EJ072200100201

J Microbiol Immunol Infect
2001;34:201-206

Klebsiella pneumoniae psoas abscess: predominance in diabetic patients and grave prognosis in gas-forming cases

Chia-Ming Chang[1], Wen-Chien Ko[1,2], Hsin-Chun Lee[1], Yao-Ming Chen[1], Yin-Ching Chuang[1,2]

[1]Department of Internal Medicine, National Cheng Kung University Hospital and [2]Department of Medicine, College of Medicine, National Cheng Kung University, Tainan, Taiwan, ROC

Received: September 7, 2000 Revised: October 31, 2000 Accepted: February 8, 2001

Seven cases of psoas abscess caused by *Klebsiella pneumoniae* were observed at the National Cheng Kung University Hospital within a period of 4.5 years. These cases constituted 25% of a total 28 episodes of non-tuberculous psoas abscess, ranking second to those caused by *Staphylococcus aureus* (8 cases). Eight cases of psoas abscess caused by *K. pneumoniae* were identified from Medline, and 5 of which were reported from Taiwan. Of these 8 cases, 1 neonatal case was excluded, and the remaining 7 adult cases were combined with the 7 cases in this series for analysis. The mean age was 53.8 years, and diabetes mellitus was the most common underlying disease. Fever and pain on the flank and back area were the common findings. The interval between the onset of symptoms and diagnosis ranged from 1 to 60 days. The most common sites of concurrent infection were the urinary tract (6 cases; 43%) and bone (3 cases; 21%). All patients received percutaneous or surgical drainage in addition to antibiotic treatment. Gas formation was present in 5 of the 12 patients recorded, and 4 of them died during hospitalization. Only 1 patient had a metastatic infection with osteomyelitis of the left radius and right humerus; he had experienced 5 episodes of recurrent *K. pneumoniae* infections in different sites. We concluded that *K. pneumoniae* should be considered as an important endemic pathogen of psoas abscess in diabetics in Taiwan. The high mortality rate in the gas-forming cases should also be highlighted. Early recognition, empiric antimicrobial coverage for *K. pneumoniae*, and aggressive drainage or debridement are indicated in these patients.

Key words: Diabetes mellitus, *Klebsiella pneumoniae*, psoas abscess, pyomyositis

Pyogenic psoas abscess (PPA), a rare but life-threatening infection, results from primary suppuration or is secondary to the spread of infection from an adjacent structure. The causative organisms are different between primary and secondary PPA–*Staphylococcus aureus* is predominant in primary PPA, whereas secondary PPA is frequently caused by mixed pathogens, with common enteric flora and *Escherichia coli* being the leading organisms [1-3]. In 1986, Ricci *et al* [2] reviewed 367 cases of PPA from the literature worldwide. A single organism was cultured in 215 (87.5%) of 246 cases, and *S. aureus* (190 cases; 88.4%), streptococci (12 cases; 4.9%), and *E. coli* (7 cases; 2.8%) were the most common pathogens. After the report of the first case in 1962, PPA caused solely by *Klebsiella pneumoniae* has been rarely reported [1,4-8]. Basing on the clinical observation of several cases of *K. pneumoniae* PPA in Taiwan, this study aimed to review the clinical characteristics of this disease and to delineate its possible relevance.

Corresponding author: Dr. Yin-Ching Chuang, Department of Medical Research, Chi-Mei Foundation Medical Center, Tainan, Taiwan, ROC.

Materials and Methods

To identify patients with *K. pneumoniae* PPA at the National Cheng Kung University Hospital (NCKUH), total medical charts of a diagnosis of PPA from July 1995 through December 1999 were reviewed. Information on clinical presentations, demographic data, underlying diseases, concurrent infections, laboratory findings, radiological imaging, treatment course, and outcomes were collected. A search of the literature in Medline from 1960 through 1999 was performed, and relevant studies were reviewed to include additional cases of *K. pneumoniae* PPA for analysis.

The diagnoses of *K. pneumoniae* PPA were based on clinical, roentgenographic or computed tomographic (CT) images, and isolation of *K. pneumoniae* from the culture of the drained abscess. Identification was based on colony morphology and traditional biochemistry reactions in test tubes. Antimicrobial susceptibility tests

©2001 Chinese Society of Microbiology, Chinese Society of Immunology, and Infectious Diseases Society of the Republic of China

were determined by adopting the disc diffusion method as described by the National Committee for Clinical Laboratory Standards in the United States [9]. A concurrent source of *K. pneumoniae* infection was recorded if an isolation of the same species from another body site was microbiologically documented, or if there were clinical evidence based on physical examination or imaging.

Shock was defined as a decrease in systolic blood pressure of 30 mm Hg or more, or that a decrease to a value of less than 90 mm Hg is recorded. Fever was considered to be present if the maximal axillary temperature was 38°C or higher on a single measurement at admission. Leukocytosis was defined as a total white blood cell count of above 10 000 /mm^3. Bandemia was considered when the band cell count was more than 5%. Elevated C-reactive protein (CRP) was defined as a value higher than 5 mg/L. The diagnosis of liver cirrhosis was based on sonographic, laboratory, and clinical findings; histological confirmation was not required. Diabetes mellitus was diagnosed as a random plasma glucose level of above 200 mg/dL, a fasting plasma glucose level of above 126 mg/dL, or a fasting venous whole blood glucose level of above 120 mg/dL on more than 1 occasion. Alcoholism was considered if a prolonged consumption of excessive amounts of alcohol was shown in the patient's history.

Results

Within the 4.5-year period, 4 men and 3 women were given a diagnosis of PPA that was caused solely by *K. pneumoniae* (cases 1-7, Table 1) at NCKUH. These cases constituted 25% of the total 28 episodes of non-tuberculous PPA at NCKUH, ranking second to those caused by *S. aureus* (8 cases). Table 2 showed the microbiological pathogens that have caused PPA in 3 series of patients in Taiwan [1,10]. In this series, 5 (71%) of the 7 cases who had experienced *K. pneumoniae* PPA were diabetics, whereas only 5 (24%) of the 21 cases of non-*K. pneumoniae* PPA were diabetics ($p<0.05$, Fisher's exact test).

Only 8 cases of PPA caused by *K. pneumoniae* (cases 8-15, Table 1) were found from an extensive survey of the literature in Medline from 1960 through 1999, 5 of which were reported in Taiwan [1,5,7]. One neonatal case was excluded, and the remaining 7 adult cases in the literature were combined with the 7 cases from this study for analysis. The mean age of these 14 patients was 53.8 years (range, 32-75 years). Eleven (79%) patients had underlying conditions, which included 8 (57%) cases of diabetes mellitus, 4 (29%) cases of alcoholism, and 2 (14%) cases of liver cirrhosis. None had previous trauma to the affected lesion. Patient 7 had a perianal abscess and has received incision and drainage 2 weeks before admission. Patient 9 had undergone surgical drainage to treat a deep neck infection caused by *K. pneumoniae* 43 days before admission.

At the onset of the infection in the psoas muscle, 12 patients had localized symptoms, which included flank pain (6 cases), back pain (3), abdominal pain (2), and painful swelling of the buttock (1). The median interval between onset of symptoms and diagnosis was 10 days (range, 1 day-2 months). At admission, fever was present in all but 2 cases, shock in 4, and disturbed consciousness in the 3 oldest patients. Gas formation was found in 5 (42%) of the 12 patients recorded, and 4 (80%) of them died after admission. In contrast, only 1 (14%) of the 7 cases of non-gas-forming *K. pneumoniae* PPA died ($p<0.05$, Fisher's exact test). Of the 9 patients recorded, the mean white blood cell count was 13 756 /mm^3, with leukocytosis in 7 (78%) and bandemia in 6 (67%) patients.

Blood cultures yielded growth of *K. pneumoniae* in 9 (75%) of the 12 patients and urine cultures in 3. Apart from *K. pneumoniae*, the urine culture of patient 2 contained a mixed growth of *E. coli*. The most common sites of concurrent infection were the urinary tract (6 cases; 43%) and bone (3 cases; 21%). Cases of concurrent bone infection comprised of 2 cases of infection in the vertebra and 1 in the radius and humerus.

In addition to antibiotic treatment, all patients underwent percutaneous or surgical abscess drainage. Among the surviving patients, the duration of inpatient stay ranged from 21 to 116 days. Five patients died after admission, and the overall mortality was 36%. Death was directly related to the *K. pneumoniae* infections in 3 patients (patients 1, 5, and 8). Patient 10 died of a ruptured abdominal aorta secondary to the psoas abscess. Patient 2 died of a recurrent stroke and nosocomial pneumonia 18 days after admission, when the primary infection was under control. Of the 5 patients who died, either shock or disturbed consciousness was present at admission, and gas-forming abscess was present in 4 (80%) cases.

Two patients had recurrent episodes of *K. pneumoniae* infections. Patients 6 had experienced 5 episodes of recurrent *K. pneumoniae* infections. Before the episode of psoas abscess, bilateral scrotal abscesses, bilateral perirenal abscesses, bacteremia of unknown origin, and liver abscess developed during a 27-month period, and each episode was separated by disease-free intervals of 6 to 9 months. One month after discharge

Table 1. Clinical characteristics of 15 patients with psoas abscess caused by *Klebsiella pneumoniae*

Reference	Patient no./ country/year	Age/sex	Underlying condition(s)	Concurrent infection(s)	Duration of symptoms	Systemic symptom(s)	Localized symptom(s)	Source(s) of positive culture other than absces	Definite duration of antibiotics (day)	Drainage	Outcome	Hospital day	Remarks
Present report	1/Taiwan/1995	42/M	DM, liver cirrhosis, alcoholism	Pararenal abscess	3 days	Fever, shock	Abdominal pain	Blood	Cefotaxime (4) Oxacillin (4) Metronidazole (4)	PCD	Died	4	Gas+
Present report	2/Taiwan/1996	75/F	DM, chronic renal failure, old stroke	Emphysematous pyelonephritis, perirenal and pararenal abscess	1 day	Fever, confusion	Abdominal pain	Urine	Cefoxitin (6) Cefuroxime (11) Ofloxacin (11)	PCD	Died of restroke and nosocomia pneumonia	21	Gas+
Present report	3/Taiwan/1996	58/F	DM	Perirenal and retroperitoneal abscess	1 week	Fever, chills	Flank pain	None	Cefotaxime (5) Cefuroxime (19) Cephalothin (10)	PCD	Improved	35	–
Present report	4/Taiwan/1998	49/M	Alcoholism	Vertebral osteomyelitis	2 months	Fever	Back pain	Blood	Cefazolin (24)	PCD	Improved	24	–
Present report	5/Taiwan/1998	62/F	DM	None	10 days	Fever, chills, shock, confusion	Flank and thigh pain, limited hip movement	Blood	Cefotaxime (4) Metronidazole (4)	PCD	Died	4	Gas+
Present report	6/Taiwan/1999	32/M	Alcoholism, DM, chronic pancreatitis	Perirenal abscess, osteomyelitis (left radius)	1 week	Fever, dyspnea	None	Blood, urine	Cefmetazole (15) Cefuroxime (11) Lomefloxacin (13)	PCD	Improved	32	Recurrent infections
Present report	7/Taiwan/1999	53/M	None	Vertebral osteomyelitis	1 week	Fever	Flank pain	Blood	Piperacillin/ tazobactam (19) Moxalactam (13) Imipenem (10) Ciprofloxacin (90)	PCD	Improved	51	Prior perianal abscess after I & D
[4]	8/US/1961	71/M	DM	Perinephric abscess, UTI	1 month	Fever, confusion	Flank pain, positive psoas sign	Blood, urine	Penicillin Streptomycin	Surgery	Died	4	Gas+
[5]	9/Taiwan/1996	55/M	DM	Pneumo-retroperitoneum	1 month	Fever, shock	Back pain, thigh pain, crepitus	Blood	Ceftriaxone Metronidazole	PCD 3 times	Improved	116	Gas+ Prior deep neck infection
[6]	10/Japan/1994	42/F	Alcoholism, chronic pancreatitis	Abdominal aortic aneurysm	3 weeks	Fever, shock	Back pain	Blood	Cefazolin	Surgery	Died	7	Ruptured abdominal aorta
[7]	11/Taiwan/1994	75/M	None	Wound infection	1 month	Fever	Flank pain, limited hip movement	NR	NR	PCD	Improved	NR	Gas?
[7]	12/Taiwan/1994	66/F	DM	UTI	1 day	Fever	None	NR	NR	Surgery	Improved	NR	Gas?
[1]	13/Taiwan/1999	33/M	None	Retroperitoneal abscess, trauma	3 weeks	None	Flank & back pain	None	Cefazolin	Surgery	Improved	24	
[1]	14/Taiwan/1999	39/F	Liver cirrhosis	Perirenal abscess	10 days	None	Buttock swelling & pain	Blood	Cefazolin (10) Ceftriaxone (38)	PCD	Improved	48	
[8]	15/Greece/1997	18 d /F	Small for gestational age	Septic arthritis	13 days	Apnea		Blood, synovial fluid	Co-trimoxazole, Amoxicillin/clavulanate	Surgery	Improved		

Abbreviations: DM = diabetes mellitus; I & D = incision and drainage; NR = not recorded; PCD = percutaneous drainage; UTI = urinary tract infection

Table 2. Microbiological pathogens of pyogenic psoas abscess in three series of patients from Taiwan

Pathogen	No. of patients		
	Lee *et al* 1988-1998 (n = 11)	Lin *et al* 1993-1998 (n = 27)	Chang *et al* (present study) 1995-1999 (n = 28)
Staphylococcus aureus	3	10	8
Klebsiella pneumoniae	0	2	7
Salmonella spp.	1	1	3
Escherichia coli	1	6	1
Coagulase-negative staphylococci	2	1	1
Streptococcus agalactiae	0	2	2
Viridans streptococci	0	2	1
Fungi	1	1[a]	0
Others	1[b]	0	2[c]
Mixed bacteria	1[d]	0	2[e]
No growth	1	4	1

[a]*Candida albicans.*
[b]*Pseudomonas aeruginosa.*
[c]*Serratia marcescens* and *Norcardia* spp.
[d]*Klebsiella pneumoniae* and *Klebsiella ozaenae.*
[e]The one PPA was caused by *Klebsiella pneumoniae, E. coli,* and *Enterococcus,* the other was caused by *E. coli* and *Proteus mirabilis.*

from hospitalization for psoas abscess, the patient was brought to an emergency room and died of *K. pneumoniae* urosepsis and hepatic failure. No other causative organism except *K. pneumoniae* were associated with these episodes. The antibiograms of the isolates of *K. pneumoniae* from this patient were similar in their resistance to ampicillin and susceptibility to all cephalosporins, new fluoroquinolones, and aminoglycosides. Patient 9, as mentioned, had a deep neck infection caused by *K. pneumoniae.*

On admission, all 7 patients at NCKUH had elevated levels of CRP (mean, 189.8 mg/L; range, 40.7-340 mg/L). Abnormal liver function was found only in patient 4, who had a liver function of about 3 times above the normal level. Patients 1 and 7 had thrombocytopenia of 79 000 and 49 000 /mm^3, respectively. Patients 2, 3, 5, and 6 showed elevated glucose levels of 276, 468, 380, and 1195 mg/dL, respectively.

All 7 isolates from patients at NCKUH were resistant to ampicillin. The strain isolated from a blood culture of patient 6 was resistant to cephalothin. The isolate from the pus of patient 2 was also resistant to cephalothin, gentamicin, and tobramycin, and showed intermediate susceptibility to cefixime. The isolate from patient 7, who was considered as experiencing a nosocomial infection, was susceptible only to cefmetazole, moxalactam, piperacillin/tazobactam, and imipenem. Except for patient 7, all isolates were susceptible to the new fluoroquinolones and second- and third-generation cephalosporin, with the cefixime mentioned above excluded.

Discussion

Pyogenic psoas abscess caused solely by *K. pneumoniae* is an uncommon disease. *K. pneumoniae* is one of the major causes of pyogenic liver abscess [11,12], bacteremia [13], and gram-negative bacillary meningitis in Taiwan [14,15]. *K. pneumoniae* is also not an uncommon causative agent of gram-negative psoas abscess in Taiwan [1,7], especially among the diabetics. Diabetes mellitus is a common underlying disease in patients infected with *K. pneumoniae*; about 27% to 75% of patients with *K. pneumoniae infections* in Taiwan were diabetics [12,13,15]. It has been well documented that diabetics have an impaired chemotaxis, phagocytosis, and bactericidal function [16-18], but the reason why diabetics in Taiwan are particularly vulnerable to *K. pneumoniae* infections remains unknown.

Muscles seem to have an intrinsic resistance to bacterial infection unless they were damaged by electrical shock, trauma, or ischemia. The anatomy of the psoas muscle makes it exceptionally susceptible to infection by both direct extension and distant seeding [19,20]. In contrast to the reported etiologies of PPA, which were usually from a gastrointestinal source [2], the urinary tract and the vertebra are the infectious foci more commonly found in Taiwan, as reported by Lin *et al* [1] and in this series. Apart from the low incidence of Crohn's disease, there has been limited research on gastrointestinal abnormality in Taiwan, and the cause of the higher rate of PPA in Taiwan remains unknown.

Four of the 5 fatal cases in this study had gas-

forming abscesses, and all of the 5 gas-forming cases were diabetics. A significantly higher mortality rate has been reported in patients with gas-forming *K. pneumoniae* liver abscesses [11]. Huang *et al* [21] has postulated 4 factors involved in the pathogenesis of emphysematous pyelonephritis, which include gas-forming bacteria, high level of tissue glucose, impaired tissue perfusion, and a defective immune response (eg. diabetes mellitus). Of the 5 patients who died, either disturbed consciousness or shock was present. A grave prognosis should be considered for diabetic patients with any of these presentations.

A low relapse rate (2%-4.4%) for *K. pneumoniae* infections is reported in the literature [12,13,15]. Two of the 14 adult cases (14%) had previous or subsequent episodes of *K. pneumoniae* infections that involve body sites other than the psoas muscle. As metastatic infection is a characteristic feature of *K. pneumoniae* liver abscess [22], the high rate of positive blood cultures in this series suggested that hematogenous spread might be a likely cause of recurrent infection. Any case of pyogenic infection caused by *K. pneumoniae* should be closely observed for potential recurrence in addition to metastatic infections.

Although fever and localized pain were present in most cases, the classical symptomatic triad of fever, abdominal or loin pain, and limping or limitation of hip movement [7,23] were not evidenced in all 14 cases. A highly raised CRP and leukocytosis with bandemia were usually present. To ensure an early diagnosis and appropriate management, physicians and radiologists should maintain a high degree of suspicion towards PPA [23]. Diagnostic evaluations of suspected PPA include kidney-ureter-bladder radiography, ultrasonography, and computed tomography (CT). Kidney-ureter-bladder radiography, however, is neither sensitive nor specific, and can not be used as a single tool for definitive diagnosis of PPA. Ultrasonography is inexpensive and easily available, but it is technique-dependent [7]. Computed tomography is the most sensitive tool, and the diagnosis of PPA can be confirmed by the finding of hypodense lesions within the psoas muscle. This method also allows the extent of the PPA to be defined. It is therefore suggested that PPA should be considered in febrile patients with abdominal, loin, or back pain; a highly elevated CRP; and leukocytosis with bandemia; and these patients should receive a CT examination if possible.

Aggressive abscess drainage and antibiotic therapy are important in the management of psoas abscess. With improving imaging techniques, percutaneous drainage becomes the treatment of choice in treating psoas abscess [24,25]. Repeated percutaneous drainage is a feasible treatment in severe cases of PPA, especially when there is a high surgery risk [14]. Because diabetic patients are more vulnerable to infections caused by *K. pneumoniae*, which is usually susceptible to the first-generation cephalosporins and aminoglycosides in the community-acquired cases, this combination can be used as an initial empirical therapy for PPA in diabetics.

In conclusion, *K. pneumoniae* should be considered an important endemic pathogen that causes PPA in diabetics in Taiwan. Gas-formation in psoas muscle caused by *K. pneumoniae* is likely to herald a poor prognosis. The classical symptomatic triad was incomplete in a number of cases, and clinical alertness is necessary for early diagnosis.

Acknowledgments

The authors would like to thank Dr. Ming-Feng Lin of the Taichung Veterans General Hospital for providing data of patients 13 and 14.

References

1. Lin MF, Lau YJ, Hu BS, Shi ZY, Lin YH. Pyogenic psoas abscess: analysis of 27 cases. J Microbiol Immunol Infect 1999; 32:261-8.
2. Ricci MA, Rose FB, Meyer KK. Pyogenic psoas abscess: worldwide variations in etiology. World J Surg 1986;10:834-43.
3. Santaella RO, Fishman EK, Lipsett PA. Primary vs secondary iliopsoas abscess: presentation, microbiology and treatment. Arch Surg 1995;130:1309-13.
4. Clifford NJ, Katz I. Subcutaneous emphysema complicating renal infection by gas-forming coliform bacteria. N Engl J Med 1962;266:437-9.
5. Jang TN, Juang GD, Fung CP. Fulminating gas-forming psoas muscle abscess due to *Klebsiella pneumoniae* following a deep neck infection. J Formos Med Assoc 1997;96:134-6.
6. Sugawara Y, Sato O, Miyata T, Kimura H, Yamaoka M, Uozaki H, Oka T, Makuuchi M. Ruptured abdominal aorta secondary to psoas muscle abscess due to *Klebsiella pneumoniae* in an alcoholic. J Infect 1997;35:185-8.
7. Chern CH, Hu SC, Kao WF, Tsai J, Yen D, Lee CH. Psoas abscess: making an early diagnosis in the ED. Am J Emerg Med 1997;15:83-8.
8. Andreou A, Karasavvidou A, Papadopoulou F, Koukoulidis A. Ilio-psoas abscess in a neonate. Am J Perinatol 1997;14:519-21.
9. National Committee for Clinical Laboratory Standards. Performance standards for antimicrobial disk susceptibility test: approved standards. 6th ed. NCCLS document M2-A6. Villanova, PA: National Committee for Clinical Laboratory Standards; 1997.
10. Lee YT, Lee CM, Su SC, Liu CP, Wang TE. Psoas abscess: a 10-year review. J Microbiol Immunol Infect 1999;32:40-6.
11. Yang CC, Chen CY, Lin XZ, Chang TT, Shin JS, Lin CY. Pyogenic liver abscess in Taiwan: emphasis on gas-forming liver abscess in diabetics. Am J Gastroenterol 1993;88:1911-5.
12. Wang JH, Liu YC, Lee SS, Yen MY, Chen YS, Wang JH, Wann SR, Lin HH. Primary liver abscess due to *Klebsiella pneumoniae* in Taiwan. Clin Infect Dis 1998;26:1434-8.

13. Wang LS, Lee FY, Cheng DL, Liu CY, Hinthorn DR, Jost PM. *Klebsiella pneumoniae* bacteremia: analysis of 100 episodes. J Formos Med Assoc 1990;89:756-63.
14. Jang TN, Wang FD, Wang LS, Yu KW, Liu CY. Gram-negative bacillary meningitis in adults: a recent six-year experience. J Formos Med Assoc 1993;92:540-6.
15. Tang LM, Chen ST, Hsu WC, Chen CM. *Klebsiella* meningitis in Taiwan: an overview. Epidemiol Infect 1997;119:135-42.
16. Mowat A, Baum J. Chemotaxis of polymorphonuclear leukocytes from patients with diabetes mellitus. N Engl J Med 1971; 284:621-7.
17. Bybee JD, Rogers DE. The phagocytic activity of polymorphonuclear leukocytes obtained from patients with diabetes. J Lab Clin Med 1964;64:1-13.
18. Repine JE, Clawson CC, Goetz FC. Bactericidal function of neutrophil from patients with acute bacterial infection and from diabetes. J Infect Dis 1980;142:869-75.
19. Dolfin D, Barkin J, Arenson AM, Herschorn S. Psoas abscess after operation on the lumbar spine. Urology 1983;21:544-6.
20. Walsh TR, Reilly JR, Hanley E, Webster M, Peitzman A, Steed DL. Changing etiology of iliopsoas abscess. Am J Surg 1992; 163:413-6.
21. Huang JJ, Chen KW, Ruaan MK. Mixed acid fermentation of glucose as a mechanism of emphysematous urinary tract infection. J Urol 1991;146:148-51.
22. Cheng DL, Liu YC, Yen MY, Liu CY, Wang RS. Septic metastatic lesions of pyogenic liver abscess. Arch Intern Med 1991; 151:1557-9.
23. Thomas A, Albert AS, Bhat S, Sunil KR. Primary psoas abscess-diagnostic and therapeutic considerations. Br J Urol 1996;78: 358-60.
24. Vatandaslar F, Alemdaroglu A. CT-guided percutaneous drainage of psoas abscess Urology 1987;29:450-3.
25. Mueller PR, Ferrucci JT, Wittenberg J, Simeone JF, Butch RJ. Iliopsoas abscess: treatment by CT-guided percutaneous catheter drainage. Am J Roentgenol 1984;142:359-62.