RESISTANCE & RESISTOGRAMS OF THE MAJOR HUMAN MAMMARY ASSOCIATED BACTERIAL PATHOGENS.

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Abstract

One hundred forty-four women patients with lactional mastitis were diagnosed. Milk samples, under sterile conditions were collected, examined by direct wet, direct stained preparation as well as culturing used standardized methodology monomicrobic was with rate of 65.3%, sterile primary plate culture was 31.2% and dimicrobic 3.5%. Antibiogram studies by disc diffusion technique with 15 different antibacterial chemotherapeutics were done to assess resistance and resistogrames if multiple resistance. The major dominant mammary associate bacterial pathogens were *S. aureus* (49.49%) and *K pneumoniae* (13.13%).

The resistogram profiles were; double, triple, associated and multiple drug resistance (MR- I 5R) were noticed among these isolates.

Drug resistance lacting mastitis is problem that nasciatate more clinical and laboratory attention, it is being a real alert needs management by specialist.

Introduction

Several studies have been published about human puerperal mastitis or lactational mastitis (Fetherson, 1998). The threat from lactimal mastitis is due to the possible complications like breast abscess, septicemia and toxic shock syndrome (Foxman *et al.*, 1994). The infection routes for mammary glands are the; reterograd route (WHO 2000, Fetherston 2001), haematogenous (Rench & Baker 1989), and lymphogenous (Gupta *et al.*, 1982). As a pathogen it should find port of entry and multiply, it as to avoid the mammary defence mechanisms like leukocytes (Semba and Neville, 1999), IL 8 in high concentrations (Semba *et al*, 1999), Lacto fermin in high concentrations (Semba *et al*, 1999), C (Ogundele, 1999) and secretory IgA (Kelleher and Lonnerdad, 2001), when Pathogen has escaped the defence mechanisms, it will begin to cause tissue damage by virulence factors (Fetherston, 2001). The repeated antibiotics abuse in mastitis and/or antibiomia (Devereux, 1970, Merchant, 2002) As will as drug resistance (Matheson *et al* 1980; Hook et al 1993), that may cause failure in treatment.

The objectives of the present work were at reporting major mammary pathogens and their antibiotics resistance and the resistogrames.

Materials and Methods

One hundred forty-four milk samples were collected from clinically proven women mastitis (Thomson *et al*, 1984; Norak *et al*, 2000). Samples were cultured using Standard culture techniques (Baron et al, 1994), direct preparations characterized biochemically (Macfaddin 2000, Baron *et al*, 1994) and subjected to antibiogram, of the mammary isolates using disc diffusion technique (Bauer *et al*, 1966). The associated mammary pathogen were scored following the below-mentioned criteria (Shnawa, 1996);

i- Presence of inflammatory cell infiltration.
ii- Notable numbers of microbes in direct milk preparations.
iii- Growth density and purity in primary plate cultures.
iv- a- Growth of pure dense, one morphotypes means single infection.

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iv- a- Growth of pure dense, one morphotypes means single infection.

b- Presence of Equivalent populations of two colony morphotypes means double infections.

v- *S. epidermides* appeared in normal twenty non mastitis as well as mastitis women so it will excluded from the associated mammary pathogens.

Results

I- Mammary Associated Bacterial Pathogens;

Out of the assessed 144 mastitis milk samples 99 were culture positive and 45 were culture negative. Among which; *S. aureus* was 46/99 (46.47%) *St. epidermidis* 13:99 (13.13%), *K. pneumoniae* 11/99 (11.11%), *St. fecalis* 7/99 (7.07%), *St. pyogenes* 5/99 (5.05%) and *E. coli* 5/99 (5.05%).

Thus the major pathogens were *S. aureus* (46.47%) *K. pneumonia.* (11.11%) .

II- Drug Resistance;

S. aureus were highly resistance to Amx, Am, CT, Cp and Top.

K. pneumoniae showed high resistance rates to; Am, Cp, OB, E, RA, S, and VA.

St. pyogense expressed marked resistance to Am, CT and OB. While *St. viridanse* noably resistance to CT, TOB, S, TE and CM.

P. aeruginosa showed multidrug resistance to most of the drugs (Table 1).

III- The Resistogrames of the major pathogens;

III- 1: *S. aureus*;

No single drug resistance can be matched. Associated double drug resistance were noted as Erythromycin -Cloxacillin, Clexaciam-Culistin sulfate Multiple resistant of up to 3, 4, 5 to 12 drugs were also evident (Table 2).

III-2: *K. pneumoniae*;

Neither single nor double associated drug resistance can be matched. However, multiple resistance was mapped up to 13 drugs (Table 4).

IV- The Resistograms of the minor pathogen'

V- 1: *St. pyogenes*

No single drug resistance was noted. Associated double drug resistance were noted to Am-ST. Multiple drug resistance up to seven antibioties 3)

IV- 2: *St. viridance*

Neither single nor associated double drug resistance were seen among these mammary isolates. Multiple drug resistance of up to: 6, 8 and 10 drugs (Table 3).

IV- 3: *E. coli*

Neither single nor double but multiple drug resistance were mapped. Resistances of 7,11 and 13 drugs were noted (Table 4).

IV- 4: *Ps. aeruginosa*;

No single, No double but multiple drug resistance of up to 9, and up to 12 drugs (Table 4).

IV- 5: *S. epidermidis*

Resistogrames were studied but, since it is questionable pathogen is not mentioned.

Discussion

Women lactional mastitis have motley been associated with mono-microbic casuals and lessely associated with di-microbic casuals (Niebyl *et al*, 1978; Thomson, 1982).

In 45:144 (31.2%) there were inflammatory cell infiltration, temperature rise, headache, redness and swelling of the mammary glands. However, the milk culture were negative, a finding which may be due to one or more of the followings;

1- Virus or fungal infection (WHO 2000).

2- Mycobacterial, Chiamydial and Mycoplasma infections (Thomsen *et al* 1983).

3- Antibody coated bacteria (Thomson 1982).

4- Non infections mastitis (Merchant 2002).

5- Presence of cell wall detective bacteria (Mattman, 1991).

Gram positive mammary pathogens were dominating the gram negative (Tables 2-7). *S. aureus* was among the major mammary pathogens (Devereux 1970; Thomsen *et al* 1934; Aabo *et al* 1990). *St. pyogenes* was found to be minor mammary pathogen (Table 1; 4) (Marshau *et al* 1975; Thomsen *et al* 1983).

E. coli were recovered in 5.05% which agreed with Thomsen *et al* (1984.) *K pneumoniae* was among the major pathogen, a finding that parallels that of Fetherston(2001) *P. aeruginosa* were recovered in 4.04% in this study. Flowing with this result Thomsen *et al* (1 984)lave indicated that *P. aeruginosa* constitute one of the important mammary pathogen and it was ascertained by Fetherston(2001)

The drug resistance among mammary pathogen (Tables 1-7). can be explained on the bases of the nature of the antibiotics and the nature of the pathogens as;

Resistance to B lactam antibiotics may be due to; i- Production of B lactamases encoded by chromosomal genes (Moellering 1993; Fournier et al 1999; ii- Tolerance to the antibiotics .such as that of S. aureus (Bradely et al 1980, Sabath 1980; iii- Chromosomal mutation in the gene encoding the Penicillin Binding Proteins leading to change (Henry 1993) and iv- Lose of cell wall containing porine as in the case of K pneumoniae (Martize *et al* 1999).

Resistance to Aminoglycosides like Tobromycin, Gentamycin and Streptomycin could be attributed to either of the followings;

i- Protein 12 encoding chromosomal gene mutation which acts as Streptomycin receptor (Metha and Champney,2002).

ii- Impaired drug permeability due to defective gene encoding the generation absorption energy (Miller *et al*, 1980).

iii- Production of drug modifying enzymes through changing of amino or carboxyl groups of the anti amino glycosides (Mandel 1984; Shanon and Phillips 1982).

The resistance to Erythromycin and Clindamycin can be due to acquiring other drug receptor on 50S ribosome on rRNA (Bingin *et al*, 2002).

Refadin resistance due to mutant B subunite gene on RNA polymerase which lead to non functional combination (Damon *et al*, 2002).

Tetracycline and vancomycin resistance can be as a result of permeability impairment of the bacterial cell membrane avoiding the intrence of the antibiotics (Farrell *et al* 1978; Stewart and Dublin 1994).

In chloramphenicol resistance, bacteria developed enzyme system that can modify the drug to non-functional inert form (Shaw, 1984).

P. aeruginosa drug resistance can be attributed to;

i- Presence of membrane associated protein that can transport the drug away of the cell render its ineffective in swish matrix (James, 1999).

ii- modification to non functional compound, charging target drug receptor or reduce drug permeability (Iyobo *et al*, 1994). Working with profile of bacterial mastitis with emphasis on drug

resistance and resistograms appeared to just like digging through in a virgin area the therapy irresponding lactional mastitis.

Finally, we can sum up these findings as;

i- Lactional mastitis is culture positive and culture negative.

ii- Monomicrobioc mastitis were dominating dimicrobic mastitis.

iii-*S. aureus* and *K. pneumoniae* were the major mammary pathogens.

iv- Double and multiple drug resistance mammary isolates are rather common.

Being a theraputic problem one has to be borne in his mind that it should be to be managed

Table 1: The Antibiotic Resistance of Mammary Pathogens

Antibiotics	Major Pathogens		Minor Pathogens			
	S. aureus	*K. pneumeniae*	*St. pyogenes*	*St. viridanse*	*E. coli*	*Ps. aeruginosa*
Amx	27:49(55.1%)	13:13(100)	2:5(40)	3:6(50)	5:5(100)	4:4(100)
Am	42:49(85.7%)	13:13(100)	3:5(60)	4:6(66.7)	5:5(100)	4:4(100)
C	2:49(4.5%)	1:13(7.7)	2:5(40)	0:6(0)	2:5(40)	4:4(100)
CO	12:49(24.5%)	1:13(7.7)	2:5(40)	0:6(0)	1:5(20)	2:4(50)
CM	9:49(18.4%)	11:13(84.6)	1:5(20)	4:6(66.6)	0:5(5)	4:4(100)
CP	18:49(36.7%)	12:13(92.3)	0:5(0)	1:6(16.5)	3:5(60)	2:4(50)
CT	44:49(91.8%)	7:13(53.8)	4:5(80)	5:6(83.7)	3:5(60)	2:4(50)
E	28:49(57.1%)	13:13(100)	2:5(40)	4:6(66.6)	2:5(40)	4:4(100)
GM	10:49(20.4)	2:13(15.4)	2:5(40)	2:6(33.4)	2:5(3.34)	4:4(100)
OB	31:49(63.3)	13:13(100)	3:5(60)	4:6(66.6)	5:5(100)	4:4(100)
RA	7:49(14.3)	13:13(100)	0:5(0)	4:6(66.6)	5:5(100)	4:4(100)
S	25:49(51)	3:13(33.1)	4:5(80)	4:6(66.6)	5:5(100)	2:4(50)
TE	17:49(32.7)	3:13(33.1)	2:5(40)	4:6(66.6)	2:5(40)	4:4(100)
TOB	15:49(30.6)	3:13(33.1)	2:5(40)	5:6(83.7)	3:5(60)	2:4(50)
VA	0:49(0.0)	13:13(100)	0:5(0)	0:6(0)	5:5(100)	4:4(100)

Table 2: *S. aurens* Resistograms

Number of R	Resistograms	Incidence %
2 R:	EOB, Am CO, OB CT	1: 49 (2.04)
3 R:	Am S CT	3: 49 (6.1)
	Am E CT	4: 49 (8.2)
	Am x E CT, Am x TE CT, Am OB CT, Am x Am co; each of	1:49 (2.04)
	Am x Am CT,	2: 49 (4.08)
	Am TE CT	1:49 (2.04)
4 R:	Am x Am OB CT	3: 49 (6.1)
	Am x Am S OB	1: 49 (2.04)
	Am S CP CT	1: 49 (2.04)
	Am TE OB CT	1: 49 (2.04)
5 R:	Am x Am S OB CT	1: 49 (2.04)
	Am E TE OB CT	1: 49 (2.04)
	Am E S TE CT	1: 49 (2.04)
6 R:	Am CO E S TE CT	2: 49 (4.08)
	Am x Am TE OB TOB CT	1: 49 (2.04)
	Am x Am E TE OB CT	2: 49 (4.08)
8 R:	CO GM ES OB TOB CPCT	2: 49 (4.08)
	Am x Am RA TE OB TOB CP CT	1: 49 (2.04)
	Am x Am ES OB TOB CP CT	1: 49 (2.04)
9 R:	Am x Am GM ES OB TOB CPCT	1: 49 (2.04)
	Am x Am CM RA ES OB CP CT	1: 49 (2.04)
10 R:	Am CO GM ES TE OB TOB CP CT	1: 49 (2.04)
	Am x Am, GM, CM, ES OB TOB CP CT	2: 49 (4.08)
	Am x Am CO CM SE OB TOB CP CT	1: 49 (2.04)
	Am x Am CM RA E S TE OB CP CT	1: 49 (2.04)
11 R:	Am x Am CO GM CM ES OB TOB CP CT	2: 49 (4.08)
	Am x Am RA E S TE OB TOB CP CTC	1: 49 (2.04)
	Am x Am GM RA E S OB TOB C CP CT	1: 49 (2.04)
12 R:	Am x Am CO CM RA E S TE OB C CP CT	1: 49 (2.04)
	Am x Am CO GM CM RA E S OB TOB CP CT	

Table 3: *S. pyogenes* and *S. viridanse* Resistograms

Frequency Number of R		Resistograms	Incidence %
S. pyogenes	2 R	Am x S	1: 5 (20)
	6 R	GM CM E TE TOB CT	1: 5 (20)
	7 R	Am GM E S TE OB CT	1: 5 (20)
		Am CO S OB TOB CP CT	1: 5 (20)
		Am x Am CO S OB CP CT	1: 5 (20)
S. viridans	6 R	Gm CM E TE TOB CT	2: 6 (33.3)
		Am x Am S OB TOB CT	2: 6 (33.3)
	8 R	Am x Am CM RA S E TE OB	1: 6 (16.7)
	10 R	Am CM RA S E TE OB TOB CP CT	1: 6 (16.7)

Table 4: *K. pneumonia, E. coli* and *Ps. aurginosa* Resistograms

Frequency Number of R		Resistograms	Incidence %
E. coli	7 R:	Am x Am CM RA VA E OB	2:5 (40)
	11R:	Am x Am CM GM RA VA E TE OB TOB CP CT	1:5 (20)
	13R:	Am x Am GM CM RA VA ES OB TOB CB CT C	1:5 (20)
	15R:	Am x Am CO GM CM RA VA ES TE OB TOB CB CTC	1:5 (20)
K. pneumoniae	7 R:	Am x Am RA VA E OB CT	1:13 (7.7)
	8 R:	Am x Am CM RA VA E OB CP	2:13 (15.4)
		Am x Am CM RA VA E OB CP	1:13 (7.7)
	9 R:	Am x Am CM RA VA E OB CP CT	5:13 (38.5)
		Am x Am CM CO RA VA E OB CP	1:13 (7.7)
	10R:	Am x Am CM RA VA ES OB CP CT	1: 13 (7.7)
	12R:	Am x Am GM CM RA VA ES TE OB TOB CP	1:13 (7.7)
	13R:	Am x Am GM CM RA VA E S TE OB TOB C CP	1:13 (7.7)
P. aeruginosa	9 R:	Am x Am CM RA VA ES OB TOB	1:4 (25.0)
	12R:	Am x Am CO CM RA VA ETE OB C CP CT	2:4 (50.0)
		Am x Am GM CM RA VA E STS OB TOB CP	1:4 (25.0)

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المقاومة وهيئة المقاومة المتعددة للادوية بين البكتريا المشتركة مع خمج الغدة النفاسي

الخلاصة

تم تشخيص 144 حالة خمج غدة لبنية نفاسي وجمعت عينات تحت الظروف المعقمة من هؤلاء النسوة ثم زرعت وحضر لها تحضير مباشرة ورطبة ومصبوغة باستخدام تقنيات قياسية.

كانت نسبة 65.3% احادية المسبب المشارك و 35.9% او ثنائية المسبب المشارك و 32.1% سلبية الزرع. جرت دراسة حساسية للعزول لمضادات البكتريا بطريقة الانتشار من القرص ولخمس عشر دواء مختلف كان من بين المسببات الاكثر شيوعاً *S. aureus* 49.49% و *K. pneumoniae* 13.13%.

تراوحت المقاومة بين 2-100% لمختلف مضادات البكتريا العلاجية وكانت هيئات المقاومة ثنائية او متعددة وكان مدى عدد المضادات 2-15 دواء. وتعد مشكلة المقاومة في خمج الغدة اللبنية النفاسي بمثابة انذار يحتاج لمعالجة من قبل اختصاصيين بهذا المجال.