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Neonatal renal abscess caused by *Staphylococcus aureus*

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Renal abscesses are uncommon in children and rarely reported in neonates. In a recent survey Sood et al.[1] identified only three reported cases of neonatal renal abscess formation. *Staphylococcus aureus* was the causative organism in two cases whereas *Escherichia coli* was identified in the third case. He reported a fourth case caused by *Klebsiella pneumoniae*. We present a case of *S. aureus* renal abscess in the neonate to emphasize its potentially insidious onset and to review current management.

CASE

JY was a 28-day-old white male infant who was admitted to a community hospital with a 1-day history of fever, irritability and decreased feeding. There were no previous problems in the neonatal period, and although weight gain had been slow while breast feeding, he had been well. There was no recent infection of the family or of anyone else in contact with the infant. On admission he was noted to be irritable with a rectal temperature of 103°F, heart rate of 170/minute, respiratory rate of 32/minute, blood pressure of 108/58 mm Hg and a weight of 3.4 kg. The remainder of his physical examination was normal except for a glanular hypospadius.

Cultures of blood, cerebrospinal fluid and urine were obtained. A complete blood count showed a hemoglobin of 10.6 g/dl and a hematocrit of 31.4%. The white blood cell count was 9400/mm^3 with 44% segmented neutrophils and 14% band forms. The urinalysis showed cloudy urine containing 4+ protein, large occult blood, too numerous to count white blood cells and many bacteria. The blood urea nitrogen was 6 mg/dl and the creatinine was 0.3 mg/dl; both remained normal throughout the hospital course.

Empiric therapy was started with ampicillin, gentamicin and acyclovir because of a past history of maternal genital herpes. Blood and urine cultures grew *S. aureus* which was susceptible to gentamicin but resistant to ampicillin; therefore the ampicillin was discontinued. During a 10-day course of gentamicin the patient remained intermittently febrile, although his urinalysis cleared completely and follow-up blood and urine cultures showed no growth. He had intermittently elevated blood pressure with sustained hypertension noted on the eighth hospital day with values ranging from 110 to 140/79 to 102 mm Hg. A renal ultrasound which had been obtained on the day following admission was normal; the patient was given hydralazine but his hypertension did not resolve or improve.

On Hospital Day 11 the infant was transferred to Thomas Jefferson University Hospital (TJUH) for further evaluation of the hypertension. On admission to TJUH he was in no distress and his vital signs were: blood pressure, 140/80 mm Hg in all extremities; heat rate, 156/minute; respiratory rate, 40/minute; and rectal temperature, 101°F. A Grade II/VI systolic murmur was heard over the entire chest. The lungs were clear on auscultation and the abdominal examination was unremarkable. The hypertension responded to the addition of propranolol to the hydralazine medication. The initial laboratory studies at TJUH showed a hemoglobin of 7.9 g/dl, hematocrit of 23%, a white blood cell count of 21 600/mm^3 with 45% segmented neutrophils and 6% band forms, blood urea nitrogen of 11 mg/dl and creatinine of 0.4 mg/dl. A urinalysis was normal. Blood and urine cultures showed no growth.

A renal ultrasound scan was obtained the following morning (Day 12) which showed a markedly abnormal right kidney which was enlarged (6.7 cm long) and demonstrated hydropyonephrosis with possible renal abscess (Fig. 1). The left kidney was normal. A [^{99m}] Tc-diethylenetriaminepentaacetic acid renal scan showed decreased perfusion of the right kidney with evidence of obstruction (Fig. 2). The perfusion of the left kidney was normal.

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FIG. 1. Sagittal sonogram of right kidney. The upper pole is to the left. A round zone suggestive of cavitation and containing internal echoes, compatible with an abscess, is present in the upper renal pole (*arrowheads*). Distention and deformity of the renal pelvis and calyces (*arrows*), also with internal echoes, are compatible with pyonephrosis.

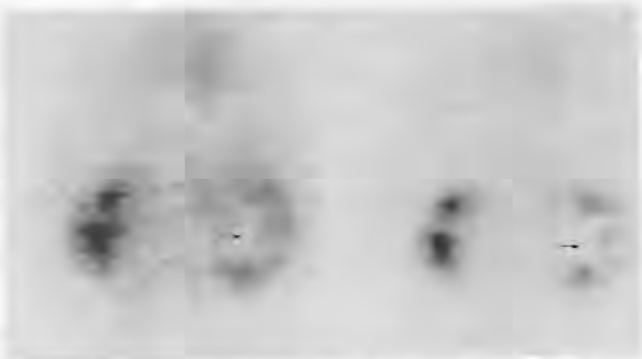

FIG. 2. Two posterior images of the kidneys from a [^{99m}]Tc-diethylenetriaminepentaacetic acid isotope renal scan. The right kidney demonstrates a large central photopenic zone (*arrow*) corresponding to the pyonephrosis and abscess shown by sonography. The left kidney is normal.

The infant was treated with intravenous nafcillin but remained febrile and hypertensive for the next 3 days. Therefore under ultrasound guidance, a percutaneous nephrostomy tube was placed in the abscess cavity, pus was aspirated and contrast media were injected. Eventually with further removal of pus, the entire dilated system was visualized and contrast media passed down the ureter, relieving the obstruction. Within 48 hours of this procedure the infant became afebrile and normotensive and no longer required antihypertensive medication. The aspirated pus grew *S. aureus*, susceptible *in vitro* to nafcillin and gentamicin. A repeat renal ultrasound on the day after placement of the nephrostomy tube showed moderate decompression of the previously dilated right collecting system and a residual 1.9-cm-diameter hypoechoic area consistent with a renal abscess. A repeat [^{99m}]Tc-diethylenetriaminepentaacetic acid isotope scan revealed marked improvement in right kidney function. The infant's condition was stable for the remainder of his hospital course. The nephrostomy tube was removed after 7 days, and before discharge a voiding cystourethrogram showed Grade 2 reflux on the right.

JY was treated with a 14-day course of intravenous nafcillin with a decrease in the white blood cell count to 10 900/mm^3 and was then treated orally at home with dicloxacillin for 4 weeks. Subsequently he was given trimethoprim and sulfamethoxazole prophylaxis because of the right ureteral reflux. Two weeks after discharge from TJUH the infant was well and a renal ultrasound revealed normal kidneys bilaterally. At 7 months of age when last seen he remained normotensive and renal ultrasound was normal.

DISCUSSION

Hematogenous seeding of the kidney or ascending infection from the urinary collecting system are two proposed mechanisms of renal abscess formation. Gorrill et al.[2] demonstrated abscess formation in mouse kidneys by intravenous injection of staphylococci. After injection of *S. aureus*, the bacteria disseminated and a proportion was deposited in the kidney. There was a direct relationship between the number of organisms injected and those deposited. The authors concluded that if enough organisms were present in the bloodstream, some were able to lodge and establish replication in the kidney leading to abscess formation. Other studies have demonstrated that coagulase-positive staphylococci are not cleared as readily as Gram-negative organisms by a normal mouse kidney when arterially infused.[3] Gram-negative organisms have been implicated in hematogenous abscess formation only when the kidney has been traumatized or is obstructed.[4] The development of abscess formation via ascending and lymphatic extension has been shown to be primarily a result of infection by Gram-negative bacilli. Experimental reflux of urine inoculated with *E. coli* has been associated with focal renal parenchymal infection and subsequent abscess formation in the pig.[5] In contrast Freedman et al.[6] inoculated 10^8 colony-forming units of staphylococci directly into six rat bladders and reported the development of a renal parenchymal infection in the right kidney of only one rat. A renal infection rate of almost 100% was achieved when the same inoculum of *S. aureus* was injected intravenously. Thus it appears that *S. aureus* renal abscesses develop primarily through hematogenous seeding, whereas those caused by Gram-negative organisms form as a result of ascending infection.

These experimental data have been corroborated by reported clinical cases.[7-9] In a review of 10 children with renal abscesses treated during a 25-year period at Boston Children's Hospital, it was discovered that most children with Gram-negative renal abscesses had an associated anatomic abnormality (66%) and uri-

nary tract infection (100%).[10] By comparison none of those treated for a *S. aureus* abscess had an associated underlying abnormality and only 1 of 7 had a urinary tract infection. Timmons and Perlmutter[11] also reported a correlation between Gram-negative abscess formation and vesicoureteral reflux in children. Since the advent of antibiotics there has been a significant reduction in the number of renal abscesses attributed to hematogenous seeding of the kidney by staphylococci from cutaneous, respiratory or dental foci and concomitantly an increase in the number of cases secondary to Gram-negative organisms in both adults and children.[9, 12, 13] Whether or not this represents a true rise in Gram-negative abscesses or a decrease in Gram-positive cases is unclear. However, 3 of 5 reported cases in the neonate, including our own, were caused by *S. aureus*. The case of *E. coli* abscess was found in an infant with congenital nephrotic syndrome, a disorder associated with an increased risk of sepsis caused by *E. coli* and *Streptococcus pneumoniae*.[14] The second case of neonatal Gram-negative abscess formation was caused by *K. pneumoniae* in a neonate with posterior urethral valves.[1] Schiavetti et al.[15] described a case of *S. aureus* renal abscess in a neonate without anatomic abnormalities. Although reflux was noted in our case of *S. aureus* abscess formation, it could not be determined whether the reflux was secondary to the infection or represented an underlying anatomic abnormality which allowed reflux and abscess formation.

A high index of suspicion is important in the early detection of renal parenchymal infections. Fever, lumbar pain, abdominal pain and occasionally flank mass are the usual presenting complaints in the older child. These findings may be accompanied by an increased erythrocyte sedimentation rate, by leukocytosis and rarely by a positive blood or urine culture.[7, 16, 17] Findings in the neonate may be similar to those for the older child. In previous reports of neonatal renal abscess, the involved kidney was palpably enlarged, whereas hyperemia and edema of the overlying skin in the lumbar area were seen in the third.[1, 14, 15] In four of five reported cases hematuria was noted, whereas pyuria and proteinuria were seen in three cases.[1, 14, 15] The urine culture was positive in our patient and in both cases of Gram-negative abscess formation.[1, 14] Our patient developed hypertension suggestive of obstruction and upper urinary tract involvement. This was not reported in any of the other cases.

Historically intravenous pyelography and arteriography along with surgical exploration and drainage have been the mainstays of diagnosis and therapy for renal abscesses[10, 18, 19]. In recent years renal sonography has replaced these modalities in the diagnosis and follow-up of renal abscess and nephronia and is the preferred initial imaging procedure.[20–22] Ultrasonography may demonstrate hypoechoic areas with debris in the parenchyma, as well as surrounding edema, which may be characteristic of an abscess.[23,24] Computed tomography will also effectively identify renal abscesses but requires administration of intravenous contrast and results in substantial radiation exposure. Chelates of ^{99}Tc can be used to identify renal abscesses and abnormalities of renal perfusion and filtration. In our patient, [^{99m}]Tc-diethylenetriaminepentaacetic acid, which is quickly secreted and has minimal renal cortical binding, was used to detect perfusion defects and obstruction in the right kidney after sonography indicated the presence of pyonephrosis. [^{99m}]Tc-dimercaptosuccinic acid, which binds strongly to cortical tissue, can be used to define areas of hydronephrosis, pyonephrosis or abscess, but because of its strong binding it may result in significant radiation exposure.[20, 25]

Treatment should include aggressive antibiotic therapy which provides *S. aureus* and Gram-negative coverage. Specific antibiotic coverage is dependent on isolation of organisms from urine, from blood or preferably from ultrasound or computer-assisted tomography-guided percutaneous aspirates of the abscess contents. Although the *S. aureus* isolated in our patient was susceptible to gentamicin, the renal abscess enlarged radiologically during 10 days of therapy with this antibiotic. Gentamicin is not the initial drug of choice for *S. aureus*, yet it is active against *S. aureus in vitro* and is concentrated in the renal medulla and papillae. On the other hand it is also known that within the relatively anaerobic contents of an abscess cavity the MIC of *S. aureus* to gentamicin may be >10-fold that found *in vitro*.[26] Thus in suspected or proven *S. aureus* renal abscess, nafcillin or other penicillinase-resistant penicillins should be utilized.

The need for surgical intervention in neonatal renal abscess is controversial. Hantman and Berger[27] have stated that surgical drainage is mandatory if there is evidence of extension beyond the renal capsule. Other authors conclude that only a failure of adequate antibiotic therapy warrants surgical intervention.[28] Recently percutaneous aspiration under ultrasound guidance and placement of a nephrostomy tube for drainage has been used successfully by Crawford et al.[14] and our group when antibiotic therapy alone failed to resolve the abscess. In both cases there was resolution of the abscess after percutaneous aspiration with no further surgical intervention needed.

CONCLUSION

Renal abscess formation in the neonate is rarely reported, with only four previous cases identified in the literature. A high index of suspicion is probably the most important single factor in its early detection and treatment. We feel that ultrasound is the initial diagnostic method of choice, with computer-assisted tomography and isotope scans also being effective in

identifying abscesses and renal obstruction. We believe that aggressive antibiotic treatment along with percutaneous aspiration and drainage are the best means of management. There continues to be controversy over the need for any surgical intervention. We believe at present that aspiration and drainage are necessary, but with earlier detection and intervention, this therapy may be unnecessary.

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REFERENCES

1. Sood SK, Mulvihill D, Daum RS. Intrarenal abscess caused by *Klebsiella pneumoniae* in a neonate: modern management and diagnosis. Am J Perinatol 1989;6:367–70.
2. Gorrill RH. The establishment of staphylococcal abscesses in the mouse kidney. Br J Exp Pathol 1958;39:203–12.
3. Gorrill RH, Klyhn KM, McNeil EM. The initiation of infection in the mouse kidney after intravenous injection of bacteria. J Pathol Bacteriol 1966;91:157–72.
4. Cotran RS. Experimental pyelonephritis. In: Rouiller C, Muller AF, eds. The Kidney. New York: Academic Press, 1969;2:269–361.
5. Hodson CJ. The pathogenesis of reflux nephropathy. In: Margulis AR, Gooding CA, eds. Diagnostic radiology. San Francisco: University of California, 1978:95–107.
6. Freedman LR, Werner AS, Beck D, Paplanus S. Experimental pyelonephritis: IX. The bacteriological course and morphological consequences of staphylococcal pyelonephritis in the rat, with consideration of specificity of the pathological changes observed. Yale J Biol Med 1961;34:40–51.
7. Cobb OE. Carbuncle of the kidney. Br J Urol 1966;38:262–7.
8. Moore CA, Gangai MP. Renal cortical abscess. J Urol 1967;98:303–6.
9. Fair WR, Higgins MH. Renal abscess. J Urol 1970;104:179–83.
10. Rote AR, Bauer SB, Retik AB. Renal abscess in children. J Urol 1978;119:254–8.
11. Timmons JW, Perlmutter AD. Renal abscess: a changing concept. J Urol 1976;115:299–301.
12. Boone EB, Smith MJV. Renal abscess: old problem with a new look. Va Med Monthly 1976;103:483–5
13. Khashu B, Seery WH, Rothfeld SH. Nonstaphylococcal bacteria in renal cortical abscess. Urology 1976;3:256–9.
14. Crawford DB, Rasoulpour M, Dhawan VM, Klauber GT, Little HK, McLean RH. Renal carbuncle in a neonate with congenital nephrotic syndrome. J Pediatr 1978;93:78–80.
15. Schiavetti E, Torricelli M, Cerri M, Perfetti L. Perinephric abscess in the newborn. Ann Radiol [Paris] 1981;24:532–4.
16. Levin R, Burbige KA, Abramson S, Berdon WE, Hensle TW. The diagnosis and management of renal inflammatory processes in children. J Urol 1984;132:718–21.
17. Moenne-Loccoz JP, Bomsel F, Gatti JM, Prot D. Renal abscess in children. Pediatr Radiol 1978;7:150–4.
18. Segwa JW, Kelalis PP. Localized renal parenchymal infections in children. J Urol 1973;109:1029–32.
19. Lebowitz RL, Fellows KE, Colodny AH. Renal parenchymal infections in children. Radiol Clin North Am 1977;15:37–47.
20. Sty JR. Pediatric nuclear medicine. East Norwalk, CT: Appleton-Century-Crofts, 1983:170–81.
21. Sty JR, Wells RG, Schroeder BA, Starshak, RJ. Diagnostic imaging in pediatric renal inflammatory disease. JAMA 1986;256:895–9.
22. Schneider K, Helmig FJ, Eife R, Belohradsky BH, Kohn MM, Devens K, Fendel H. Pyonephrosis in childhood: is ultrasound sufficient for diagnosis? Pediatr Radiol 1989;19:302–7.
23. Morehouse HT, Weiner SN, Hoffman JC. Imaging in inflammatory disease of the kidney. Am J Roentgenol 1984;143:135–41.
24. Funston MR, Fisher KS, van Blerk PJP, Bortz JH. Acute focal bacterial nephritis or renal abscess? A sonographic diagnosis. Br J Urol. 1982;54:461–6.
25. Verber IG, Strudley MR, Meller ST. ^{99m}Tc dimercaptosuccinic acid (DMSA) scan as first investigation of urinary tract infection. Arch Dis Child 1988;63:1320–5.
26. Kucers A, Bennett NM, Kemp RJ. The use of antibiotics. Philadelphia: Lippincott, 1987:619–75.
27. Hantman IE, Berger BW. Nonsurgical treatment of renal cortical abscesses. Urology 1981;17:415–9.
28. Schiff M Jr, Glickman M, Weiss RM, Ahern MJ, Touloukian RJ, Lytton B, et al. Antibiotic treatment of renal carbuncle. Ann Intern Med 1977;87:305–8.