Study of Organisms Causing Osteomyelitis in a Tertiary Care Hospital

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ABSTRACT

BACKGROUND

The term osteomyelitis (OSM) was first used by the French surgeon Edouard Chassaignac in 1852, who defined the disease as an inflammatory process accompanied by bone destruction caused by an infecting microorganism. The clinical manifestation and the natural history of OSM depend on several factors. OSM mostly affects the growing ends of long bones. We wanted to study the organisms causing osteomyelitis and their antimicrobial susceptibility pattern.

METHODS

Pus and bone aspirate were collected from 115 diagnosed patients of osteomyelitis and were processed for isolation of organisms by standard microbiological techniques. Isolates were identified by various biochemical reactions and were subjected to antimicrobial susceptibility test as per CLSI guidelines by Kirby-Bauer disk diffusion technique on Mueller Hinton agar (MHA). Data collected in the questionnaire was entered and analysed in Epi Info software version 7.2.

RESULTS

In 101 samples, 116 organisms were isolated. In 14 samples no organism was isolated, which can be attributed to the viral aetiology, parasites and anaerobes. Acute Osteomyelitis (AOSM) was found to be more common in the age group of 1-10 years, whereas chronic osteomyelitis (COSM) was found more commonly in 21-30 and 31-40 years age group. Male to Female ratio was 2.2:1. Bones involved in AOSM and COSM were mostly femur followed by tibia and humerus. *S. aureus* was the most predominant isolate. All the isolates of *S. aureus* showed 100% sensitivity to Vancomycin, Amikacin, Netilmicin, Chloramphenicol. Out of 48 isolates of *S. aureus*, 37.50% were MRSA, 6.25% were ICR, 14.58% were MRSA+ICR found.

CONCLUSIONS

Osteomyelitis is found to be highest in third decade, with the males being predominantly affected. Acute osteomyelitis is predominantly seen in children, whereas chronic osteomyelitis in adults. Even though *Staphylococcus aureus* has always remained the most common etiological agent of osteomyelitis, increasing infections due to Gram negative bacilli and even poly-microbial infections are gaining importance. MRSA infection is known to increase post-operative complications. Introduction of MBL or carbapenemase production in Gram negative bacilli is a matter of great concern. Timely knowledge of aetiology and antimicrobial resistance pattern of osteomyelitis isolates can help in rational use of antibiotics and control of drug resistance.

KEY WORDS

Osteomyelitis, *Staphylococcus aureus*, MRSA, Long Bones, Males

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BACKGROUND

Bone and joint infections are painful for patients and frustrating for them and their doctors.[1] The term osteomyelitis (OSM) was first used by the French surgeon Edouard Chassaignac in 1852, who defined the disease as an inflammatory process accompanied by bone destruction and caused by an infecting microorganism.[2] This disease is differentiated according to the aetiology, pathogenesis, and degree of bone involvement, as well as age and the immune condition of the patient. It can involve different structures such as the bone marrow, cortex, periosteum, and parts of the surrounding soft tissues, or remain localized. The clinical manifestation and the natural history of OSM depend on several factors like age of patients, site of infection, virulence of infecting organism and the patients resistance.[3] OSM mostly affects the growing ends of long bones and it is more common in the lower extremity at metaphysis of femur and proximal end of tibia.[4] Historically, acute haematogenous osteomyelitis has been described in prepubertal children. It involves mostly the metaphysis of long bones (particularly tibia and femur), in most cases as a single focus. Although rare in adults, it most frequently involves the vertebral bodies.[1] Osteomyelitis due to local spread from a contiguous contaminated source of infection follows trauma, bone surgery, or joint replacement. It implies an initial infection that gains access to bone. It can occur at any age and can involve any bone.[1] Historically, osteomyelitis has been categorized as acute, subacute or chronic based on the time of disease onset (i.e., occurrence of infection or injury). The duration of symptoms of infection is in fact associated with peculiar anatomo-pathological findings and clinical and diagnostic features and influences the therapeutic decisions. Acute osteomyelitis is diagnosed within 2 weeks after disease onset, subacute osteomyelitis within one to several months and chronic osteomyelitis after a few months.[2]

Osteomyelitis in children is most often acute and secondary to haematogenous spread. The diagnosis can usually be made from the clinical signs, but requires a high index of suspicion. In neonates and infants, osteomyelitis has certain peculiar features.[3] The subacute and chronic forms of osteomyelitis usually occur in adults. Generally, these bone infections are secondary to an open wound, most often an open injury to bone and surrounding soft tissues. Localized bone pain, erythema and drainage around the affected area are frequently present. The cardinal signs of subacute and chronic osteomyelitis include draining sinus tracts, deformity, instability and local signs of impaired vascularity, range of motion and neurologic status.[2] The majority of osteomyelitis cases in adults are generally chronic in nature and are associated with a traumatic insult to the involved area. The diagnosis of osteomyelitis is first suspected on clinical grounds. The most important step, is to isolate the offending organisms so that appropriate antimicrobial therapy can be chosen.[1]

Other conditions that may be confused with OSM are cellulitis, septic arthritis, trauma and bone infarction. Acute osteomyelitis can respond to antibiotic treatment alone. Chronic osteomyelitis is associated with avascular necrosis of bone and formation of sequestrum (dead bone); Surgical debridement is therefore necessary for cure in addition to antibiotic therapy.[1]

Thus, because of the changes in the manifestations, epidemiology, and etiological agents, it is important to make a precise microbiological diagnosis. It is important to know microbiological aetiology in different types of osteomyelitis in our region. Early antibiotic treatment, before extensive destruction of bone or necrosis, produces the best results. Hence, the study was done to evaluate the microorganisms causing OSM along with their antimicrobial susceptibility pattern.

METHODS

The study was carried out in department of Microbiology at a tertiary care hospital in Nagpur region of Central India, over the period of 2 years. Pus and bone aspirate were collected from clinically and radiologically diagnosed 115 patients of osteomyelitis, attending outpatient department and/or admitted to wards of the hospital. Sample size of 115 patients was selected on the basis of previous articles related to our topic and statistician consultation. Data collected in the questionnaire were entered and analysed in Epi Info software version 7.2[5]. Categorical data was analysed by means of mean, standard deviation and quantitative data by proportion and percentage. The group differences were tested using chi-square, or others depending on the type of variable. p- value < 0.05 was considered to derive a level of significance.

Diagnosis was made on the basis of duration, as acute OSM and chronic OSM. Acute OSM characterised by systemic illness, absence of bony radiological changes at presentation, history less than 10 days. Chronic OSM characterised by systemic illness present or absent depending on presentation, Bony radiological changes in MRI and X-ray at presentation, history of previous episode or episodes of infection. Though it is a deep-seated infection we did not include CBC, CRP, ESR in our study, since it was not having any correlation with any parameter and was not significant. Specimens were processed for isolation of aerobic bacteria, Mycobacteria and Fungi by standard Microbiological techniques.[6] Pus sample was inoculated on the following media[6] Blood agar (BA), MacConkey agar (MA), Chocolate agar (CA), Sabouraud dextrose agar (SDA), Lowenstein Jensen medium (LJ). Isolates were identified by various biochemical reactions. Each isolate was subjected to antimicrobial susceptibility test as per CLSI guidelines[7] by Kirby-Bauer disk diffusion technique on Mueller Hinton agar (MHA).[8] In our study maximum isolates were of *Staphylococcus aureus*, so MIC of Vancomycin was done by E-strip. We have done disk diffusion test for other antibiotics, since it is recommended for antimicrobial susceptibility test according to CLSI guidelines. Tobramycin is tested for Gram negative organisms and *Staphylococcus aureus* as a third line drug for deep seated infections according to CLSI guidelines.

RESULTS

Organism	AOSM (n=20)			COSM (n=96)		
	Single	Mixed	Total (%)	Single	Mixed	Total (%)
S. aureus	08	--	08 (40.00)	40	--	40 (41.67)
S. epidermidis	--	01	01 (5.00)	02	--	02 (2.08)
E. faecium	--	--	--	02	--	02 (2.08)
K. pneumoniae	01	02	03 (15.00)	12	05	17 (17.41)
C. koseri	--	01	01 (5.00)	03	03	06 (6.26)
E. coli	--	01	01 (5.00)	01	03	04 (4.17)
S. Typhi	01	--	01 (5.00)	01	--	01 (1.04)
P. mirabilis	--	01	01 (5.00)	--	--	--
E. cloacae	--	--	--	01	--	01 (1.04)
A. baumannii	01	02	03 (15.00)	07	03	10 (10.42)
P. aeruginosa	01	--	01 (5.00)	03	06	09 (9.38)
A. lwoffii	--	--	--	--	01	01 (1.04)
S. maltophilia	--	--	--	--	01	01 (1.04)
C. albicans	--	--	--	02	--	02 (1.04)
Total			20			96

Table 1. Pathogens Isolated Singly and in Mixed Cultures in OSM

Antibiotics/ Organisms (N)	Resistance n (%)								
	P	AMP	AMC	CZ	CX	CAZ	CTX	CXM	CPM
S. aureus (48)	47 (97.92)	-	-	-	25 (52.08)	-	-	-	-
S. epidermidis (3)	3 (100)	-	-	-	1 (33.33)	-	-	-	-
E. faecium (2)	2 (100)	2 (100)	-	-	-	-	-	-	-
K. pneumoniae (20)	-	20 (100)	19 (95)	8 (40)	15 (75)	15 (75)	14 (70)	17 (85)	12 (85)
C. koseri (7)	-	6 (85.71)	7 (100)	6 (85.71)	6 (85.71)	6 (85.71)	6 (85.71)	6 (85.71)	6 (85.71)
E. coli (5)	-	5 (100)	5 (100)	3 (60)	4 (80)	4 (80)	4 (80)	4 (80)	4 (80)
S. Typhi (2)	-	2 (100)	2 (100)	1 (50)	1 (50)	1 (50)	1 (50)	1 (50)	1 (50)
P. mirabilis (1)	-	1 (100)	00	00	00	00	00	00	00
E. cloacae (1)	-	1 (100)	1 (100)	00	1 (100)	1 (100)	1 (100)	1 (100)	1 (100)
A. baumannii (13)	-	-	-	-	-	13 (100)	13 (100)	-	13 (100)
P. aeruginosa (10)	-	-	-	-	-	6 (60)	-	-	7 (70)
A. lwoffii (1)	-	-	-	-	-	1 (100)	13 (100)	-	13 (100)
S. maltophilia (1)	-	-	-	-	-	1 (100)	-	-	-

Table 2a. Antimicrobial Resistance Pattern among the Isolates of OSM

P- Penicillin, AMP- Ampicillin, AMC- Amoxyclavulanate, CZ- Cefazolin, CX- Cefoxitin, CAZ- Ceftazidime, CTX- Cefotaxime, CXM- Cefuroxime, CPM- Cefipime

Antibiotics/ Organisms (N)	Resistance n (%)									
	PI	PIT	AT	IPM	CL	GEN	HLG	AMK	TOB	E
S. aureus (48)	-	-	-	-	-	4 (8.33)	-	00	3 (6.25)	17 (35.42)
S. epidermidis (3)	-	-	-	-	-	1 (33.33)	-	1 (33.33)	1 (33.33)	00
E. faecium (2)	-	-	-	-	-	-	2 (100)	-	-	2 (100)
K. pneumoniae (20)	11 (55)	6 (30)	14 (70)	1 (5)	-	6 (30)	-	4 (20)	5 (25)	-
C. koseri (7)	6 (85.71)	4 (57.14)	6 (85.71)	2 (28.57)	-	5 (71.43)	-	5 (71.43)	5 (71.43)	-
E. coli (5)	2 (40)	2 (40)	5 (100)	1 (20)	-	3 (60)	-	2 (40)	2 (40)	-
S. Typhi (2)	1 (50)	00	1 (50)	00	-	00	-	00	00	-
P. mirabilis (1)	00	00	1 (100)	00	-	00	-	00	00	-
E. cloacae (1)	1 (100)	00	1 (100)	00	-	00	-	00	1 (100)	-
A. baumannii (13)	12 (92.31)	11 (84.62)	6 (46.15)	5 (38.46)	-	8 (61.54)	-	7 (53.85)	8 (61.54)	-
P. aeruginosa (10)	4 (40)	6 (60)	4 (40)	1 (10)	1 (10)	6 (60)	-	6 (60)	5 (50)	-
A. lwoffii (1)	1 (100)	1 (100)	1 (100)	1 (100)	-	00		00	00	
S. maltophilia (1)	-	-	-	-	-	-		-	-	

Table 2b. Antimicrobial Resistance Pattern among the Isolates of OSM

PI- Piperacillin, PIT- Piperacillin tazobactam, AT- Aztreonam, IPM- Imipenem, CL- Colistin, GEN- Gentamycin, HLG- High level Gentamycin, AMK- Amikacin, TOB- Tobramycin, E- Erythromycin

Antibiotics/ Organisms (N)	Resistance n (%)									
	NET	TET	CIP	LEVO	CD	CHL	RIF	LZ	VAN	PB (300)
S. aureus (48)	00	6 (12.50)	18 (37.50)	-	11 (22.92)	00	27 (56.25)	1 (2.08)	00*	
S. epidermidis (3)	1 (33.33)	1 (33.33)	2 (66.67)	-	1 (33.33)	00	3 (100)	00	00*	
E. faecium (2)	-	2 (100)	2 (100)	-	-	00	2 (100)	-	00*	
K. pneumoniae (20)	5 (25)	7 (35)	4 (20)			5 (25)				
C. koseri (7)	5 (71.43)	4 (57.14)	5 (71.43)			2 (28.57)				
E. coli (5)	2 (40)	3 (60)	4 (80)			2 (40)				
S. Typhi (2)	00	1 (50)	2 (100)			00				
P. mirabilis (1)	00	1 (100)	1 (100)			00				
E. cloacae (1)	1 (100)	00	00			00				
A. baumannii (13)	-	-	8 (61.54)	-	-	-	-	-	-	00
P. aeruginosa (10)	5 (50)	-	4 (40)	-	-	-	-	-	-	00
A. lwoffii (1)	-	-	00	-	-	-	-	-	-	00
S. maltophilia (1)	-	-	-	00	-	00	-	-	-	-

*Vancomycin MIC was performed by E-strip

Table 2c. Antimicrobial Resistance Pattern among the Isolates of OSM

NET- Netilmicin, TET- Tetracycline, CIP- Ciprofloxacin, LEVO- Levofloxacin, CD- Clindamycin, CHL- Chloramphenicol, RIF- Rifampicin, LZ- Linezolid, VAN- Vancomycin, PB- Polymyxin B

Organisms	No. of Isolates	ESBL Producer (%)	AmpC Producer (%)	ESBL + AmpC (%)	MBL Producer (%)
K. pneumoniae	20	04 (20)	03 (15)	03 (15)	00
C. koseri	07	01 (14.29)	00	00	01 (14.29)
E. coli	05	02 (40)	00	00	01 (20)
S. Typhi	02	00	00	00	00
P. mirabilis	01	00	00	00	00
E. cloacae	01	00	01 (100)	00	00
A. baumannii	13	1 (7.69)	0 (0)	0 (0)	3 (23.08)
P. aeruginosa	10	0 (0)	0 (0)	0 (0)	1 (10)
A. lwoffii	01	0 (0)	0 (0)	0 (0)	0 (0)
S. maltophilia	01	0 (0)	0 (0)	0 (0)	1 (100)
Total	61	8 (13.11)	4 (6.57)	3 (4.92)	7 (11.48)

Table 3. β Lactamase Production in the Isolates of OSM

Out of 115 cases of osteomyelitis, 98 (85.22%) cases were of chronic osteomyelitis and 17 (14.78%) were of acute osteomyelitis. Osteomyelitis was more common in males 79 (68.70%) as compared to females 36 (31.30%). However, the difference was not statistically significant (p>0.05) using pooled out data for up to 20 years, 21-40 years, >40 years. It is observed that 21-30 years age group patients were affected the most (26.96%). Long bones were maximally affected in osteomyelitis. Femur was the most common bone affected comprising (60.87%), followed by tibia (27.83%), humerus (5.22%).

DISCUSSION

The present study was carried out in 115 clinically diagnosed patients of Osteomyelitis (OSM). In 101 samples, 116 organisms were isolated. And in 14 samples no organism was isolated. No growth can be attributed to the viral aetiology, parasites and anaerobes. The major systems of classification presently are Waldvogel classification[9] and Cierny-Mader staging system.[10] In the present study, AOSM was found to be more in the age group of 1-10 years of age group, whereas

COSM was found more in 21-30 and 31-40 years of age group. In the present study, prevalence of OSM was found to be more in males (68.70%) as compared to females (31.30%) with Male to Female ratio was 2.2:1. Similar result was observed by Carvalho VC et al (2012)[11] affecting 63.4% males and 36.6% females with OSM. Similar result was obtained by Waldvogel et al (1970)[9] with Male to Female ratio was 2:1, Hassani U et al (2014)[12] observed male predominance with Male to Female ratio was 1.95:1, and Wadekar MD et al (2014)[13] observed Male to Female ratio was 2.7:1. While Izadi et al (2012)[14] observed, prevalence of COSM was (81.7%) in males and (18.3%) in females, Ali M et al (2014)[15] noted incidence of OSM in males (84%) and in females (16%).

In the present study, bones involved in AOSM were femur (70.59%), tibia (11.76%), humerus (11.76%). whereas in COSM were femur (59.18%), tibia (30.61%). Similarly, Ali M et al (2014)[15] observed bones involved in COSM were femur (46%), tibia (30%), humerus (4%). Wadekar MD et al (2014)[13] observed femur (48%), tibia (23%), humerus (9%) and ulna (4%). Whereas, Izadi et al (2012)[14] found COSM mostly affected tibia (33%), femur (27). Out of the 16 culture positive samples from AOSM, 20 organisms were isolated. *S. aureus* leads followed by *K. pneumoniae* and *A. baumannii*. Carvalho VC et al (2012)[11] found *A. baumannii* (21.4%), *P. aeruginosa* (19.8%), *K. pneumoniae* (8.2%) and E.coli (4.9%) in AOSM. Mirnejad R et al (2008)[16] found *S. aureus* (55.9%), Klebsiella spp. (14.8%), Coagulase negative Staphylococcus (7.4%) Acinetobacter spp. (3.7%) as cause of AOSM. Craigen MAC et al (1992)[17] observed *S. aureus* (88.2%), *S. pyogenes* (3.8%) and E.coli (0.6%) isolates in AOSM.

In the present study, out of the 85 culture positive samples from COSM, 96 organisms were isolated. *Staphylococcus aureus* 40 (41.67%) was the most common isolated followed by *K. pneumoniae* and *A. baumannii*. Among the fungal agents, Candida albicans was isolated. In this study, *S. aureus* was the most predominant isolate as also seen in other studies such as Rahbar M et al (2010)[18] as (26.3%), Wadekar MD et al (2015)[19] as (32.9%), Izadi M et al (2012)[14] as 48.9%, Wirbel R et al (2014)[20] as (74%). Ali M et al (2014)[15] observed *S. aureus* (58%), Coagulase negative Staphylococcus (14%). Thus, *Staphylococcus aureus* has been found to be the major etiological agent in our study, which is similar to other studies. Three cases in the present study were of sickle cell disease presenting with chronic osteomyelitis. Of which two cases yielded Salmonella Typhi from the pus aspirate and in one case *Staphylococcus aureus* was isolated. Thanni LOA et al (2006)[21] isolated 304 cases of COSM with sickle cell disease, out of which 129 were *S. Typhi* and 82 were *S. aureus*.

All the isolates of *S. aureus* showed 100% sensitivity to Vancomycin, Amikacin, Netilmicin, Chloramphenicol and resistant to Penicillin G, Rifampicin. Ali M et al (2014)[15] found *S. aureus* was 100% sensitive to Vancomycin, highly resistant to Cephalosporins, least sensitive to Ciprofloxacin, Amikacin and Gentamicin. Izadi et al (2012)[14] found *S. aureus* was most sensitive to Vancomycin (97.7%) and least sensitive to Penicillin (7%). Wadekar MD et al (2014)[13] observed that in COSM *S. aureus* was (100%) sensitive to Vancomycin, (97.1%) to Linezolid and resistant to Ciprofloxacin, Erythromycin, Gentamicin, Clindamycin and Amikacin. Increasing resistance to Penicillin has been observed over the years. The studies in literature clearly show this resistance pattern. In the study done by Izadi et al (2012)[14] 93% of the *S. aureus* were found resistant to penicillin.

In the present study, out of 48 isolates of *S. aureus*, 37.50% were MRSA, 6.25% were ICR, 14.58% were MRSA + ICR found. By E-strip method, 95.83% of the *S. aureus* isolates were sensitive and 4.17% were intermediate sensitive (IS) to Vancomycin. Ali M et al (2014)[15] observed 42% of MRSA strains in COSM. Wadekar MD et al (2015)[19] found 40% MRSA in COSM. Izadi M et al (2012)[14] found 75% of MRSA in COSM. Whereas, Wirbel R et al (2014)[20] observed 10% of MRSA in COSM. The probable reason for this can be, the difference in location of samples and consequently difference in strains.[14] *K. pneumoniae* showed 100% resistant to Ampicillin, followed by Amoxyclav, Cefuroxime, Cefoxitin and Ceftazidime. Similarly, Ali M et al (2014)[15] observed *K. pneumoniae* was 100% resistant to Ampicillin, followed by Gentamicin and Cefuroxime. 100% sensitive to Imipenem followed by Amikacin, Ciprofloxacin, Cefotaxime and Ceftazidime. Whereas, Wadekar MD et al (2014)[13] found *K. pneumoniae* was 100% resistant to Ampicillin, Gentamicin, followed by Cefotaxime, Amikacin and 78.5% sensitive to Imipenem.

A. baumannii were 100% sensitive to Polymyxin B (300), followed by Imipenem, Aztreonam and Amikacin. *A. lwoffii* was sensitive to Gentamicin, Amikacin, Tobramycin, Ciprofloxacin and Polymyxin B (300). Carvalho VC et al (2012)[11] observed that *A. baumannii* was sensitive to Imipenem (62%), Gentamicin (54%) and Amikacin (27%). *P. aeruginosa* isolates were 90% sensitive to Imipenem, 60% to Ciprofloxacin, Piperacillin, Aztreonam and 40% sensitive to Piperacillin-Tazobactam, Amikacin, Gentamicin. Ali M et al (2014)[15] reported that *Pseudomonas aeruginosa*, was 100% sensitive to Imipenem, 60% to Ceftazidime, 40% to Amikacin and Ciprofloxacin. Wadekar MD et al (2014)[13] observed that *Pseudomonas aeruginosa*, was 76.40% sensitive to Imipenem, 58.80% to Amikacin.

In the present study, ESBL rates for *K. pneumoniae* isolates is 20 %, whereas Wadekar MD et al (2015)[19] observed that ESBL rates were 85.7% for *K. pneumoniae* isolates and 75% for Enterobacter isolates in their study. In this study we observed, 4 (6.57%) AmpC producers, while AmpC production reported by Rawat et al (2013)[22] was 20.8% and 36.6% by Haider et al (2014).[23] All these were Inducible AmpC producers. However, Haider et al (2014)[23] have found only 21.7% Inducible AmpC producers and 78.3% non-inducible AmpC producers, which is an alarmingly high percentage of derepressed mutants.

Co-expression of different beta lactamase enzymes were found in the present study. There were three strains showing ESBL and AmpC co-production (4.92%). Rawat et al (2013)[22] have found that ESBL and AmpC were co-produced by 25% isolates in their study. Out of 13 Acinetobacter spp. isolated, ESBL was found in 1 (7.69%). Goel V et al (2013)[24] found that 17.9% of *A. baumannii* to be ESBL producers. In our study we observed that 23.08% *A. baumannii* and 10% *P. aeruginosa* were MBL producers, whereas Goel V et al (2013),[24] found that 48.72% *A. baumannii* and 53.85% *P. aeruginosa* were plasmid mediated MBL enzyme producing strains detected by Imipenem-EDTA disk method. OSM resulting from fungi is uncommon.[25] In the present study, 2 (1.72%) isolates of Candida albicans were isolated from cases of OSM.

CONCLUSIONS

Osteomyelitis is found to be highest in third decade, with the males being predominantly affected. Acute osteomyelitis is predominantly seen in children, whereas chronic osteomyelitis in adults. Even though *Staphylococcus aureus* has always remained the most common etiological agent of osteomyelitis, increasing infections due to Gram negative bacilli and even poly-microbial infections are gaining importance. MRSA infection is known to increase post-operative complications. Introduction of MBL or carbapenemase production in Gram negative bacilli is a matter of great concern. Timely knowledge of aetiology and antimicrobial resistance pattern of osteomyelitis isolates can help in rational use of antibiotics and control of drug resistance.

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