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Evaluation of Prevalence of Inducible Clindamycin Resistance among Coagulase Negative *Staphylococci* (CoNS) Isolated from Various Clinical Samples in a Tertiary Care Hospital of North India

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A B S T R A C T

Coagulase-negative *Staphylococci* (CoNS) have emerged as predominant pathogens in hospital and community acquired infections. Clindamycin is a good therapeutic agent in treatment of skin, soft tissue as well as serious infections caused by both methicillin sensitive and resistant *Staphylococci*. However, strains with inducible clindamycin resistance often lead to therapeutic failure. Hence, each laboratory should be aware of the local prevalence of this resistance among clinical staphylococcal isolates. Therefore, the present study was done to evaluate the prevalence of inducible clindamycin resistance among CoNS isolates. A total of 152 CoNS isolated from clinical samples were evaluated for antimicrobial susceptibility testing using Kirby Bauer disk diffusion method and simultaneously D-zone test was interpreted by putting clindamycin and erythromycin disks adjacent to each other. Out of 152 CoNS isolates tested, 38.8% were methicillin resistant (MRCoNS) and 61.2% were methicillin sensitive (MSCoNS), with 33.6% hospital acquired strains and 66.4% community acquired strains. The prevalence of inducible clindamycin resistance was found to be 14.5%, with higher prevalence among MRCoNS (20.3%) as compared to MSCoNS (10.8%), also higher prevalence among community acquired (68.2%) as compared to hospital acquired strains (31.8%). All isolates with inducible resistance were 100% sensitive to vancomycin, linezolid and teicoplanin. To conclude, D-zone test should be performed routinely for appropriate prescription of clindamycin therapy for isolates with true clindamycin sensitivity only.

Introduction

Coagulase-negative *Staphylococci* (CoNS), which are the normal skin flora, have emerged as predominant pathogens in nosocomial as well as community acquired infections (Kloos and Bannerman, 1994; Roopa and Biradar, 2015). Risk factors include patients with intravascular catheters or prosthetic devices. These bacteria usually infect immune compromised hosts, such as premature newborns and patients with leukemia or other malignant diseases (Sheikh and Mehdinejad, 2012). CoNS species have been documented

as a cause of nosocomial bacteremia, infections of wound, urinary tract, central nervous system shunt, endocarditis, surgical site infections, endophthalmitis, peritonitis, bone and joint as well as pediatric and neonatal infections (Tayyar *et al.,* 2015). These infections are not easy to treat because of the risk factors and the multiple drug resistance displayed by these organisms (Asangi *et al.,* 2011).

The increasing prevalence of methicillin resistance among *Staphylococci* is a therapeutic threat (Novotna *et al.,* 2005). The macrolides, lincosamides (clindamycin) and type B streptogramins (MLS_B) are considered to be good alternative therapeutic agents available to address this issue. These agents are chemically distinct but exert similar action by binding to 50S ribosomal subunit inhibiting bacterial protein synthesis. Their unrestricted consumption has increased the rate of resistance to these drugs (Delialioglu *et al.,* 2005).

In *Staphylococci*, resistance to MLS_B antibiotics is usually caused either by active efflux mechanism encoded by *msrA* gene, affecting macrolides and type B streptogramins, which results in MS phenotype (resistance to macrolides and group B streptogramins and susceptibility to lincosamides); or by target site modification via 23S rRNA methylation encoded by *erm* genes which confers constitutive or inducible resistance to MLS_B agents (Fiebelkorn *et al.,* 2003). Strains with constitutive MLS_B resistance ($cMLS_B$) phenotype show resistance to all MLS_B drugs without any need to an inducer. In contrast, in inducible MLS_B resistance ($iMLS_B$), exposure to a strong methylation inducer (e.g., erythromycin) results in the expression of resistance to lincosamides and streptogramins B (Fiebelkorn *et al.,* 2003). However, it has been demonstrated that spontaneous mutations can transform $iMLS_B$ phenotype to $cMLS_B$, without the presence of an inducer (Lewis and Jorgensen, 2005).

In clinical practice, relying just on routine disk diffusion methods lead to misidentification of $iMLS_B$ phenotype as these appear erythromycin resistant and clindamycin sensitive *in vitro* when not placed adjacent to each other. This may result in inappropriate institution of clindamycin therapy leading to selection of constitutive *erm* mutants and consequently treatment failure (Thapa and Sapkota, 2016). On the other hand, considering all erythromycin-resistant strains as clindamycin resistant makes the effect of latter drug underestimate in infections caused by clindamycin susceptible isolates (MS phenotypes) (Aghazadeh *et al.,* 2015). Hence, using an appropriate method like double disk diffusion method (D-zone test) as recommended by Clinical and Laboratory Standards Institute (CLSI, 2015) for determination of $iMLS_B$ phenotypes would be very informative for better control of CoNS infections.

As reports on inducible clindamycin resistance, especially, among CoNS were scanty from our geographic area, therefore, the present study was undertaken to find out the prevalence of inducible clindamycin resistant CoNS isolated from various clinical samples in our hospital by using D-zone test.

Materials and Methods

A total of 152 consecutive, non-duplicate strains of CoNS isolated from clinical samples like pus, synovial fluid, blood, sputum and intravenous catheter received in the clinical bacteriology laboratory of department of Microbiology from both inpatients and outpatients attending Hind Institute of Medical Sciences, Mau, Ataria, Sitapur, were included in the study being conducted over a period of

1 year from August 2015 to July 2016. Other Gram positive bacterial isolates and duplicate CoNS isolates from the same patient were excluded from the study. The study was approved by Institutional Ethics Committee. The clinical isolates were identified as CoNS by performing standard microbiological procedures (Koneman *et al.*, 2006). Antibiotic susceptibility testing was performed on Mueller-Hinton agar (HiMedia Laboratories, Mumbai, India) by Kirby Bauer disk diffusion method as per CLSI guidelines. The antibiotic disks (HiMedia Laboratories, Mumbai, India) used were penicillin (10 units), gentamicin (10µg), amikacin (30 µg), doxycycline (30 µg), trimethoprim/sulfamethoxazole (1.25/ 23.75µg), cefoxitin (30µg), erythromycin (15µg), clindamycin (2µg), ciprofloxacin (5µg), chloramphenicol (30µg), linezolid (30µg) and teicoplanin (30 µg) (CLSI, 2015). D-zone test was performed simultaneously by placing erythromycin and clindamycin disks adjacent to each other at a distance of 15mm edge to edge (CLSI, 2015). After 18 hours of incubation at 35°C, blunting of the clindamycin zone of inhibition (D-shaped zone) proximal to the erythromycin disk was considered as positive D-zone test as it indicated an inducible type of resistance or $iMLS_B$ phenotype and resistance to both erythromycin and clindamycin indicated constitutive resistance or $cMLS_B$ phenotype (Figure 1 and 2 respectively). Isolates which were resistant to erythromycin and remained sensitive to clindamycin with circular zone of inhibition (no induction) were considered as negative D-zone test and were defined as having the MS phenotype (Figure 3).

Isolates showing resistance to 30 µg cefoxitin disk (zone of inhibition ≤ 24 mm) were reported as oxacillin resistant or methicillin resistant CoNS (MRCoNS) and those sensitive to 30 µg cefoxitin disk (zone of inhibition ≥ 25 mm) were reported as oxacillin sensitive or methicillin sensitive CoNS (MSCoNS). Susceptibility of MRCoNS to vancomycin was tested by agar dilution method as per CLSI guidelines. 10 µl of 0.5 McFarland bacterial suspensions was spot inoculated on Mueller-Hinton agar plates by using micropipette. The plates were analyzed after 24 hours of incubation at 35°C. Minimal inhibitory concentration (MIC) of vancomycin of ≤ 4µg/mL for CoNS was considered as susceptible to vancomycin. *Staphylococcus aureus* American Type Culture Collection (ATCC) 25923 was used to achieve quality control for antibiotic sensitivity tests (CLSI, 2015).

Statistical analysis

The collected data were statistically analyzed using SPSS Data Editor Software, Chicago, version 20. The statistical association was evaluated using Chi-square test and $p < 0.05$ was taken as statistically significant.

Results and Discussion

A total of 152 CoNS isolates were tested in our study. As shown in Figure 4, majority of these isolates were derived from pus samples (35.5%), followed by blood (22.4%), intravenous catheter (18.4%), synovial fluid (17.1%) and least from sputum samples (6.6%). As shown in Table 1, out of 152 CoNS isolates, only 31.6% were uniformly sensitive to both erythromycin and clindamycin disks, while 68.4% were erythromycin resistant, with predominantly MS phenotype (27.6%), followed by $cMLS_B$ (26.3%) and $iMLS_B$ phenotype (14.5%). Amongst 152 CoNS isolates tested, 59 (38.8%) were methicillin resistant (MRCoNS) and 93 (61.2%) were methicillin sensitive (MSCoNS), with 51 (33.6%) isolates derived from inpatients (hospital acquired strains) and 101 (66.4%) isolates derived from outpatients (community acquired strains). Amongst MRCoNS, $cMLS_B$ phenotype predominated

(49.2%, 29/59 isolates), followed by $iMLS_B$ (20.3%, 12/59 isolates) and MS phenotypes (18.6%, 11/59 isolates). However, the hospital acquired MRCoNS showed $cMLS_B$ phenotype predominantly (70.9%) and community acquired MRCoNS showed predominantly MS and $iMLS_B$ phenotype (28.6% each). These findings were found to be statistically significant ($p = 0.006$). Amongst MSCoNS, MS phenotype predominated (33.3%, 31/93 isolates), followed by $cMLS_B$ phenotypes (11.8%, 11/93 isolates) and $iMLS_B$ phenotype (10.8%, 10/93 isolates).

However, the hospital acquired MSCoNS showed predominantly $cMLS_B$ phenotype (50.0%) and community acquired MSCoNS showed predominantly MS phenotype (41.1%). This difference was found to be highly significant statistically ($p < 0.001$). The overall prevalence of inducible clindamycin resistance ($iMLS_B$ phenotype) amongst the CoNS isolates was found to be 14.5% (22/152 isolates), with its prevalence being found more among MRCoNS (20.3%, 12/59 isolates) as compared to MSCoNS (10.8%, 10/93 isolates), also its prevalence was more among community acquired strains (68.2%, 15/22 isolates) as compared to hospital acquired strains (31.8%, 7/22 isolates).

Table 2 depicted that the prevalence of $iMLS_B$ phenotypes predominated in isolates recovered from blood (20.6%, 7/34 isolates), followed by pus (18.5%, 10/54 isolates), synovial fluid (15.4%, 4/26 isolates) and intravenous catheter (3.6%, 1/28 isolates). None of the isolates recovered from sputum sample showed $iMLS_B$ phenotype. These findings were found to be statistically significant ($p = 0.029$).

Table 3 shows the comparative evaluation of the antibiotic susceptibility of MRCoNS and MSCoNS having inducible clindamycin resistance. It was found that all the isolates with inducible resistance phenotype were 100% sensitive to linezolid, teicoplanin and vancomycin, followed by sensitivity to amikacin (86.4%), doxycycline (81.8%), gentamicin (77.3%) and chloramphenicol (68.2%). This finding was statistically significant ($p = 0.041$). The MRCoNS isolates were more resistant to trimethoprim / sulfamethoxazole and ciprofloxacin as compared to MSCoNS isolates. These differences were found to be statistically significant ($p = 0.035$ and $p = 0.003$ respectively).

In our study, maximum CoNS isolates were derived from pus samples (35.5%), followed by blood (22.4%), intravenous catheter (18.4%) and synovial fluid (17.1%).

This finding is in agreement to another study which also reported maximum isolation of CoNS strains from pus and pus swabs (37.2%) followed by blood (25.2%) (Bansal *et al.*, 2012). However, in contrast to our findings studies from Jaipur and Uttarakhand reported maximum isolation of CoNS from blood followed by pus samples (Pal *et al.*, 2010; Juyal *et al.*, 2013).

Our study reported 38.8% MRCoNS and 61.2% MSCoNS, which is similar to another study that reported 35.6% MRCoNS and 64.4% MSCoNS (Bansal *et al.*, 2012). A study from Uttarakhand also reported higher prevalence of MSCoNS (71.6%) as compared to MRCoNS (28.4%) (Juyal *et al.*, 2013). However, the prevalence of MRCoNS in clinical samples has been reported between 55%-77% and even 86% in intensive care units, from different countries (Piette and Verschraegen, 2009).

Clindamycin is a good therapeutic agent for the treatment of both meticillin-resistant and -susceptible staphylococcal infections (Kloos and Bannerman, 1994).

Table.1 Distribution of CoNS isolates on the basis of their strain type and their susceptibility to cefoxitin and to erythromycin and clindamycin in D-zone test

CoNS isolates, N=152 (100%)	Strain type	Susceptible phenotype, N=48 (31.6%)	$cMLS_B$ phenotype, N=40 (26.3%)	MS phenotype (Negative D-zone test), N=42 (27.6%)	$iMLS_B$ phenotype (Positive D-zone test), N=22 (14.5%)	Chi-Square (χ^2) & *p value
MRCoNS N=59 (38.8%)	Hospital acquired, N=31 (52.5%)	2 (6.5%)	22 (70.9%)	3 (9.7%)	4 (12.9%)	$\chi^2 = 12.530$ p = 0.006
	Community acquired, N=28 (47.5%)	5 (17.8%)	7 (25.0%)	8 (28.6%)	8 (28.6%)	
MSCoNS N=93 (61.2%)	Hospital acquired, N=20 (21.5%)	6 (30.0%)	10 (50.0%)	1 (5.0%)	3 (15.0%)	$\chi^2 = 39.099$ p < 0.001
	Community acquired, N=73 (78.5%)	35 (47.9%)	1 (1.4%)	30 (41.1%)	7 (9.6%)	

CoNS = coagulase negative *Staphylococci*; N = Number of CoNS isolates; MRCoNS = Methicillin resistant CoNS i.e. isolates resistant to cefoxitin; MSCoNS = Methicillin sensitive CoNS i.e. isolates sensitive to cefoxitin; Hospital acquired strains = isolates recovered from inpatients; Community acquired strains = isolates recovered from outpatients; Susceptible phenotype = isolates sensitive to both erythromycin and clindamycin; $cMLS_B$ phenotype = isolates with constitutive resistance to clindamycin; MS phenotype = isolates with susceptibility to clindamycin and negative D-zone test; $iMLS_B$ phenotype = isolates with inducible resistance to clindamycin and positive D-zone test. *p value < 0.05 was considered as statistically significant.

Table.2 Distribution of CoNS isolates with inducible clindamycin resistance among clinical samples tested

Samples tested	$iMLS_B$ phenotype			Chi-Square (χ^2) & *p value
	Present in MRCoNS, N=12 (7.9%)	Present in MSCoNS, N=10 (6.6%)	Not Present**, N=130 (85.5%)	
Pus	9 (16.7%)	1 (1.8%)	44 (81.5%)	$\chi^2 = 16.807$, p = 0.032
Synovial fluid	1 (3.8%)	2 (7.7%)	23 (88.5%)	
Blood	2 (5.9%)	5 (14.7%)	27 (79.4%)	
Sputum	0 (0%)	0 (0%)	10 (100%)	
Intravenous catheter	0 (0%)	1 (3.6%)	27 (96.4%)	

CoNS = Coagulase negative *Staphylococci*. $iMLS_B$ phenotype = isolates with inducible clindamycin resistance. N = Number of CoNS isolates. MRCoNS = Methicillin resistant CoNS; MSCoNS = Methicillin sensitive CoNS. *p value < 0.05 was considered as statistically significant. **signifies presence of isolates having susceptible, constitutive resistance and MS phenotypes.

Table.3 Comparison of antibiotic susceptibility pattern among MRCoNS and MSCoNS isolates having inducible clindamycin resistance (N=22)

Antibiotics tested		MRCoNS, N = 12 (100%)	MSCoNS, N = 10 (100%)	Chi-Square (χ^2) & *p value
Penicillin	Resistant	12 (100%)	7 (70.0%)	$\chi^2 = 4.168$, p = 0.041
	Sensitive	0 (0%)	3 (30.0%)	
Gentamicin	Resistant	3 (25.0%)	2 (20.0%)	$\chi^2 = 0.078$, p = 0.781
	Sensitive	9 (75.0%)	8 (80.0%)	
Amikacin	Resistant	2 (16.7%)	1 (10.0%)	$\chi^2 = 0.206$, p = 0.650
	Sensitive	10 (83.3%)	9 (90.0%)	
Doxycycline	Resistant	2 (16.7%)	2 (20.0%)	$\chi^2 = 0.041$, p = 0.840
	Sensitive	10 (83.3%)	8 (80.0%)	
Trimethoprim-sulfamethoxazole	Resistant	10 (83.3%)	4 (40.0%)	$\chi^2 = 4.426$, p = 0.035
	Sensitive	2 (16.7)%	6 (60.0%)	
Cefoxitin	Resistant	12 (100%)	0 (0%)	$\chi^2 = 22.000$, p < 0.001
	Sensitive	0 (0%)	10 (100%)	
Ciprofloxacin	Resistant	11 (91.7%)	3 (30.0%)	$\chi^2 = 8.964$, p = 0.003
	Sensitive	1 (8.3)%	7 (70.0%)	
Chloramphenicol	Resistant	5 (41.7%)	2 (20.0%)	$\chi^2 = 1.180$, p = 0.277
	Sensitive	7 (58.3%)	8 (80.0%)	
Vancomycin	Resistant	0 (0%)	0 (0%)	NA
	Sensitive	12 (100%)	10 (100%)	
Linezolid	Resistant	0 (0%)	0 (0%)	NA
	Sensitive	12 (100%)	10 (100%)	
Teicoplanin	Resistant	0 (0%)	0 (0%)	NA
	Sensitive	12 (100%)	10 (100%)	

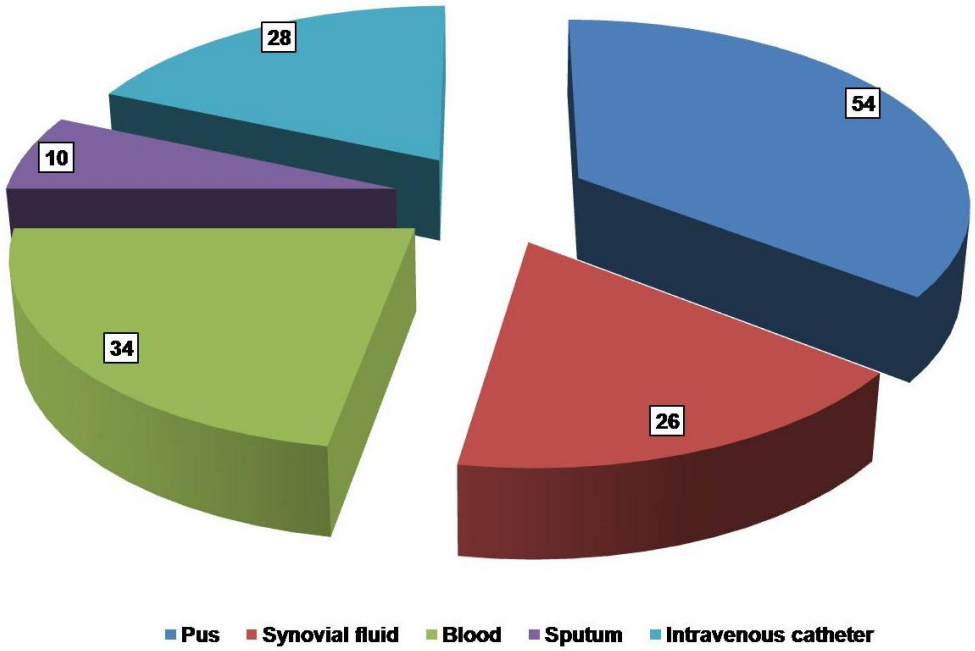
N = Number of CoNS isolates with inducible clindamycin resistance; MRCoNS = Methicillin resistant CoNS; MSCoNS = Methicillin sensitive CoNS; NA = Not Applicable. *p value < 0.05 was considered as statistically significant.

Fig.1 Isolate with inducible clindamycin resistance or positive D-zone test with blunting (D-shaped) of clindamycin zone of inhibition proximal to the erythromycin disk

Fig.2 Isolate with constitutive resistance to both erythromycin and clindamycin disks

Fig.3 MS phenotype or negative D-zone test with resistance to erythromycin and sensitivity to clindamycin (circular zone of inhibition or no induction)

Fig.4 Distribution of CoNS isolates among clinical samples tested



However, resistance can develop in staphylococcal isolates with inducible phenotypes due to selection of constitutively resistant mutants both *in vitro* and *in vivo* during clindamycin therapy (Rao, 2000). Therefore, such isolates should be identified prior to subscription of clindamycin. This is where the D-test becomes significant. On putting D-zone test we found that amongst 59 MRCoNS, $cMLS_B$ phenotype predominated (49.2%), followed by $iMLS_B$ (20.3%) and MS phenotypes (18.6%), whereas, amongst 93 MSCoNS, MS phenotype predominated (33.3%), followed by $cMLS_B$ phenotypes (11.8%) and $iMLS_B$ phenotype (10.8%). Another study reported that among 89 MRCoNS isolates, $cMLS_B$ resistance predominated (51.7%), followed by $iMLS_B$ resistance and the MS phenotype (25.8% and 12.4% respectively), whereas, among 161 MSCoNS isolates, MS phenotype was predominant (27.3%), followed by $iMLS_B$ (13.7%) and $cMLS_B$ (11.8%) resistance respectively (Bansal *et al.,* 2012). This finding was in partial agreement to our report. A study from Rajasthan reported that among 153 MRCoNS, $iMLS_B$ phenotype predominated (21.56%), followed by MS (19.60%) and $cMLS_B$ phenotypes (13.07%) (Supriyarajvi *et al.,* 2015). This finding was in contrast to our study. However, their findings of MSCoNS were in agreement to that of our study as they reported that among 133 MSCoNS, MS phenotype predominated (26.31%), followed by $cMLS_B$ (15.03%) and $iMLS_B$ phenotype (11.21%). Another contrasting finding was reported by a study from Telangana which showed that among 50 MRCoNS isolates, MS phenotype predominated (34.0%), followed by $cMLS_B$ (22.0%) and $iMLS_B$ phenotype (18.0%) (Zachariah *et al.,* 2016).

In our study the overall prevalence of inducible clindamycin resistance among CoNS isolates was 14.5%. A study from Rajasthan reported prevalence of $iMLS_B$ resistance phenotypes amongst CoNS isolates to be 16.8% (48/286 isolates) (Supriyarajvi *et al.,* 2015). Study from Uttarakhand reported prevalence of $iMLS_B$ resistance among CoNS isolates to be 19.4% (26/134 isolates) (Juyal *et al.,* 2013). These variations in the prevalence of inducible

clindamycin resistance in different geographical areas could be due to differences in bacterial susceptibility and varying antimicrobial subscribing patterns of physicians (Bansal *et al.*, 2012).

In our study, we found higher prevalence of inducible clindamycin resistance ($iMLS_B$ phenotype) among community acquired strains (68.2%) as compared to hospital acquired strains (31.8%). Probable reason behind this finding could be that clindamycin being an oral drug is frequently prescribed by physicians in outdoor clinical settings leading to increased incidence of $iMLS_B$ resistance phenotype among community acquired strains of CoNS.

In the present study we found that all the CoNS isolates with inducible clindamycin resistance were 100% sensitive to vancomycin, linezolid and teicoplanin, followed by sensitivity to amikacin (86.4%), doxycycline (81.8%), gentamicin (77.3%) and chloramphenicol (68.2%), with least sensitivity to penicillin (13.6%). Similarly a study from Jaipur also reported that isolates with $iMLS_B$ phenotype were 100% sensitive to vancomycin and linezolid, followed by sensitivity to ciprofloxacin (78.78%), doxycycline and piperacillin / tazobactam (69.56% each) (Pal *et al.*, 2010).

Clindamycin has long been an attractive option in the treatment of skin and soft tissue infections as well as serious infections because of its efficacy against both methicillin sensitive and resistant *Staphylococci*. However, treatment failure may result when clindamycin is used for therapy of staphylococcal strains that exhibit inducible resistance. Therefore, it is important for all laboratories to be aware of the local prevalence of inducible clindamycin resistant *Staphylococci* as the incidence is highly variable with regard to geographical area. The D-zone test is an easy, sensitive, and reliable means for detection of such strains in clinical laboratory settings and should be performed routinely for proper guidance of clinicians in prescription of clindamycin for treatment of patients with true clindamycin susceptible strains and its avoidance when inducible resistance is detected thus preventing treatment failure.

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