous membrane, sometimes haemorrhagic for the presence of gastric ectopias with mucosal ulcerations. Clinically cystic duplications appear as an asymptomatic mass or with a framework occlusive (intussusception or volvulus). Bleeding and melena symptoms are rare, because the duplication cysts do not communicate with the adjacent bowel. Duodenal duplications which are located mainly in the wall of the second portion of the duodenum in relation with the stomach, the pancreas or the bile ducts. The CT examination revealed cystic lesions, which by their nature can create differential diagnostic difficulties especially with the cysts of the extrahepatic biliary tract (common bile duct cysts) and pancreatic cysts. Duodenal or jejunal duplication, exceptionally (1.8% of cases), they can go back into the thorax through the right pillar of the diaphragm and cross from behind the esophagus and aorta "thoraco-abdominal duplication." Duodenal duplication remain mostly asymptomatic or cause partial obstruction of the duodenum. The presence in 50% of cases of heterotopic gastric mucosa in the duplication, is responsible for the formation of peptic ulcer with possible bleeding or perforation. (11,12,13,14,15) Surgical treatment of duodenal duplication is the partial excision, mucous removed (stripping of the mucosa), leaving in place the sero-muscular lining finally have a high incidence of associated malformations: spinal, DRFI, intestinal atresia, skeletal and urinary abnormalities

CONCLUSION

In intestinal duplication 70% of cases are diagnosed within the 1st year of life the rest within two years of life; even though, as in the case observed duplication presented a late symptom, this shows how the symptoms are variable as a function the venue, the type, size and gastric heterotopia. Often the diagnosis is occasionally during other investigations and abdominal duplication: the pathognomonic signs are the mass, abdominal pain, vomiting, bleeding, acute abdomen. The considerable increase in the diagnosis more reliable due to the diagnostic features of images, sending in the attic the traditional diagnosis. The treatment of choice is the one related to the surgical with the 'radical removal of duplication.

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