ORIGINAL RESEARCH PAPER

Volume-7 | Issue-4 | April-2018 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

# INTRAPERITONEAL FOCAL FAT INFARCTION : CASE REPORTS OF TWO VARIANTS OF THIS ENTITY

General Surgery

**Dr. Adheesh Goriwal** — DNB General Surgery

**Dr. Kalpit Goriwal*** — Dnb General Surgery, M.s. Office Indraprastha Apollo Hospitals Sarita Vihar New Delhi 110076 India * Corresponding Author

**Dr. Rahul Patel** — M.S(GENERAL SURGERY)

## ABSTRACT

Intraperitoneal Focal Fat Infarction is a broader umbrella term which includes two disease entities viz. Epilploic Appendagitis and Omental Infarction. The importance of differentiating between the two entities lies in the fact that management of both is contrasting. We here present 2 case reports where we differentiated between the two disease entities and both cases were managed accordingly with satisfactory outcomes.

## KEYWORDS

omental infarction, segmental infarction, idiopathic infarction, epiploic appendagitis, intraperitoneal focal fat infarction, greater omentum.

**INTRODUCTION:** ***Epiploic appendagitis***, also known as appendicitis epiploica, hemorrhagic epiploitis, epiplopericolitis, or appendagitis, is a benign and self-limited condition of the epiploic appendages. Epiploic appendages [EA] are small outpouchings of fat-filled, serosa-covered structures present on the external surface of the colon projecting into the peritoneal cavity. Each appendage encloses small branches of the circular artery and vein that supply the corresponding segment of the colon. Subserosal lymphatic channels either terminate in a lymph node within an appendage or loop through its base en route to mesenteric nodes. They have three morphologically different types: stalked appendices and others that are attached with their base in longitudinal and vertical direction to the colon axis. Although they are located throughout the colonic wall, occasionally they have also been found in the small bowel and in the parietal peritoneum. Their function is still controversial, they are presumed to serve a protective and defensive mechanism similar to that offered by the greater omentum and may have a role in colonic absorption. They may also act as a cushion, protecting colonic blood supply during peristalsis. Although little has been written about epiploic disorders (inflammation, torsion, necrosis, etc.) Inaccurate diagnosis can lead to unnecessary hospitalizations, antibiotic therapy, and surgical intervention.They occur more frequently than expected. Thus, they should be kept in mind.

***Omental Infarction*** is a rare differential diagnosis of acute abdomen. Omental Infarction per se is due to vascular compromise of the greater omentum.

***Primary Omental Infarction*** occurs with staggering predominance in the right lower quadrant of abdomen, medial to ascending colon or cecum.

The vascular compromise occurs along the right edge of the greater omentum due to the tenuous blood supply. Sometimes, it can occur due to kinking of venous channels in the inferior part of the greater omentum in the pelvis. When omental torsion is the cause of infarction, both, arterial and venous compromise occur. When omentum infarction occurs exclusive of omental torsion, it is known as Primary Idiopathic Segmental Infarction.

**Secondary Omental Infarction** may be post-operative, due to omental infalmmation or following an abdominal trauma.

Obesity has been suggested as a precipitating factor for both, primary and secondary omental infarction. Other strong associations include adult age (peak being 40-50 yrs age group; less than 15% cases occur in pediatric population), male sex, heavy exertion and sudden change in body position.

The exact etiopathogenesis of primary omental infarction is still elusive, however, redundant omentum, vascular congestion, increased intra-abdominal pressure and hyperperistalsis due to over eating have been implicated.

Clinical Picture is hazy and is predominated by sudden onset diffused or right lower abdominal pain and tenderness, absence of fever and other gastrointestinal symptoms.

Diagnosis is based on high index of suspicion and imaging studies (USG and CT scan). Ultrasonography abdomen will demonstrate non-compressible focal area of increased echogenecity in the omental fat. Computed Tomography will reveal focal area of fat stranding, a fluid cavity, hyperdense peripheral halo and swirling of omental vessels in omental torsion.

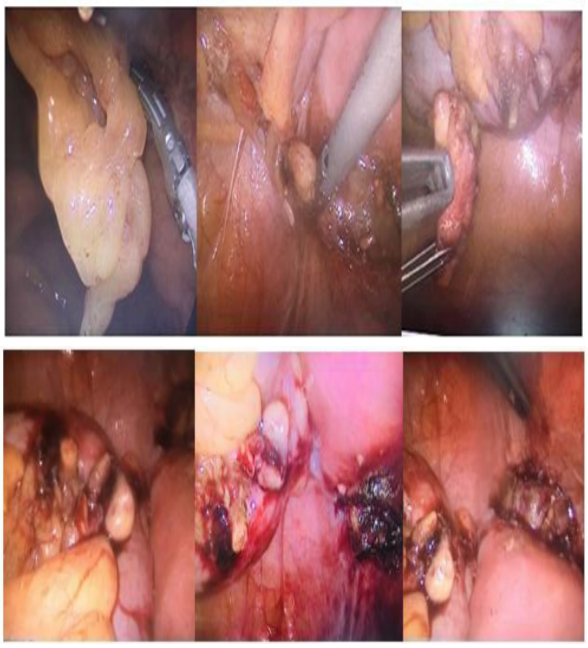
Treatment of idiopathic omental infarction is predominantly on conservative grounds and in majority of cases, this is what suffices for the patient.

Important differentials for the pathology include epiploic appendagitis, acute appendicitis, diverticulitis and mesenteric panniculitis.

**CASE REPORT - I**

A 41-year-old female was admitted to the Indraprastha Apollo Hospital New Delhi, complaining of severe colicky pain in the left lower quadrant on and off in nature for the last 2 days. Her past medical history was unrewarding except for controlled hypothyroidism. At physical examination, the patient was tachycardic (110 beats per minute), and exquisite tenderness and rebound were found in the left lower quadrant. White blood cell count was 12,600, and the rest of the laboratory tests were within normal limits. Abdominal X-rays showed a dilated sigmoid colon, and a CT scan suggestive of epiploic appendagitis.

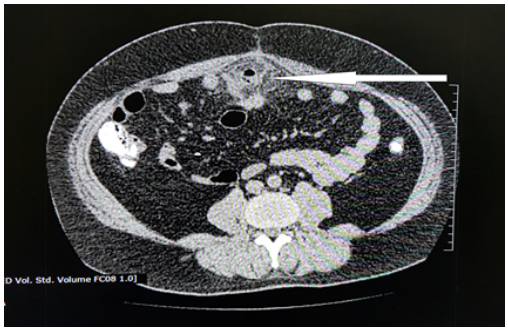
An emergency diagnostic laparoscopic procedure was performed under general anesthesia. After establishing the pneumoperitoneum, a 10-mm 0° laparoscope was introduced through the umbilicus. The peritoneal cavity exploration revealed adhesions inflamed left sided epiploic appendages adhere to uterus and pelvic wall present and scanty yellowish exudate in the left pelvic area. Two additional 5-mm ports were placed in the right lower quadrant and in the right flank, and intestinal forceps were used to manipulate the bowel gently, adhesions were released and excision of inflamed epiploic appendages were done, Its pedicle was cauterized using a bipolar electrocautery, Excised specimen was sent for histopathological examination which subsequently confirmed the diagnosis of epiploic appendagitis. The following morning, the patient passed flatus and tolerated a liquid diet followed by soft and normal diet. She was discharged 48 hours later and remains asymptomatic.



**CASE REPORT - II**

Here we present the case of a 43 year old gentleman who is obese (BMI-32.10) and a known case of diabetes mellitus type II and hypertension since 10 years. He presented to us with the chief complaint of diffuse pain abdomen and discomfort of 1 day duration. There was no associated complaint of fever, vomiting, loose stools or difficulty in micturition. No significant drug or family history was noted. The patient is obese with a BMI of 32.10 and on interrogation, was found to have a voracious apetite with average daily calorie intake of around 4000kcals.

The patient consumes around 40gm of alcohol each day(by proof spirit calculations). On examination, the abdomen was mildly distended with vague lump palpable in periumbilical region and moderate tenderness in the same region. The patient was advised CECT Abdomen which demonstrated area of fat stranding intraperitoneally in the region of umbilicus. The area measured around 4 x 3 cm. There was presence of a hollow cavity in the centre of the lump, most probably due to ensuing necrosis due to infarction. A diagnosis of Omental Infarction was made and patient was managed conservatively on antibiotics, analgesics and close monitoring. The patient responded satisfactorily to the management protocol and was discharged with advise of weight reduction and improving eating habits. At 8 weeks post discharge patient was actively followed up and was found to be completely asymptomatic during this period.




**CECT ABDOMEN**
**Arrow points at the fat stranded area**
**A hollow cavity is appreciable in the centre of the pathology**

**DISCUSSION:** Epiploic appendages [EA] First anatomically described in 1543 by Vesalius, they were not given any surgical significance until 1853 when Virchow suggested that their detachment might be a source of free intraperitoneal bodies, The term Epiploic appendagitis was introduced by Lynn et al. in 1956 and describes an uncommon diagnosis which is associated with rapid onset of localized left or right lower quadrant pain. Due to the lack of pathognomonic clinical features, the diagnosis is difficult, Acute inflammation, spontaneous torsion, fat necrosis, infarction, and calcification occur in the epiploic appendages, as well as enlargement due to lipomas, malignant tumors (including metastasis), and incarceration in hernias. The incidence of torsion and necrosis is almost impossible to estimate. Aronsky et al reported that abdominal fat tissue necrosis (including the omentum) occurs in 1.1% of patients with abdominal pain. Some authors have found the disease to be more common during the 4th and 5th decades of life, while others cannot find a preferred age group. A slight male preponderance has been described in a review by Carmichael and Organ. Primary epiploic appendagitis with subsequent necrosis is caused by torsion with compromise of its blood supply or by venous thrombosis of its draining system. It tends to occur in the sigmoid colon in more than 40% of the cases. Factors such as obesity and a narrow epiploic appendix base have been implicated in the etiology of torsion, whereas exertion has been related to events of venous thrombosis. Epiploic appendagitis has no specific manifestations. Focal abdominal pain is the most important symptom, and, depending on the localization of the affected appendage, the clinical picture might resemble that of colonic diverticulitis, acute appendicitis, a gynecological disorder, or even acute cholecystitis. As Shvetzov states referring to the torsion of an EA, ―It occurs under the mask of other emergencies.‖ Abdulzhavadov describes ―two new characteristic symptoms of this disease‖: 1) pain appearing or intensifying when the abdomen is thrust forward and in mild tapping on the healthy side of the anterior abdominal wall with the fingertips, and 2) intensification of pain when the skin fold on the abdomen is pulled upward. This, of course, needs to be confirmed by others. Although pain is acute in most cases, Chatziioannou et al have reported a patient with abdominal pain of three weeks' duration that was caused by a necrotic EA. In the majority of patients, there are no other significant signs or symptoms, although nausea, vomiting, fever and a palpable mass have been mentioned frequently. Asymptomatic infarctions are sometime found incidentally as loose bodies at laparotomy performed for other reasons. Epiploic appendagitis has been, until recently, exceptionally diagnosed before laparotomy due to the fact that the clinical picture is non-specific and confusing. However, Shvetsov et al claim they have been able to diagnose two-thirds of their patients on clinical findings Primary Idiopathic Omental Infarction is encountered in medical practice with increased prevalence due to increased incidence as a result of lifestyle changes and better imaging modalities available. A typical patient will be a middle age obese male presenting with acute onset pain abdomen. On imaging studies, the pathology is diagnosed. A high index of suspicion is required to diagnose this rare disease entity. Diagnosis is important because it will decide the management which is predominantly conservative.

**CONCLUSION:** The importance of differentiating between the two disease entities under the broader term Intraperitoneal Focal Fat Infarction lies in the fact that management protocols for both are contrasting.

Epiploic Appendagitis warrants surgical management whereas the basis for treating omental infarction is on conservative lines.

Both disease entities can be equated to two sides of a coin where a misdiagnosis will invert the management and thus results.

Misdiagnosis will lead to unwarranted surgical exploration in primary omental infarction and useless waiting in Epiploic appendagitis.

Therefore, there is a fine line between the heads and tails of this coin which can make the clinician win the match or lose it.

**REFERENCES**

[1]. Carmichael DH, Organ CH., Jr Epiploic disorders. Arch Surg. 1985;120:1167–1172 [PubMed]
[2]. Hansen HH, Heine H. Blood supply and histophysiology of the appendices epiploicae. Lagenbecks Arch Chir. 1976;340:191– 197[PubMed]
[3]. Mittal VK, Pierce AK, Priestley JC, et al. Infarcted small-bowel appendiceepiploica: a cause of acute abdomen. Am J ProctolGastroenterol Colon Rect Surg. 1981;32:23–24 [PubMed]
[4]. Bundred NJ, Clason A, Eremin O. Torsion of an appendix epiploica of the small bowel. Br J ClinPract. 1986;40:387. [PubMed]

[5]. Polukhin SI, Letemin GG, Povorozniuk VS. Torsion of epiploic appendages of the parietal peritoneum. VestnKhirIm II Grek. 1990;145:50 [PubMed]
[6]. Rioux M, Langis P. Primary epiploic appendagitis: clinical, US, and CT findings in 14 cases. Radiology. 1994;191:523–526 [PubMed]
[7]. Molla E, Ripolles T, Martinez MJ, Morote V, Rosello-Sastre E. Primary epiploic appendagitis: US and CT findings. EurRadiol. 1998;8:435–438 [PubMed]
[8]. Ghahremani GG, White EM, Hoff FL, Gore RM, Miller JW, Christ ML. Appendices epiploicae of the colon: radiologic and pathologic features. Radiographics. 1992;12:59–77 [PubMed]
[9]. Metrevili VV, Gondzhilashvili GV, Kuzanov EI, Chkhikvadze TF. Acute diseases of the appendices epiploicae. Khirurgiia (Mosk). 1989;(4):99–101 [PubMed]
[10]. Ramdial PK, Singh B. Membranous fat necrosis in appendices epiploicae. A clinicopathological study. Virchows Arch. 1998;432:223– 227[PubMed]
[11]. Vesalius A. De humaniscorporisfabricalibriseptem [Title page: Andreae VesaliiBruxellensis, scholaemedicorumPatauinaeprofessoris De humani corporisfa bricalibriseptem] Basileae [Basel, Switzerland]: Ex officina JoannisOporini. p. 1543.
[12]. Vinson DR. Epiploic appendagitis: a new diagnosis for the emergency physician. Two case reports and a review. J Emerg Med. 1999;17:827–32. doi: 10.1016/S0736-4679(99)00090-6. [PubMed][Cross Ref]
[13]. Lynn TE, Dockerty MB, Waugh JM. A clinicopathologic study of the epiploic appendages. SurgGynecol Obstet. 1956;103:423–33. [PubMed]
[14]. Habib FA, McAleese P, Kolachalam RB. Laparoscopic approach to the management of incarcerated hernia of appendices epiploicae. SurgLaparoscEndosc. 1998;8:425–428 [PubMed]
[15]. Aronsky D, Z'graggen, Banz M, Klaiber C. Abdominal fat tissue necrosis as a cause of acute abdominal pain. Laparoscopic diagnosis and therapy. SurgEndosc. 1997;11:737–740 [PubMed]
[16]. Fieber SS, Forman J. Appendicesepiploicae: clinical and pathological considerations. Arch Surg. 1953;66:329–338 [PubMed]
[17]. Pines B, Rabinovitch J, Biller SB. Primary torsion and infarction of the appendices epiploicae. Arch Surg. 1941;42:775–787
[18]. Mazza D, Fabiani P, Casaccia M, Baldini E, Gugenheim J, Mouiel J. A rare laparoscopic diagnosis in acute abdominal pain: torsion of epiploic appendix. SurgLaparoscEndosc. 1997;7:456–458 [PubMed]
[19]. Vlahakis E. Torsion of an appendix epiploica of the ascending colon. Med J Aust. 1973;2:1148–1149[PubMed]
[20]. Brady SC, Kliman MR. Torsion of the greater omentum or appendices epiploicae. Can J Surg. 1979;22:79–82 [PubMed]
[21]. Shvetsov SK, Bol'shakov IA. Torsion of the colonic epiploic appendages. Khirurgiia (Mosk). 1992;2:76–80 [PubMed]
[22]. Shamblin JR, Payne CL, Soileau MK. Infarction of an epiploic appendix. South Med J. 1986;79:374–375 [PubMed]
[23]. McGeer PL, McKenzie AD. Strangulation of the appendix epiploica: a series of 11 cases. Can J Surg. 1960;3:252–258
[24]. Rao PM, Rhea JT, Wittemberg J, Warshaw AL. Misdiagnosis of primary epiploic appendagitis. Am J Surg. 1998;176:81–85 [PubMed]
[25]. LambreH,ManzurR,OxenghendlerG,etal. Computedtomographyofa cute primary epiploic appendicitis. ActaGastroenterolLatinoam. 1998;28:337–338 [PubMed]
[26]. Shirokikh VV. Torsion of an epiploic appendage of the trans-verse colon simulating acute cholecystitis. VestnKhir. 1972;109:102– 103 [PubMed]
[27]. Abdulzhavadov IM. The symptoms of diseases of the epiploic appendages of the large intestine. Khirurgiia (Mosk). 1992. February;(2):80–83 [PubMed]
[28]. Chatziioannou AN, Asimacopoulos PJ, Malone RS, Pneumaticos SG, Safi HJ. Torsion, necrosis, and inflammation of an epiploic appendix of the large bowel: a diagnostic and therapeutic dilemma. South Med J. 1995;88:662–663 [PubMed]
[29]. Levret N, Mokred K, Quevedo E, Barret F, Pouliquen X. Primary epiploic appendicitis. J Radiol. 1998;79:667–671 [PubMed]
[30]. Diaco JF, Diaco DS, Brannan AN. Endoscopic removal of an infarcted appendix epiploica. J Laparoendosc Surg. 1993;3:149–
151 [PubMed]
[31]. Cueto J, Diaz O, Garteiz D, Rodrvguez M, Weber A. The efficacy of laparoscopic surgery in the diagnosis and treatment of peritonitis. Experience with 107 cases in Mexico City. SurgEndosc. 1997;11:366–370 [PubMed]
[32]. Memon MA, Fitzgibbons RJ., Jr The role of minimal access surgery in the acute abdomen. SurgClin North Am. 1997,77:1333– 1353 [PubMed]
[33]. Chung RS, Diaz JJ, Chari V. Efficacy of routine laparoscopy for the acute abdomen. SurgEndosc. 1998;12:219–222 [PubMed]
[34]. Salky BA, Edye MB. The role of laparoscopy in the diagnosis and treatment of abdominal pain syndromes. SurgEndosc. 1998;12:911– 914[PubMed]
[35]. Navez B, Tassetti V, Scohy JJ, et al. Laparoscopic management of acute peritonitis. Br J Surg. 1998;85:32–36 [PubMed]
[36]. Buell, K. G., Burke-Smith, A., Patel, V., & Watfah, J. (2017, December 13). Omental Infarction: The Great Impersonator.
[37]. Coulier, B. (n.d.). Spontaneous and rapid healing of massive symptomatic postoperative right-sided infarction of the greater omentum.
[38]. Criado, I., Andrino, N. F., & López-Dóriga, P. (n.d.). [Omental infarction: An unusual cause of acute abdomen].
[39]. Sánchez-López-Gay, J., Becerra-Almazán, J. M., Reyes-Aguilar, R., Rodríguez-Barón, B., & Navarro-Duarte, J. C. (2017, October). [Cause of acute non-surgical abdomen: omental infarction].
[40]. Sun, X. W., Luo, B., & Lin, H. W. (n.d.). A rare case of acute primary omental infarction.
[41]. Patel, S. K., Kumar, A., Setya, A., Deo, A., & Raj, V. (2016, January). Primary omental infarction: A confusing diagnosis.
[42]. Arigliani, M., Dolcemascolo, V., Nocerino, A., Pasqual, E., Avellini, C., & Cogo, P. (2016, September). A Rare Cause of Acute Abdomen: Omental Infarction.
[43]. Aiyappan, S. K., Ranga, U., & Veeraiyan, S. (2015, December). Omental infarct mimicking acute pancreatitis.
[44]. Amo, R., De, J., Loza, A., Santos, F., Sánchez-Ocaña, R., & Arenal, J. J. (2015, November). Infarction of the greater omentum. Case report.
[45]. Ong, W. M., Matheson, J., Chandra, R., & Stella, D. L. (2018, January). Omental infarction: a case of a whole omental infarct.
[46]. Walia, R., Verma, R., Copeland, N., Goubeaux, D., Pabby, S., & Khan, R. (2014, July 08). Omental Infarction: An Unusual Cause of Left-Sided Abdominal Pain.
[47]. Armas, A. L., Pradillos, J. M., Rivera, L. L., Perri, L. E., García, M., Rodríguez, P., . . . Bautista, A. (2014, July). [Conservative treatment for omental infarction].
[48]. Smolilo, D., Lewis, B. C., Yeow, M., & Watson, D. I. (n.d.). Omental infarction mimicking cholecystitis.
[49]. Tonerini, M., Calcagni, F., Lorenzi, S., Scalise, P., Grigolini, A., & Bemi, P. (2015, August). Omental infarction and its mimics: imaging features of acute abdominal conditions presenting with fat stranding greater than the degree of bowel wall thickening.
[50]. Sánchez, P. A., López, V., Febrero, B., Ramírez, P., & Parrilla, P. (n.d.). Omental infarction: Surgical or conservative management?
[51]. Wertheimer, J., Galloy, M. A., Régent, D., Champigneulle, J., & Lemelle, J. L. (2014, March). Radiological, clinical and histological correlations in a right segmental omental infarction due to primary torsion in a child.
[52]. Wang, W., Wang, Z. J., Webb, E. M., Westphalen, A. C., Gross, A. J., & Yeh, B. M. (n.d.). Omental infarction preceded by anatomically upturned omentum.