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CLINICAL OBSERVATION

A RARE CASE OF MEGAESOPHAGUS REVEALED BY LOW DYSPHAGIA ASSOCIATED WITH VOMITING

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Summary:

Megaesophagus or cardiospasm, also known as achalasia, is a rare pathology of the oesophagus characterised by the absence of peristalsis at the level of the oesophageal body associated with a defect of relaxation of the lower sphincter following swallowing. It is a rare disease in the tropics. The present work reports a case of acute lower dysphagia associated with vomiting admitted to the emergency department revealing idiopathic megaesophagus. The aim of this observation is to develop a reliable diagnostic strategy for any dysphagia and to draw up a schedule of the complementary examinations available to support it in order to implement the current treatments according to their availability.

Key words: achalasia, cardiospasm, dysphagia, megaesophagus

Introduction:

Megaesophagus or achalasia is a disease of unknown origin characterised essentially by the complete absence of normal peristaltic contraction in the oesophageal body and by the absence of normal relaxation of the gastroesophageal sphincter after swallowing (1, 2). This condition is thought to be rare in the tropics where the literature is more likely to report tumour or inflammatory pathology (3).

We report here a case of megaesophagus observed in the emergency department following gastroenterological and radiological investigations at Sikasso hospital in April 2023.

Observation:

Miss O.S, 20 years old with a history of epigastralgia 3 years ago for which she underwent a biological check-up for H.P and a FOGD which found erythematous gastritis.

The results of these examinations came back without any particularity and she was put under symptomatic treatment with PPI with a notable clinical improvement.

The current episode was 6 months ago and was marked by the onset of low-grade dysphagia to solid foods and early episodes of intermittent postprandial vomiting. The clinical evolution was marked by the appearance of a retro sternal pain and a nocturnal cough 4 weeks ago, increasingly frequent vomiting and anorexia causing a progressive alteration of her general state and anxiety leading the parents to a consultation in the emergency room.

Clinical examination found: a conscious patient GCS 15, altered, slightly slowed, haemodynamically stable, conjunctiva well stained, BP=110mmHg/80 mm Hg, Pulse=93 b/mm, FR=15 cycles/mn, T°=37.4°, SPO2=98%, moderate dehydration folds, dry tongue not stripped, height=1.63m, Weight=41kg (usual weight=52kg), a BMI=15.47. Slightly decreased osteotendinous reflexes.

The rest of the physical examination was unremarkable.

The biological assessment revealed hyponatremia at 126 meq/l, hypocalcemia at 83 mg/l, Hg=12 g/dl, the renal assessment was normal (Urea= 0.34, creatinemia= 5.71mg/l), blood glucose at 0.83 g/l, kalemia at 3.3 meq/l

Albumin=24 g/l, total protein= 52.40g/l.

At the endoscopic assessment:

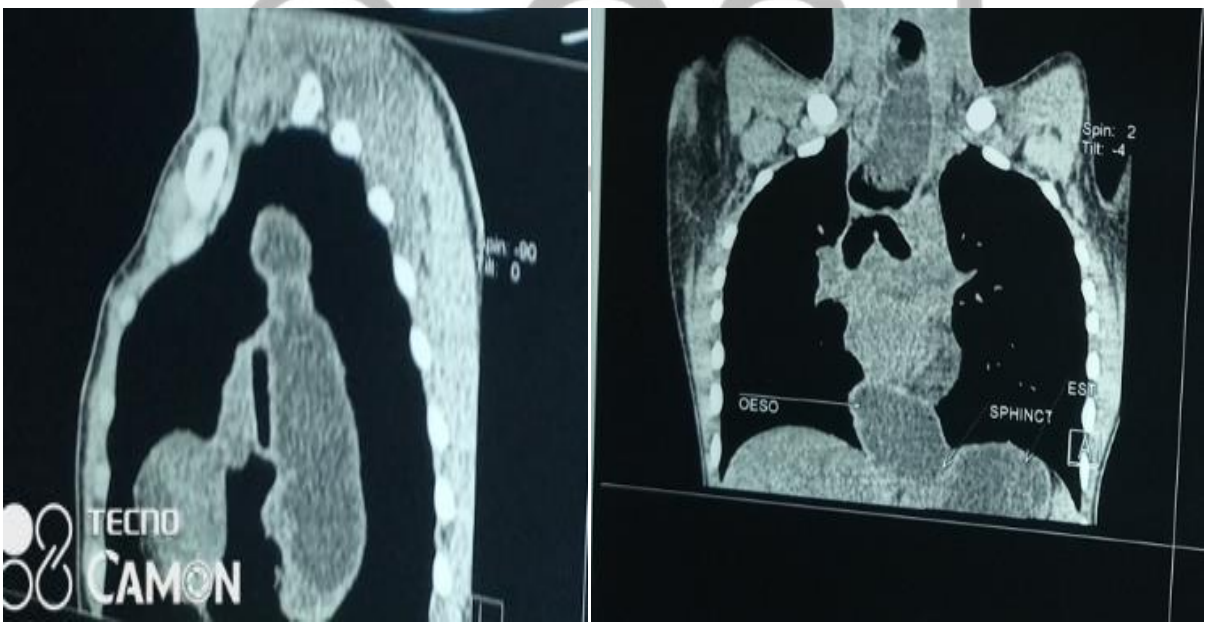
FOGD:

Upper GI endoscopy showed memorial dilatation of the lower oesophagus with supracardial stenosis without associated mucosal abnormality and the presence of a "protrusion" when the endoscope passed.

On radiological examination:

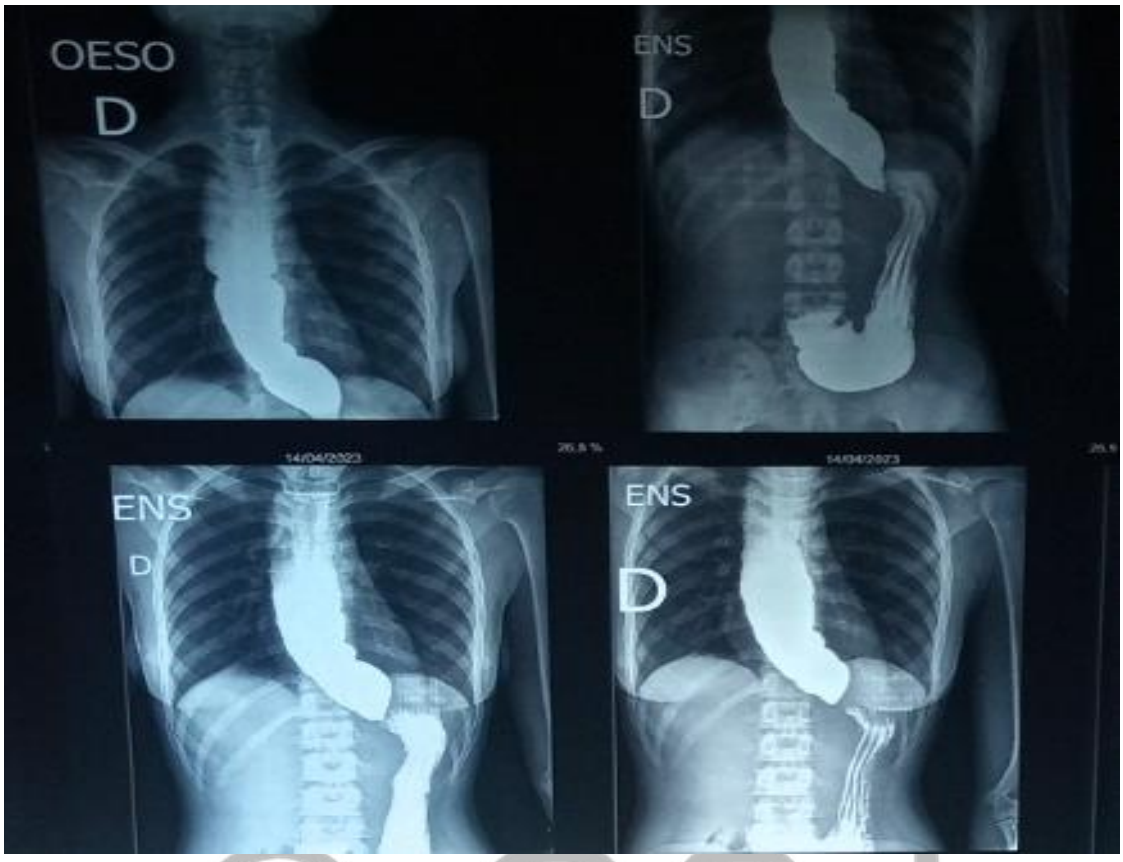
Frontal X-ray of the thorax: no special features.

The thoracoabdominal CT scan showed an appearance suggestive of congenital achalasia or megaesophagus.



Figures 1: significant esophageal dilatation.

The oesogastroduodenal transit (TOGD)



Figures 2:

The oesogastroduodenal transit (TOGD) showed a radiological aspect evoking the diagnosis of achalasia: oesophageal dilatation with a "bird's beak" narrowing of the oeso-gastric junction

Comments:

Achalasia is a primitive motor disorder of the oesophagus characterised by dysfunction of lower oesophageal sphincter (LES) relaxation and aperistalsis of the oesophageal body. The etiopathogenic mechanism appears to involve autoimmune, viral or neurodegenerative processes associated with a genetic predisposition resulting in damage to the neuroganglionic cells of the oesophageal myenteric plexus [4].

Achalasia is the most common oesophageal motor disorder with a mean annual incidence of 0.03-1.63 per 100,000 populations in adults [5-6]. There is no gender or ethnic predilection [7, 6]. However, the incidence increases with age and occurs most frequently between the fourth and seventh decade [6].

Low-level dysphagia to solids revealed the disease in our patient, initially associated with vomiting and then marked by the appearance of retro-sternal pain and a nocturnal cough.

Low-grade dysphagia is almost always selective to solids in half of the cases, sometimes resolved by ingestion of liquids [8]. Pain is indicative of the disease in 2% of cases, constrictive, retro-sternal, diurnal or nocturnal, with or without meals [9, 8].

Disorders of oesophageal motricity lead to food stasis upstream of the stenosis, which can lead to the passage of food residues into the airways responsible for episodes of nocturnal coughing as observed in our patient.

Respiratory symptoms are present in 22 to 45% of cases, sometimes even revealing the disease, with a symptomatology consisting of paroxysmal nocturnal cough, sometimes with inhalation pneumopathy in 10% of cases, and respiratory discomfort at mealtimes [10].

A decrease in weight with an alteration in general condition was noted in our patient in relation to the blockage of food at the level of the lower oesophagus stenosis and the associated episodes of vomiting.

Weight loss was noted in more than half of the cases [11], associated with an altered general condition in the advanced stage in relation to oesophageal blockages [12, 13].

The radiological images are typical of megaesophagus showing dilatation of the oesophagus in our patient, particularly the TOGD showing aperistalsis and regular narrowing of the lower oesophagus.

The oesogastroduodenal transit (TOGD) can demonstrate a loss of tertiary contractions associated with a relaxation defect of the LES [11].

Endoscopy is a first-line examination in the presence of dysphagia, which, together with thoraco-abdominal CT, allows a tumour process to be ruled out, even if it is normal; it does not rule out achalasia [11].

Manometry, another key examination after having eliminated an obstacle to confirm the diagnosis, could not be performed due to lack of availability on site.

In terms of treatment, the different therapeutic options discussed to reduce pressure in the lower oesophageal sphincter were pharmacological treatment, endoscopic treatment (botulinum toxin injections, per endoscopic balloon dilatation, PerOral Endoscopic Myotomy) and surgical treatment. After giving extensive information on each, the patient chose the pharmacological treatment although its effect was temporary. She was put on nifédipine 10 mg (ADALATE) which decreases cardiac muscle contractility and acts on the basal pressure of the lower oesophageal sphincter at a rate of one tablet per day taken continuously, combined with oesomeprazole 40 mg injectable in IVD every 12 hours until the remission of vomiting and then the relay by oral form.

The clinical evolution was marked by a clear improvement of the symptoms and especially a moderate resumption of feeding for the moment.

Conflicts of interest: no conflicts of interest.

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