“TETHERED FAT SIGN”: THE SONOGRAPHIC SIGN OF OMENTAL INFARCTION

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Abstract—Our purpose is to describe the ultrasound sign for a correct non-invasive diagnosis of omental infarction in children. From January 2014 to December 2018, a total of 234 children (109 boys and 125 girls, age range 3–15 y) with acute right-sided abdominal pain, admitted to our hospital with a presumptive diagnosis of acute appendicitis, were prospectively evaluated. In all patients, abdominal ultrasound was performed, and the omental fat was always evaluated. In 228 patients, the omental fat resulted to be normal or hyperechogenic, never tethered, and they results affected by other causes of abdominal pain different from omental infarction (such as appendicitis, pancreatitis, urolithiasis and others). In the remaining 6 children, we found a hyperechoic mass between the anterior abdominal wall and the ascending or transverse colon in the right abdomen quadrant, suggesting the diagnosis of omental infarction. This subhepatic mass was always tethered to the abdominal wall, motionless during respiratory excursions. We named this finding the “tethered fat sign.” The diagnosis was confirmed with laparoscopy in 4 children. The other 2 children were treated with conservative therapy. In these 2 patients, a sonographic follow-up was performed, showing a progressive reduction in size of the right-sided hyperechoic mass. In conclusion, our study suggests that the presence of the “tethered fat sign” may be an accurate sonographic sign for non-invasive diagnosis of omental infarction in children. (E-mail: mario.diplomatico@gmail.com) © 2020 World Federation for Ultrasound in Medicine & Biology. All rights reserved.

Key Words: Omental infarction, Ultrasound, Children, Omental thickening, Inflammation.

INTRODUCTION

Infarction of the greater omentum is a rare cause of acute abdominal pain in children (only 15% of reported patients are children; more than 85% of reported cases occur in adults) although its incidence has been increasing through the years (Puylaert 1992; Grattan-Smith et al. 2002; Varjavandi et al. 2003; Nubi et al. 2009). Infarction of the greater omentum may mimic acute appendicitis and other acute abdomen diseases because of its non-specific symptoms, such as acute abdominal pain—more often localized in right lower quadrant—nausea, vomiting and fever (Puylaert 1992; Helmrath et al. 2001; Lee et al. 2005; Tonerini et al. 2015). A definitive diagnosis is usually made with histologic examination of the omentum removed during surgery, but a correct and non-invasive diagnosis is also possible, allowing the surgeon to choose between a conservative or a surgical treatment (Loh et al. 2005). Several studies have retrospectively evaluated the role of ultrasound (US) in non-invasive diagnosis of omental infarction in children (Grattan-Smith et al. 2002; Bachar et al. 2005).

Our aim is to report the corresponding sonographic sign specific for omal infarction—not described in the literature to date—which, in our experience (including other cases not discussed in this report), has both a sensibility and a specificity of 100%. Moreover, we want to highlight the role of US for a correct non-invasive diagnosis of omental infarction, differential diagnosis with inflammatory omental thickening and appropriate therapeutic management in children.

MATERIALS AND METHODS

All procedures performed were in accordance with the ethical standards of the institutional and national
research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Data obtained in this study did not interfere with the course of treatment for patients included. Informed consent was obtained from all individual participants included in the study.

From January 2014 to December 2018, a total of 234 children (109 boys and 125 girls, age range 3–15 y) with acute right-sided abdominal pain, admitted to our hospital with a presumptive diagnosis of acute appendicitis, were prospectively evaluated both clinically and sonographically by the same radiologist. In all patients, abdominal US was performed, and the omental fat was always evaluated. In 228 patients, the omental fat resulted to be normal or hyperechogenic, never tethered, and the results affected by other causes of abdominal pain different from omental infarction (such as appendicitis, pancreatitis, urolithiasis and others). Of the remaining 6 children (4 boys and 2 girls, from 9–13 y of age, mean age of 11 y old), 3 showed reduced appetite, nausea and vomiting, and 5 patients had fever. At physical examination, they presented with acute abdominal pain localized in the right quadrant, with peritoneal signs. In 5 of 6 children, we observed a high weight for their age (body mass index >25). Blood tests were normal or revealed a moderate neutrophilia (normal value 1500–8000/μL), with a slight elevation of C-reactive protein (normal value <5 mg/L), and all patients underwent US examination performed by the same radiologist (F. E.), using a convex (3–6 Mhz) and linear transducer (7.5–18 Mhz) (GE Logiq E9, GE, Milwaukee, WI, USA).

RESULTS

In 2 patients, a normal appendix on US excluded the diagnosis of acute appendicitis.

In 4 patients, US failed to identify the appendix. However, other sonographic features of appendicitis (thickening or non-compressibility of the periappendicular peritoneal fat tissue, abscesses and free intra-peritoneal fluid) were not detected.

In all the patients, US showed the presence of a hyperechoic, incompressible, motionless, oval mass localized in the right upper abdominal quadrant, between the umbilicus and the inferior hepatic edge, between the anterior abdominal wall and the ascending or transverse colon, suggesting the diagnosis of omental infarction (Fig. 1). At US, the normal subhepatic fat moves during respiratory movements in relation with the abdominal wall; this is what we call the “sliding fat sign” (Video S1). On the other hand, in case of omental infarction, this subhepatic mass is tethered to the abdominal wall and it is motionless during respiratory excursions; we named this finding the “tethered fat sign” (Fig. 2, Video S2).

The first 4 children underwent surgery, which confirmed the diagnosis of omental infarction (Fig. 3).

In the next 2 patients, because of the presence of the “tethered fat sign” and of mild symptomatology, a conservative management plan (i.e., hydration, analgesics, anti-inflammatories and antibiotics) was adopted (Fragoso et al. 2006). In these patients, a sonographic follow-up was performed every 15 d for 2 mo, showing a progressive reduction in size of the right-sided hyperechoic mass (Fig. 4).

DISCUSSION

US represents an important tool in examining children with acute abdominal pain because it is widely available and, overall, it does not expose pediatric patients to ionizing radiations. Computed tomography (CT) is indicated when symptoms persist and/or normal findings on US do not fit the clinical setting and/or sonographic findings are abnormal. None of our patients underwent CT.

Some US signs suggestive of omental infarction have been already described, both in adults and in children (Puylaert 1992; Varjavandi et al. 2003; Goh and Koong 2006; Tonerini et al. 2015). Omental infarction typically becomes visible as a triangular/oval hyperechoic mass and involves the inferior portion of the right side of the omentum. It is localized between the anterior abdominal wall and the transverse or ascending colon, corresponding to the greater omentum.

US is specific but not sensitive in the diagnosis (Schlesinger et al. 1999). In our series, we observed a sign showing in all the affected patients—a triangular,
hyperechoic subhepatic mass tethered to the abdominal wall, motionless during respiratory excursions—which we called the “tethered fat sign” (Video S3). This finding has not been described in the literature to date, even though it is known in surgery that inflamed omentum is adherent to parietal peritoneum. Unlike normal omental fat moving along the abdominal wall during breathing, in case of omental infarction, the omentum is tethered to the anterior abdominal wall (Fig. 5). This sign is also important in distinguishing omental infarction from other inflammatory conditions (such as acute appendicitis) causing a similar omental thickening, but in which the sliding is preserved.

Infarction of the greater omentum is a rare cause of acute abdominal pain in children (Puylaert 1992; Varjavandi et al. 2003; Nubi et al. 2009; Tonerini et al. 2015). Infarction of the greater omentum can be classified into 2 groups: with or without torsion on vascular axis (Kimber et al. 1996; Grattan-Smith et al. 2002; Loh et al. 2005).

Omental infarction with torsion on its vascular axis can be divided into primary and secondary forms (Kimber et al. 1996; Loh et al. 2005; Cianci et al. 2007; AbdulAziz et al. 2013; Katagiri et al. 2013). Primary forms occur when predisposing abdominal diseases are not found and the etiology is still debated. A well-known predisposing factor in children is obesity because increased fat deposit hinders an adequate blood supply to the growth of omentum, leading to ischemia (Varjavandi et al. 2003; Lee et al. 2005; Coulier 2006; Tsunoda et al. 2012; Katagiri et al. 2013). In addition, fat accumulation can lead to increased weight of the omentum, supporting torsion on its axis. Several authors suggest that primary omental torsion is associated with embryonic variants, such as accessory omentum, bifid omentum and a tongue-like portion of omentum (Varjavandi et al. 2003; Kerem et al. 2005; Coulier 2006; Cianci et al. 2007; Katagiri et al. 2013; Tonerini et al. 2015). These embryonic variants predispose to a kinking of the veins, with progressive impairment of venous and subsequently arterial blood flow, resulting in omental infarction. Vascular congestion after a copious meal is also associated with omental infarction (Puylaert 1992; Grattan-Smith et al. 2002; Cianci et al. 2007; Tonerini et al. 2015).

Conversely, the secondary form of omental infarction with torsion is rare in childhood and it is associated with predisposing intra-abdominal factors, such as omental cysts, hernias, tumors or adhesions (Loh et al. 2005; Cianci et al. 2007; AbdulAziz et al. 2013; Katagiri et al. 2013; Tonerini et al. 2015). Both primary and secondary torsion compromise vascularization, leading to venous stasis and thrombosis, congestion and subsequent impairment of arterial blood flow with necrosis and omental infarction (Cianci et al. 2007; Tonerini et al. 2015).

Omental infarction without torsion has been reported secondary to hypercoagulability states, vasculitis, trauma, pancreatitis and compression by abdominal masses (Varjavandi et al. 2003; Loh et al. 2005). However, this classification is not clinically relevant because clinical presentation and treatment are the same in both cases (Grattan-Smith et al. 2002; Coulier 2006).

Symptoms are usually not specific. Most children experience acute right-sided abdominal pain. In some occasions, children show fever and gastro-intestinal symptoms, such as nausea and vomiting (Helmrath et al.
Laboratory tests may be normal or reveal a moderate neutrophilia with a slight elevation of the C-reactive protein. In our study, all children with acute abdominal pain underwent US. An increasing incidence of obesity is an established risk factor for developing omental infarction (Theriot et al. 2003; Rimon et al. 2009; Estevão-Costa et al. 2014; McCusker et al. 2018). This aspect is reflected in our series of patients because most of them were overweight or obese. It has been reported elsewhere that omental infarction generally affects boys more often than girls and it is usually right sided (Kimber et al. 1996; Helm-rath et al. 2001; Nubi et al. 2009; Rimon et al. 2009). This finding is consistent with our experience, and it explains why omental infarction could be confused with appendicitis. Many theories exist about the exact pathogenesis of omental infarction, including anatomic predisposition of the right epiploic vessels, venous engorgement, overeating, hypercoagulable states, coughing or sudden changes in position combined with an excessively large, pendulous greater omentum, as observed in intra-abdominal obesity (Coulier 2010); however, the precise etiology remains unknown.

Omental infarction may be associated with intraperitoneal free serosanguinous fluid (Varjavandi et al. 2003; Lee et al. 2005; Cianci et al. 2007; Tonerini et al. 2015). Another less specific sign of inflammation is the parietal thickening of an adjacent segment of small bowel, which can be incorporated into the inflammatory mass, making the diagnosis more difficult (Lee et al. 2005; Tonerini et al. 2015).

The literature describes two types of treatment: conservative medical treatment and surgical intervention (Fragoso et al. 2006; Itenberg et al. 2010; Occhionorelli et al. 2014). Conservative treatment includes appropriate hydration, analgesics, anti-inflammatory drugs and prophylactic antibiotics (Puylaert 1992; Coulier 2006; Goh and Koong 2006; Estevão-Costa et al. 2014); however, this conservative treatment has long been considered to be associated with complications, such as abscesses and adhesions induced by the persistence of necrotic tissue in the abdomen (Goti et al. 2000; Itenberg et al. 2010; AbdulAziz et al. 2013; Estevão-Costa et al. 2014; Occhionorelli et al. 2014).

Therefore, historically, surgical resection of the affected omentum has been considered the treatment of choice (Goti et al. 2000; Grattan-Smith et al. 2002; Varjavandi et al. 2003; Katagiri et al. 2013; Occhionorelli et al. 2014). Many surgeons consider laparoscopy the surgical approach of choice because it leads to complete resolution of symptoms and the post-operative recovery is more rapid, with discharge after 3 d (Grattan-Smith et al. 2002; Varjavandi et al. 2003; Lee et al. 2005). Moreover, an incision in the lower right quadrant, normally performed for an appendicectomy, may not be adequate to expose the infarcted omentum.

Conservative management is reported as the treatment of choice in cases where the diagnosis is...
confirmed by US or CT or for hemodynamically stable children (Fragoso et al. 2006; Cianci et al. 2007; AbdulAziz et al. 2013; Estevão-Costa et al. 2014; Occhionorelli et al. 2014) without adverse clinical features (Breda Vriesman et al. 1999; Wertheimer et al. 2014). On radiologic follow-up, infarcted omentum is characterized by a progressive reduction in the size of the right-sided hyperechoic mass because of fibrosis and atrophy (Cianci et al. 2007; AbdulAziz et al. 2013; Occhionorelli et al. 2014). Laparoscopic surgical treatment seems to be reserved for children with uncertain diagnosis, unclear symptoms, non-specific abdominal pain, severity of clinical presentation and complications such as omental abscess and bowel obstruction (Goti et al. 2000; Nubi et al. 2009; AbdulAziz et al. 2013).

In our cases, the first 4 children with sonographic diagnosis of omental infarction underwent surgery, as suggested by in some of the literature, because they presented with a severe abdominal pain, with initial signs of peritonitis. In the last 2 patients, we identified the “tethered fat sign” and therefore we considered the diagnosis of omental infarction. Furthermore, these patients showed a quick improvement in clinical conditions without adverse clinical features and they were clinically stable. For this reason, encouraged by the more recent literature, we prescribed conservative management (i.e., hydration, analgesics, anti-inflammatories and prophylactic antibiotics) (Kerem et al. 2005; Goh and Koong 2006; Estevao-Costa et al. 2014; Tonerini et al. 2015; Lindley and Peyser 2018).

Both patients who received conservative management showed complete clinical and laboratory remission. In these patients, a sonographic follow-up was performed every 15 d for 2 mo, showing fibrosis and a progressive reduction in size of the right-sided hyperechoic mass. They did not return to our hospital for onset of complications after the end of the sonographic follow-up.

Our study highlights the usefulness of US in the diagnosis of omental infarction in children, as already reported in literature (Schlesinger et al. 1999; Helmrath et al. 2001; Wertheimer et al. 2014). We want to highlight the validity of the US “tethered fat sign” (unreported in the literature to date), which can really make the difference in the diagnostic process, leading to an almost certain diagnosis of omental infarction without the need to submit pediatric patients to second-level investigations.

Therefore, we emphasize the importance and the diagnostic value of the “tethered fat sign” in the sonographic non-invasive diagnosis of the omental infarction to choose between a conservative treatment or the most suitable surgical approach.

CONCLUSION

Omental infarction is a rare disease in children and its clinical presentation may mimic acute appendicitis. In obese children with acute right lower abdominal pain, no fever, few prodromal symptoms and normal or a slightly increased white blood cell count, the diagnosis of omental infarction should be considered (Varjavandi et al. 2003; Wertheimer et al. 2014; Tonerini et al. 2015). US is a quick, safe, inexpensive and widely available diagnostic method to evaluate the presence of omental infarction. Our series suggests that US may be an accurate modality for a non-invasive pre-operative differential diagnosis between inflammatory conditions (as appendicitis) and omental infarction (Fragoso et al. 2006; Estevao-Costa et al. 2014; Lindley and Peyser 2018; Phalke et al. 2018). Even though not immediately visible (it must be searched for), the presence of the “tethered fat sign” appears highly suggestive of omental infarction. Obviously, this sign needs to be confirmed in a more consistent number of patients but, to date, it has been observed in all our patients. It is very important to recognize this sign to make the appropriate decision between surgical or conservative management.

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Conflict of interest disclosure—The authors declare that they have no conflict of interest.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.ultrasmedbio.2020.01.003.

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