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Review

Iliopsoas abscess – A review and update on the literature

D. Shields [a,*], P. Robinson [b], T.P. Crowley [c]

[a] Department of General Surgery, Queen Elizabeth Hospital, Gateshead, County Durham, England, UK
[b] University of Glasgow Medical School, University Avenue, Glasgow, UK
[c] Department of General Surgery, Royal Victoria Infirmary, Newcastle, England, UK

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ABSTRACT

Iliopsoas abscess is a rare condition with a varied symptomology and aetiology. Patients with this condition often present in different ways to different specialities leading to delays in diagnosis and management. Recent advances in the radiological diagnosis of this traditionally rare abscess have highlighted that there is a lack of evidence relating to its aetiology, symptomology, investigation and management. This article reviews the currently available literature to present a concise and systematic review of iliopsoas abscess.

1. Introduction

Iliopsoas abscess (IPA) is a rare condition with a reported incidence of 0.4/100,000 in the UK.[1] It may present with a varied symptomology to any acute speciality, due to the relative rarity and often non-specific features of IPA there are frequently delays in the diagnosis and effective management of the condition. The majority of literature relating to IPA is in the form of case reports and short case series. This review article brings together the available information to give a concise and systematic review of the aetiology, investigation and management of IPA.

2. Aetiology/Epidemiology

Iliopsoas abscess can be classified as primary or secondary. Primary IPA occurs due to the haematogenous or lymphatic spread of a causative organism from a distant site. Secondary IPA occurs as a result of the direct expansion of a nearby infectious/inflammatory process into the iliopsoas.[2,5] Ricci et al. found that the aetiology of IPA was linked to geographical area with over 90% of cases in Asia and Africa being primary in origin while in Europe only 18.7% of reported cases were primary.[4]

Primary IPA makes up approximately 30% of all cases seen. It is more common in children where it can be mistaken for septic arthritis of the hip. The incidence while still low in the West is rising especially in those who are immunocompromised. Patients with HIV are particularly at risk.[6,7] Trauma to the muscle also appears to be a significant risk factor. This was shown by Levin et al.[8] when pyomyositis occurred in injured muscle following administration of intravenous *Staphylococcus aureus*.

Secondary IPA accounts for the majority of cases seen and most commonly arises from intra-abdominal inflammatory processes particularly those of intestinal origin. Other causes include spinal and other skeletal pathology.[1,3,4] An overview of conditions linked to the development of secondary IPA is shown in Table 1.

The most common disease associated with secondary IPA is Crohn's disease[11] but other well recognised source conditions include appendicits, ulcerative colitis, diverticulitis and colorectal carcinoma. Urinary tract infection and instrumentation of the upper renal tract have been shown to be causes of secondary IPA[9,10] as has vertebral osteomyelitis and hip septic arthritis particularly in the context of hip arthroplasty.[12]

3. Relevant anatomy

The anatomy of iliopsoas is well described.[14–17] The main flexor of the hip iliopsoas is formed by the iliacus and psoas major muscles. An accessory psoas minor muscle is found in between 10 and 65% of people[13] Psoas major arises from the bodies of the five lumbar vertebrae, passes along the posterior abdominal wall and beneath the inguinal ligament. It passes the hip joint and inserts on the lesser trochanter of the femur via the iliopsoas tendon. As it

* Corresponding author.
E-mail address: dwshields@gmail.com (D. Shields).

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Table 1
Table indicating documented origins for secondary spread to the iliacus or psoas muscles.

Source location	Condition
Gastrointestinal	Crohn's disease
	Diverticulitis
	Appendicitis
	Colorectal carcinoma
Genitourinary	UTI
	Instrumentation
Musculoskeletal Infection	Vertebral osteomyelitis
	Infectious sacroilitis
	Septic arthritis
Others	Trauma
	Endocarditis
	Hepatocellular Carcinoma
	Femoral artery catheterisation

passes the hip joint there is occasional communication between the bursa and the hip joint. Iliacus arises from the superior portion of the iliac fossa and again enters the thigh under the inguinal ligament. It inserts again into the lesser trochanter of the femur via the iliopsoas tendon at the iliopubic eminence and secondly into a small area of the femoral shaft below the lesser trochanter. The surface of the muscle is invested in the strong psoas fascia. This fascia runs from the lumbar vertebrae to the iliopubic eminence. It is behind this dense fascia that an iliopsoas abscess forms.

The blood supply to psoas major is derived from the four lumber arteries on the ipsilateral side and venous drainage is via the lumbar veins. Iliacus receives its arterial supply from the medial circumflex femoral artery and the iliac branch of the iliolumbar artery, the first posterior branch of the internal iliac artery.

The lumbar neural plexus is enclosed within psoas major with branches exiting from the muscle. The psoas major muscle itself is innervated by the roots of L1–3. Iliacus receives innervation from the L2 and L3 roots along with branches from the femoral nerve.

Iliopsoas forms the posterior relation for the diaphragm, the kidneys, the renal vessels, the ureter, the gonadal vessels and the genitofemoral nerve. The sigmoid colon lies in close contact on the left as does the caecum and appendix on the right. Posteriorly to iliopsoas in the quadratus lumborum muscle and the transverse spinous processes. In the thigh iliopsoas forms part of the floor of the femoral triangle.

4. Microbiology

4.1. Culture modality

Blood cultures and direct abscess aspirate are the most definitive diagnostic tools available, with the abscess aspirate giving higher yield and specificity. Histology, clinical features and positive cultures from the original source (such as urine, faecal and sputum cultures in secondary abscess) are considered as indirect indicators of the causative microbe.

4.2. Causative organisms

It is important to note, that even in the largest case, a definitive microbiological diagnosis was still only found in 75% of cases.[18]The individual microbe implicated is related to the aetiology, i.e. primary vs secondary IPA.

In most cases, a single organism will be implicated, however microbiological samples may be polymicrobial, which is more frequent in abscesses arising from gastrointestinal or urinary origin.[18]

The most common organism in primary IPA, and also secondary IPA (with skeletal infection as the source) is *S. aureus*.[4, 18,19] In the other two most common aetiologies of secondary IPA, i.e. gastrointestinal and urinary, *Escherichia coli* is the most common organism.[4, 18,19] Other organisms seen include *Bacteroides* species, *Mycobacterium tuberculosis*, *Streptococcus viridans*, *Enterococcus faecalis*, *Peptostreptococcus* and *S. viridans*.[4, 18–20]

Methicillan Resistant *S. aureus* (MRSA) has been reported on a case-by-case basis,[4] and not only is it hard to gauge the true incidence in this rare condition, but it is also likely to vary with geographical location. The case series from Lopez et al. in 2009, found that of ninety three confirmed microbial causes, twenty three were due to *S. aureus*, of which only one was MRSA.[18]

4.3. HIV

There have been a number of cases of IPA in patients with HIV reported in the literature. The causative organisms appear to follow a different pattern with the most commonly seen organism being *M. tuberculosis* with *S. aureus* the second most commonly seen organism. The mean age of those affected is lower and it is more likely to be a primary IPA than a secondary. Secondary abscesses when they occur most commonly have their source in the genitourinary tract or of lumbar spondylodiscitis in origin.[18]

4.4. Tuberculosis

M. tuberculosis has generally been implicated in IPA in the context of secondary IPA with spread from an adjacent source be it skeletal, genitourinary or gastrointestinal. Primary IPA with mycobacterium tuberculosis has been seen presumably with haematological spread from the respiratory system.[18] IPA with mycobacterium tuberculosis is associated with a delay from onset of symptoms to diagnosis with a lower incidence of pyrexia and leucytosis.[18]

5. Clinical presentation

As originally described by Mynter in 1881,[21] the classic triad of back pain, limp and fever may be present, but subsequent case studies have identified that this may only be the case in 30% of cases.[22] Many patients will present with an insidious onset of non-specific features such as malaise and low grade pyrexia which may progress into more specific symptoms, like those originally described, along with others, such as; abdominal/flank discomfort, a flexed and externally rotated hip, pain on movement of the hip (due to irritation of the muscle belly and also by referred pain from the nerve roots L2, L3 and L4, which supply the psoas muscle).[23,24]

Table 2
Table adapted from Lopez et al. microbiology and outcome of iliopsoas abscess in 124 patients[18] showing the 7 most common organisms, their origins and proportion of that origin.

Microorganism	Primary abscesses (%)	Skeletal origin (%)	Gastrointestinal origin (%)	Urinary origin (%)
Staphylococcus aureus	42.9	35.2	–	7.7
Escherichia coli	14.3	2.9	42.1	61.5
Bacteroides spp	4.8	–	26.3	15.4
Mycobacterium tuberculosis	4.8	17.7		–
Streptococcus viridans	19	–	10.5	–
Enterococcus faecalis	–	–	15.8	15.4
Peptostreptococcus spp	4.8	2.9	15.8	–

Table 3
Table of differential diagnosis.

Diverticulitis	Usually left sided, giving pain in a similar area, but infrequently gives dermatomal sensory disturbance in the L1-3 roots
Appendicitis	Can present in a similar way, but the classic history of migratory pain, with McBurney's sign can differentiate. However, 'psoas sign' is a popular sign and can be present in both appendicitis and psoas abscess.
Muscle strain (groin)	Usually attributed to muscle injury by the patient. The pattern of pain radiation seen in psoas abscess is absent. No systemic symptoms.
Meralgia paresthetica	Often causes only paraesthesia but can also cause shooting pain to the anterior and lateral surface of the thigh due to compression of lateral femoral cutaneous nerve (originates from L2 and L3) around the groin.
Sciatica	Back pain due to irritation of lumbar or sacral nerve roots typically radiates to the posterior or lateral surface of the thigh, knee, or leg. However, it may involve the dorsum of the foot, first or second and third toes (L5 root), or plantar surface of the foot and forth or fifth toes (S1 root) making the diagnosis of IPA less likely. The presence of paraesthesia in the distribution of pain is suggestive of sciatica. Rarely, irritation of L3 or L4 root can lead to back pain that radiates to the anterior thigh and knee, but the knee jerk is usually diminished or absent and there is associated paraesthesia.
Renal colic/pyelonephritis	These conditions characteristically cause flank pain which radiates to the groin with nausea and vomiting being common associated symptoms. Fever and malaise are unusual except when there is an associated kidney infection.
Endometriosis	In females, retroperitoneal endometriosis can occasionally present with right hip and abdominal pain.
Primary Ewing Sarcoma	Can rarely originate from the spinal column but because an Ewing sarcoma is a very aggressive bone tumour with high proliferative and invasive potential, its clinical features and range of imaging manifestations can mimic the pathologic findings of infectious diseases.
Septic Arthritis of Hip	Both IPA and septic arthritis of the hip may present with features including a limp and pain in the hip. Abdominal pain may be more likely in IPAs but referred pain to the thigh and back may occur in both. Both may have systemic features such as a fever.
Abdominal Aortic Aneurysm	An expanding or contained ruptured abdominal aortic aneurysm may present with an insidious onset, vague abdominal pain or back/flank pain. It is a differential that must not be missed.

5.1. Common clinical features

- Flank/back pain
- Vague abdominal pain
- Fever
- Limp
- Malaise
- Weight loss
- Groin lump[22]

6. Diagnosis

An accurate history and in depth examination are essential to raise suspicion of iliopsoas abscess. An understanding of the nature of insidious onset progressing to more localised disease is helpful. History must include not only natural progression of the symptoms, but other contributing factors, as shown in the tables below. It is important to note, that in many cases, a iliopsoas abscess may be the presenting symptomatology of a new diagnosis of Crohn's disease, or as a consequence of an immunocompromised state, such as HIV. Given the deep anatomical location of the iliacus and psoas muscle bellies, their sheaths and their conjoined tendon, examination can be difficult, however, abdominal tenderness, antalgic passive hip movements, and rarely a painless subinguinal mass, can be elicited. (Tables 2 and 3).

7. Investigations

Routine laboratory investigations including full blood count, C-reactive protein, and erythrocyte sedimentation rate are useful in confirming the diagnosis of an inflammatory mass. Formal imaging is required however not only to confirm the diagnosis, but to plan further treatment.

Further investigations:

- Blood cultures
- Ultrasound
- CT scan +/− drainage

7.1. MRI vs CT sensitivity

MRI has already been shown to be more sensitive than CT in intra-abdominal abscesses, with more accurate delineation of inflammatory change, including that beyond the abscess site.[25,26] CT can give a false negative in cases where the abscess does not contain air, or has a low attenuation. Retroperitoneal MRIs are complex, and although they can be used to diagnose complicating small bowel Crohn's disease and other sources of primary disease, in many smaller centres radiologists are not freely able to report on MRI enteroclysis. The major advantage of CT, is in its role in management, and with pre-existing radiography, percutaneous drainage can be planned accordingly.

8. Treatment

The agreed first line treatment in the literature is broad spectrum antibiotic that will cover *S. aureus* and also any possible primary source for the IPA.[4, 18,27–31] Some authors have suggested that targeted antibiotics may be sufficient to treat abscesses up to 60 mm[28] however without aspiration of the abscess these antibiotics are often a 'best guess' rather than targeted therapy.

Traditionally, surgical drainage was the treatment of choice[28] and some authors have described quicker recovery following open drainage. It has since been established that image guided percutaneous drainage is a very effective and safe alternative.[29] There are still occasions in which open drainage will be preferable such as the IPA which occurs secondary to an intra-abdominal disease process which also requires open surgical intervention. For example complex Chron's disease or diverticulitis.[27,30] There are also technical limitations to CT guided drainage such as small abscess, multiple sepate within the abscess or an inaccessible abscess location.[31,32]

Overall the literature now suggests that many small abscesses can be managed with antibiotics alone[18,28] and that of those abscesses which do require drainage[18] the majority can be aspirated effectively via CT guidance. Antibiotic therapy post drainage should be tailored to the organism/organisms isolated.

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Author contribution
David Shields – Proposal, literature search, writing
Patrick Robinson – Literature search, writing
Timothy P Crowley – Writing

Conflict of interest

None.

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