Vermiform Appendix And Acute Appendicitis
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Introduction
The Vermiform appendix present only in human beings, certain arthropod apes and the wombat (a nocturnal, burrowing Australian marsupial) was probably first noted as early as the Egyptian civilization (3000 B.C). During the mummification process, abdominal parts were removed and placed in coptic jars with inscriptions describing the contents as “worm of the intestines” were discovered (1). The Vermiform appendix is considered by most to be a vestigial organ, its importance in surgery due mainly to its propensity for inflammation that results in the clinical syndrome known as Acute appendicitis. Acute appendicitis is the most common cause of “acute abdomen” in young adolescents and appendectomy is often the first major procedure performed by a Surgeon in training (2,3,4). Variations in the position of the appendix, age of the patient and degree of inflammation make the clinical presentation of appendicitis notoriously inconsistent. Misdiagnosis in different age groups is from 10 to 33% (5). Despite extraordinary advances in modern radiographic imaging and diagnostic laboratory investigations, the diagnosis of appendicitis remain essentially clinical requiring a mixture of observations, clinical acumen and surgical sense.

Anatomy: Embryologically, the appendix is continuation of the caecum and is first delineated during the fifth month of gestation. The appendix does not elongate as rapidly as the rest of the colon, thus forming a worm like structure (2,3,6). The origin of appendix is about 2.5 cms below the ileocaecal valve from the posteromedial aspect of Caecum. It is the only organ in the body that has no constant anatomic position and only constant feature is its mode of origin from the caecum where it arises from the site where three teniae coli coalesce. It varies considerably in length from 1 to 25 cms; but mostly it ranges between 5-10 cms. The appendix may be positioned as preileal, postileal, paracolic, retrocaecal, subcaecal, pelvic and rarely subhepatic (2). The incidence as per position of appendix has been reported as 65.28% for retrocaecal, 31.01% pelvic, 2.26% subcaecal, 1% preileal and 0.4% for right paracolic/ postileal (7). At routine CT Scan it was observed that retroileal appendix appears to occur much more frequently in living subjects (8,9). Absent appendix is too rare a condition and till date only 68 cases have been reported in the literature (10). Similarly duplication of appendix is also a rare anomaly and fewer than 100 cases have been reported (11-13).

Appendicular artery represents entire arterial supply of the organ an end artery arising from Ileocolic artery. Thrombosis of this artery in acute appendicitis inevitably results in gangrene and subsequently perforation. Veins from Appendix drains into the ileocolic vein which empties into superior mesenteric vein. A variable number of slender lymphatic channels traverse the mesoappendix to empty into the ileocaecal nodes.

Etio-pathogenesis: Obstruction of the lumen is the dominating factor in acute appendicitis. Fecoliths are usual cause of appendiceal obstruction. Less common is hypertrophied tissue, inspissated barium from previous X-rays, vegetable, fruit seed, worms (Entrobius vermicularis, Balantidum coli, Schistosoma haematobium) (14-15). Due to this blockade closed loop obstruction results and the secretions continuously produce distension that stimulates nerve endings of visceral afferent pain fibers producing vague, dull, diffuse pain in the mid abdomen or lower epigastrium. Distension also stimulates peristalsis that results into crampy abdominal pain superimposing visceral pain. Distension continues and
pressure in organ increases and exceeds venous pressure, thereby, occluding capillaries and venules but arterial inflow continues, resulting in engorgement and vascular congestion. Distension of this magnitude usually causes reflex nausea and vomiting and diffuse visceral pain become more severe. The inflammatory process involves the serosa of appendix, thereby, involving parietal peritoneum that produces tenderness as well as rebound tenderness. Progressive distension increases arterial pressure and the area with poorest blood supply suffers most; ellipsoidal infarcts develop in the antimesentric border. As the distension, bacterial invasion, compromise of vascular supply and infarction progress, perforation occurs through one of the infarcted areas on antimesentric border.

**Pathology:** The menace of acute appendicitis lies in the frequency with which the peritoneal cavity is infected from this focus, either by perforation or by transmigration of bacteria through the appendicular wall. The greater omentum attempts to wall off the spread of peritoneal invasion while violent peristalsis from ingested purgatives tends to spread it.

**Non-obstructive acute appendicitis:** The inflammation commences either in mucous membrane on in lymph follicles and terminates either as resolution, ulceration, suppuration, fibrosis or gangrene. Infection progresses rapidly once it reaches submucous tissue. The organ becomes turgid, dusky red and haemorrhage occurs into the mucous membrane. The vascularity of the distal part of appendix is often in jeopardy as the artery is intramural and liable to occlusion by inflammation/thrombosis thereby, leading to gangrene of the tip. The non-obstructive appendicitis progress slowly allowing protective barrier to develop and at times inflammation do not progress beyond the mucosal lining (Catarrhal appendicitis) and attack goes off without sequel.

**Obstructive acute appendicitis:** 2/3rd cases belong to this group. The obstruction is either in the lumen (Faecolith, foreign body, Parasite) or in the wall (invariably inflammatory but may be direct occlusion by carcinoma of caecum) or outside the wall (adhesions/kinking). Products of inflammation proceed more rapidly and more certainly to gangrene/perforation. Within 12-18 hrs appendix distal to the obstruction becomes gangrenous. Perforation occurs most commonly at the site of impacted faecolith before protective adhesions have had time to form. The escaping purulent and gaseous contents are under high pressure and early widespread peritonitis is liable to ensue.

**Factors encouraging Progression of Inflammation:**
(i) Very young/old. (ii) Immunosupressive agents.
(iii) Free lying appendix. (iv) Presence of faecolith.
(v) Purgatives/enema. (vi) Impaired blood supply.

**Clinical Features:** The clinical features are more pronounced and progressive in obstructive than non-obstructive acute appendicitis. Pain that starts from periumblical area/epigastrium shifts to right iliac fossa in due course of time. Coughing causes localized pain in RIF in acute appendicitis and is absent in renal disease. Once parietal peritoneum is involved it produces more intense, constant and localized somatic pain that shifts and has changed its character. This classical visceral-somatic sequence is seen in only 50% of patients of acute appendicitis as early signs and symptoms depend upon the location of the tip of the appendix that is highly variable (2-4). In early appendicitis, the patient is initially afebrile or has a low-grade fever. Appendicitis in elderly is difficult problem resulting in incorrect diagnosis as well as high rate of perforation (16). High fever is associated with a perforated appendicitis (17). The clinical symptom/signs are detailed in table I.

**Differential Diagnosis:** Although acute appendicitis is the most common acute abdomen requiring surgical intervention yet in the absence of definite supportive diagnostic investigation it requires to be differentially diagnosed from a variety of clinical conditions. Even the most experienced physicians and surgeons are not able to diagnose appendicitis 100% of the times. Table II & III.
Table II: D/D of Acute appendicitis as per anatomical variations.

Table III: D/D as per age.

<table>
<thead>
<tr>
<th>S. No</th>
<th>Attic/Upper storey</th>
<th>Ground floor</th>
<th>Basement</th>
<th>Backyard/Blatt</th>
<th>Others</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Tonsillitis</td>
<td>Perforated ulcer</td>
<td>Enteritis</td>
<td>Salpingitis</td>
<td>Urine colic</td>
</tr>
<tr>
<td>2</td>
<td>Pneumonia</td>
<td>Acute cholecystitis</td>
<td>Non-specific mesentric lymphadenitis</td>
<td>Ileocaecal perforation</td>
<td>Acute Pyelonephritis</td>
</tr>
<tr>
<td>3</td>
<td>Malignancy</td>
<td>Cyclical vomiting</td>
<td>Int. obstruction</td>
<td>Rt. Ovarian torsion</td>
<td>Spinal tuberculosis</td>
</tr>
<tr>
<td>4</td>
<td>Meckel’s diverticulum</td>
<td>Ruptured ovarian follicle (Mittelschmerz)</td>
<td>Blood diabetes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Terminal ileitis</td>
<td>Caecum</td>
<td>Henoch-Schonlein purpura</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Sigmoid diverticulitis</td>
<td>Ac. Pancreatitis</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>7</td>
<td>Rectus sheath hematoma</td>
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Table IV: Plain X-ray Abdomen and Barium Enema Findings in Acute appendicitis.

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<tr>
<th>S. No</th>
<th>Plain X-ray Findings</th>
<th>Barium Enema Findings</th>
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<tbody>
<tr>
<td>1</td>
<td>Fluid levels localized to caecum/terminal ileum</td>
<td>Persistent non visualisation of appendix</td>
</tr>
<tr>
<td>2</td>
<td>Localised ileus with gas in caecum/ascending colon</td>
<td>Partially visualised appendix</td>
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<tr>
<td>3</td>
<td>Increased soft tissue density in lower right quadrant</td>
<td>Pressure defect on the caecum</td>
</tr>
<tr>
<td>4</td>
<td>Blurring of right flank stripe</td>
<td>Irritable caecum/terminal ileum on screening</td>
</tr>
<tr>
<td>5</td>
<td>Faecolith in right iliac fossa</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Blurring of Rt. Psoas shadow</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Free intraperitoneal gas</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Deformity of the caecal gas shadow</td>
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Investigations: Although the diagnosis of acute appendicitis invariably is clinical yet it may be supported by exclusion after doing some investigations. No test yet devised that is 100% diagnostic. The only diagnostic procedure short of open exploration is diagnostic laparoscopy.

1. WBC: In 3/4th cases of acute appendicitis TLC is more than 12,000 (18). Tc- labeled WBC Scan has reported sensitivity of 98% and specificity of 95%. However, time constraint and availability is an issue (19-20).

2. Urine examination: Though normal in many instances yet it may be showing pyuria/microscopic haematuria. If the surgeon is satisfied that appendicitis cannot be ruled out, operation under such circumstances is entirely justified; that may show inflamed appendix adherent to right ureter/bladder.

3. Radiography: Finding as proposed by various authors on plain X-Ray abdomen as well as barium meal follow through are listed in table IV (3, 21, 22). It is pertinent to mention here that emergency barium enema is practiced in USA only and not in any other country.

4. Ultrasonography of abdomen: More useful for differential diagnosis. With experience one may find acutely inflamed appendix as non-compressible, aperistaltic, tubular structure with a central dilated lumen surrounded by an inner echogenic mucosal layer and outer oedematous wall that shows few echoes (23-25).

Treatment: The treatment of acute appendicitis is appendectomy. In the absence of appendicular mass, appendix should be removed at the earliest as the operative mortality is almost negligible but it may increase several fold if operation is delayed. The appendectomy may be either open or laparoscopic. Unlike Laparoscopic cholecystectomy, laparoscopic appendectomy has failed to establish itself as minimally invasive procedure of choice both in children and adults (26-28).

Open Appendectomy

Conventional-appendectomy: Done by standard methods with the help of either of the available incisions (Grid Iron, Rutherford-Morrison’s, Rockey Davis, Lanz, Paramedian, Midline)

Mini-appendectomy: This is done with the help of small transverse incision 2 to 2.5 cms starting from lateral border of rectus abdominis muscle and extended towards Mc Burney’s point. Anterior sheath is cut in line of skin incision, rectus muscle retracted medial and blended posterior sheath/peritoneum cut in line of skin incision. Once peritoneum is approached, with little manipulation appendix is delivered towards wound site and appendectomy completed as per standard protocol. Appendiceal stump is not buried and we do not close posterior peritoneum, retracted rectus muscle comes to its place once anterior sheath is closed. Skin is approximated with silk/clips/subcuticular prolene (29,30).

Laparoscopic-Appendectomy: Though done by many at many centers yet to find large scale favour from
surgical fraternity as procedure of choice for appendicitis.

Summary: Vermiform appendix probably noticed in the the egyption civilisation 3000BC has undergone so many revelations since then. It includes McBurney’s clinical findings in acute appendicitis as well as grid iron incision to laparoscopic appendectomy and mini appendectomy. The classic presentation of a patient with appendicitis includes a history of initial periumblical or epigastric gradual onset and progressively worsening pain migrating to the right lower quadrant, more specifically RIF. Diagnosis of classical acute appendicitis is established by good clinical acumen, but in atypical cases of appendicitis controversy continues over the most accurate, cost effective and rapid method of making the diagnosis. Surgical consultation remains the most effective method of deciding what additional diagnostic tools are needed. The early surgical removal of inflamed appendix remains the best method of treatment for total cure in addition to minimizing morbidity and mortality. The surgical methods available are open and laparoscopic appendectomy. In the absence of non-establishment of laparoscopic appendectomy as minimally invasive procedure of choice, the mini-appendectomy may be tried and steps taken to establish it as minimally invasive procedure of choice for appendectomy.

References

Acute appendicitis is inflammation of the vermiform appendix. Chronic appendicitis is characterized by chronic inflammatory changes of the vermiform appendix thought to be a possible factor in chronic recurrent abdominal pain, but many surgeons are unsure how often this occurs. Perforated appendicitis refers to perforation of the vermiform appendix; perforated appendicitis may result in the formation of a localized periappendiceal abscess with an appendiceal mass, or generalized peritonitis. Gangrenous appendicitis is acute appendicitis or perforated appendicitis accompanied by gangrene of the Sections Inflammation of Vermiform Appendix. Overview. Practice Essentials. In 1886, Reginald H Fitz, a Harvard pathologist, first described the clinical condition of acute appendicitis (ie, inflammation of the vermiform appendix). [1] He correctly pointed out the importance of its early diagnosis and timely treatment, as indicated by his analysis of 257 cases of perforating inflammation of the appendix and 209 cases of typhilitis or perityphilitis. [2] A few years later, Charles McBurney described the clinical findings prior to rupture and advocated early surgical intervention. Despite aggressive intervention, mortality and morbidity remained high through the rest of