

Antibiotic susceptibility of *Escherichia coli* isolates from inpatients with urinary tract infections in hospitals in Addis Ababa and Stockholm

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A high level of antimicrobial resistance of bacteria has been detected at the Tikur Anbessa Hospital (TAH), Addis Ababa, for many years. In contrast, at the Karolinska Hospital (KH), Stockholm, the level of resistance is low. Reported are the results of an investigation of the correlation between antibiotic usage and the antimicrobial resistance rates of Escherichia coli isolates from patients with urinary tract infections in these hospitals.

At TAH the strains of E. coli isolated were considerably more resistant to all seven antibiotics tested. The level of multiresistance was 63% at TAH and 7% at KH. There were no significant differences in the total amount of antibiotics used in the two hospitals, except for antituberculosis agents. The strain biotypes and antibiograms, together with the length of patients' hospitalization before a positive urine culture was obtained, suggest that the majority of the strains from TAH were of nosocomial origin.

Introduction

The antimicrobial resistance of bacteria is a problem of global concern (1). In 1983 the Fogarty International Center of the National Institutes of Health initiated a project to map the antimicrobial resistance patterns and the use of antibiotics worldwide, the results of which were published in 1987 (2). It was concluded that, although there is a correlation between antibiotic use and subsequent resistance, the level or pattern of usage or other factors that contribute to different levels of resistance are not known (3). Resistance is more prevalent in developing countries (4), but more data on the use of antimicrobial agents in such countries are required.

There have been several reports on the high level of antimicrobial resistance among isolates of

Escherichia coli from the Tikur Anbessa Hospital (TAH), Addis Ababa (5-8), and calls for a review of the antibacterial policy in the hospital have been made. There has, however, been no analysis of the kinds and quantities of antibiotics used. In contrast, at the Karolinska Hospital (KH), Stockholm, the level of resistance of *E. coli* isolates is low. The aim of the present study was to investigate the correlation between antibiotic use and resistance by comparing practices in the two hospitals. Establishment of a baseline in this way could make it possible to monitor the effects on resistance rates of guidelines on antibiotics policy and other measures. We therefore investigated the use of antibiotics in the two hospitals and the resistance pattern of *E. coli* isolates from patients with urinary tract infections over the same period. Our results are reported here.

Materials and methods

Clinical isolates of *E. coli*

Urine specimens from hospitalized adults were plated using a semiquantitative technique. Consecutive isolates of *E. coli* from specimens that contained $\geq 10^5$ colony-forming units (CFU) per ml were used in the study, and the strains were identified using the API 20E system.^a The study started simultaneously in April 1986 at TAH, Addis Ababa (62 strains collected over a 1-year period), and KH, Stockholm (61 strains collected in April and November 1986). Infor-

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^a API System, F-38390 Montalieu-Vercieu, France

Table 1. Minimum inhibitory concentration (MIC) limits and the corresponding zone-diameter interpretive breakpoints used in the study

Antibiotic	Disk content (μg)	MIC ($\mu\text{g/ml}$) ^a		Zone-diameter breakpoint (mm) ^a	
		R \geq	S \leq	R \leq	S \geq
Ampicillin	10	32	8	11	14
Cefalotin	30	32	8	14	18
Tetracycline	30	16	4	14	19
Chloramphenicol	30	25	12	12	18
Trimethoprim	5	16	4	10	16
Sulfonamides	250/300	350	100	12	17
Trimethoprim/sulfamethoxazole	1 25/23 75	8/152	2/38	10	16
Gentamicin	10	8	4	12	15
Tobramycin	10	8	4	12	15
Netilmicin	30	32	12	12	15
Amikacin	30	32	16	14	17
Kanamycin	30	25	6	13	18
Streptomycin	10	-	-	11	15
Nitrofurantoin	300	100	25	14	17
Nalidixic acid	30	32	8	13	19

^a R = resistant, S = susceptible.

mation on a patient's date of admission to the hospital, the date that the specimen was collected, and the department (medical or surgical) where the patient was treated were available for each strain of *E. coli* identified.

Susceptibility testing

The minimum inhibitory concentration (MIC) was determined by the agar dilution method (9) on Mueller-Hinton agar^b. An inoculum of 10^6 CFU was prepared, and the plates were then inoculated using a Steers applicator that delivered approximately 10^3 CFU and then incubated at 37 °C for 20 hours. All strains from both hospitals were tested simultaneously at KH. The MIC limits for susceptibility grouping, as defined by the U.S. National Committee for Clinical Laboratory Standards (10), are shown in Table 1. The following antibiotics were tested: ampicillin, cefalotin, tetracycline, trimethoprim, sulfisomidine, and gentamicin.

The standardized disk diffusion method (11) was used at TAH. The results of routine susceptibility testing of *E. coli* isolated from 1976 to 1981 from inpatients with urinary tract infections (5) were compared with those obtained for the 61 strains isolated in the hospital in the present study. The disks and zone diameter interpretive breakpoints used to analyse the data are shown in Table 1. The disk diffusion method (12) was also used at KH to determine the antibiograms of the 61 strains for aminoglycosides. For this purpose, disks that con-

tained 30 μg of the respective aminoglycoside were used, and the following interpretive breakpoints for resistance, as defined by the Swedish Reference Group for Antibiotics, were used ≤ 16 mm (gentamicin and tobramycin); 17 mm (netilmicin), 14 mm (amikacin); 13 mm (kanamycin); and 13 mm (streptomycin) (13).

Computerized biochemical fingerprinting

The strains of *E. coli* were characterized epidemiologically using a biochemical fingerprinting technique (14). The strains were tested in 24 biochemical reactions, the results read after 4, 8, 24, and 48 hours of incubation, and the data used to carry out computerized calculations of similarities among the isolates. A correlation coefficient of 0.98 between isolates was taken to indicate that they were identical and that they had originated from the same source of infection.

Collection of information on antibiotic use

Information on antibiotic use at KH from January to June 1987 was obtained from the routine registration records kept by the hospital pharmacy for each ward, while the number of bed-days for each ward was available from hospital statistics.

At TAH, wards receive drugs from the hospital pharmacy upon receipt of daily prescriptions that are noted in a separate book for each ward. Antibiotic use in December 1986 and February and April 1987 was used in the study. The amounts of antibiotics prescribed were calculated from the daily records for each ward, and the number of bed-days

^b Difco, Detroit, MI, USA

for each ward during these same 3 months was determined from the daily registration records of the number of admissions, discharges, and deaths. From the amount of each antibiotic prescribed, the number of bed-days, and the standard defined daily doses (DDD) (15) the DDD per 100 bed-days were calculated for each antibiotic prescribed at the two hospitals.

Results

Level of antibiotic resistance of *E. coli* isolates

Table 2 shows the rate of resistance of the *E. coli* isolates to the seven antibiotics studied, as indicated by the MIC values ("R" in Table 1). The strains isolated at TAH were considerably more resistant to all the drugs than were those isolated at KH. This is illustrated in Fig. 1, which shows the ratio of the rates of resistance at the two hospitals for each drug. The difference in resistance was considerable. For example, at TAH 3.4 times as many strains were resistant to trimethoprim than at KH, and at TAH 15% of all strains were resistant to gentamicin, while no resistance to this antibiotic was encountered at KH. The level of multiresistance, i.e., the resistance to four or more of the seven drugs tested, was 63% at TAH and 7% at KH. At TAH there was no difference in resistance between strains that were isolated from patients in medical or surgical departments, while at KH the number of resistant strains was too small to allow any conclusions to be drawn.

Antibiotic resistance of *E. coli* isolated at TAH in 1976-81 and 1986-87

The level of resistance of *E. coli* isolated from inpatients with urinary tract infections was high at TAH also in 1976-81. Of the eleven antibiotics tested during both periods, there was a significant change only for trimethoprim sulfamethoxazole, for which the proportion of resistant strains increased from 7% to 29%, and for gentamicin, for which the increase was from 1% to 18% (Table 3). The increase

Table 2: Percentage of *Escherichia coli* strains isolated from inpatients with urinary tract infections at Tikur Anbessa Hospital, Addis Ababa, and Karolinska Hospital, Stockholm, that were resistant to the antibiotics tested in the study

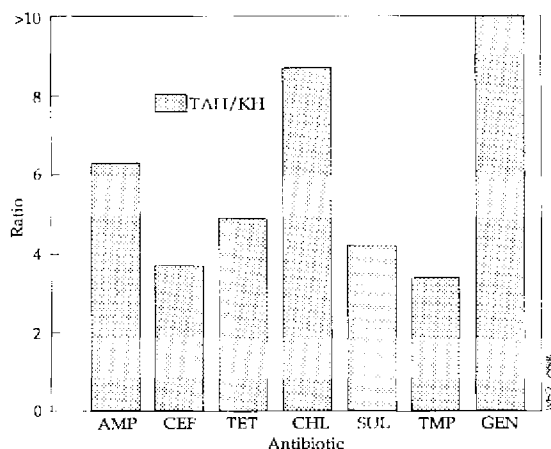
	% strains resistant to: ^a						
	AMP	CEF	TET	CHL	SUL	TMP	GEN
Tikur Anbessa ^b	81	26	79	61	76	34	15
Karolinska ^c	13	7	16	7	18	10	0

^a AMP = ampicillin; CEF = cefalotin; TET = tetracycline; CHL = chloramphenicol; SUL = sulfisomidine; TMP = trimethoprim; GEN = gentamicin.

^b 62 strains.

^c 61 strains.

Fig. 1. Ratios of the rates of resistance of *Escherichia coli* isolates from patients with urinary tract infections at Tikur Anbessa Hospital (TAH), Addis Ababa, to those at Karolinska Hospital (KH), Stockholm.



AMP = ampicillin; CEF = cefalotin; TET = tetracycline; CHL = chloramphenicol; SUL = sulfisomidine; TMP = trimethoprim; GEN = gentamicin.

Table 3: Percentage of *Escherichia coli* strains isolated from inpatients with urinary tract infections at Tikur Anbessa Hospital, Addis Ababa, in 1976-81 and 1986-87 that were resistant to the antibiotics tested

	% strains resistant to: ^a											
	AMP	CEF	TET	CHL	SUL	SXT	TMP	GEN	KAN	STR	NIT	NAL
1976-81 ^b	71	28	76	64	82	7	NT ^c	1	17	78	6	5
1986-87 ^d	82	31	81	58	71	29	34	18	21	73	2	2

^a SUL = sulfadiazine; SXT = trimethoprim-sulfamethoxazole; KAN = kanamycin; STR = streptomycin; NIT = nitrofurantoin; NAL = nalidixic acid. See footnote a, Table 2, for other abbreviations.

^b 331 strains.

^c NT = not tested.

^d 62 strains.

in resistance to trimethoprim-sulfamethoxazole resulted from the increase in that to trimethoprim in strains that were already sulfonamide resistant. Only one strain was resistant to trimethoprim without also being resistant to sulfonamides.

Epidemiological considerations

Antibiograms. At TAH, *E. coli* strains with multiple resistance had a limited number of different antibiograms, which indicates that many of these strains may have been of nosocomial origin. The nine gentamicin-resistant strains exhibited the greatest resistance. For example, three of the strains were resistant to all seven antibiotics tested, and the remaining six were resistant to all but cefalotin. The results of the aminoglycoside antibiograms (Table 4) indicate that the gentamicin-resistant strains were also resistant to tobramycin and netilmicin, but there was no cross-resistance with streptomycin or kanamycin.

Duration of hospital stay before urine cultures were positive. At TAH, 3 of the 62 patients had positive urinary cultures before the third day of hospitalization and 10 patients before the tenth day, while at KH 25 of the 61 patients had positive cultures before the third day and 41 before the tenth day. If the diagnosis of a nosocomial urinary tract infection is based on a positive urinary culture after the third day of hospitalization (16), 95% of such infections at TAH and 59% at KH were nosocomial.

Biotyping. At TAH the results of the computerized biochemical fingerprinting showed that the most common phenotype was represented by 23% of the isolates, while 33% of the isolates were single phenotypes. At KH the most common phenotype was represented by 8% of the isolates, and 71% were single phenotypes. The greater degree of homogeneity and the lower number of phenotypes at TAH than at KH indicates that a greater proportion of the strains from TAH were derived from a common source of infection.

Use of antibiotics in the two hospitals

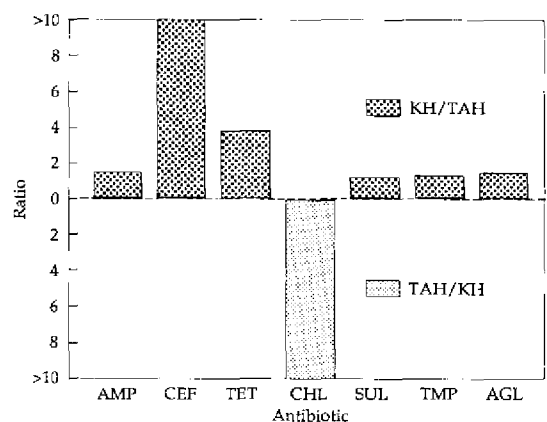
The use of antibiotics in the two hospitals, expressed as the DDD per 100 bed-days (15), is shown in Table 5. The total amount of antibiotics used in 1986-87 was higher at TAH, but, if the amount used for treatment of tuberculosis only is omitted there was no significant difference (treatment for tuberculosis is not carried out in medical or surgical departments at KH). Chloramphenicol, penicillin, streptomycin, and antituberculosis agents were used more often at TAH, but the use of all other antibiotics was greater at KH. Approximately the same amount of antibiotics was used in medical and surgical departments at KH (Table 5), but at TAH three times the amount of antibiotics were used in medical than in the surgical department. This difference arose mainly because of the use of antituberculosis agents and because of the greater use of penicillin and aminoglycosides in the medical departments. The pattern of usage in the two hospitals differs in that at KH more use is made of β -lactamase-stable penicillins and cephalosporins, while little use is made of chloramphenicol. Use of

Table 4: Phenotypes of aminoglycoside resistance in 62 strains of *Escherichia coli* isolated from inpatients with urinary tract infections, Tikur Anbessa Hospital, Addis Ababa, 1986-87

Resistance phenotypes ^a	No. of strains
—	22
Str	23
Kan	2
Str, Kan	6
Kan, Gen, Tob, Net	2
Str, Gen, Tob, Net	3
Str, Kan, Gen, Tob, Net	4
Total	62

^a Str = streptomycin; Kan = kanamycin; Gen = gentamicin; Tob = tobramycin; Net = netilmicin. No strains were resistant to amikacin.

Fig. 2. Ratios of the level of consumption of seven antibiotics (calculated as defined daily doses (DDD) per 100 bed-days) at Tikur Anbessa Hospital (TAH) and Karolinska Hospital (KH).



Dark bars: higher consumption at KH; light bars: higher consumption at TAH. AMP = ampicillin; CEF = cefalotin; TET = tetracycline; CHL = chloramphenicol; SUL = sulfisomidine; TMP = trimethoprim; AGL = aminoglycosides, excluding streptomycin.

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Table 5: Use of antibiotics in medical and surgical departments at Tikur Anbessa Hospital (TAH) and Karolinska Hospital (KH) 1986-87, expressed as standard defined daily doses (DDD) per 100 bed-days

Antibiotic	DDD per 100 bed-days:					
	TAH			KH		
	Surgical	Medical	Both	Surgical	Medical	Both
Penicillin (G, V)	16.2	41.0	24.5	7.7	12.2	10.5
Isoxazolylpenicillins	0.1	1.7	0.7	5.7	6.0	5.9
Ampicillin	5.5	12.4	7.9	18.4	7.7	11.7
Cephalosporins	0 ^a	0	0	1.8	1.9	1.9
Tetracyclines	0.2	3.0	0.5	0.2	3.0	1.9
Chloramphenicol	2.0	3.7	1.9	0	0.02	0.01
Erythromycin	0.1	1.7	0.7	0.8	3.1	2.2
Clindamycin, vancomycin	0	0	0	0.1	1.0	0.6
Fusidic acid	0	0	0	0	0.1	0.1
Aminoglycosides ^b	1.1	15.0	2.6	2.1	3.5	3.7
Streptomycin	2.9	5.4	7.2	0	0	0
Sulfamethoxazole ^c	3.3	3.3	3.3	1.8	5.2	3.9
Trimethoprim ^d	3.3	3.3	3.3	1.8	5.5	4.1
Urinary tract infection agents ^e	1.1	0.6	0.9	0.1	0.1	0.1
Metronidazole	1.2	2.1	1.9	5.8	0.6	2.6
Antituberculosis agents ^f	4.3	40.3	17.0	0	0	0
Total	41.3	134.9	71.5	46.3	49.9	49.2

^a 0 = no use registered in 1986-87.

^b Does not include streptomycin.

^c In combination with trimethoprim.

^d Alone or in combination with sulfamethoxazole.

^e Nitrofurantoin, nalidixic acid, norfloxacin.

^f Isoniazid, ethambutol, rifampicin.

streptomycin is accounted for separately from other aminoglycosides, since it is employed both for treatment of tuberculosis and as a general antibiotic at TAH and is not used at KH.

Comparison between the use of antibiotics and the resistance rates in the two hospitals

Strains of *E. coli* isolated from TAH were considerably more resistant to the seven antibiotics tested than those from KH (Fig. 1). The level of use of these drugs in the two hospitals is compared in Fig. 2. The expected positive correlation between the level of antibiotic use and *E. coli* resistance rates was not found. In contrast, there was greater resistance against all the antibiotics at TAH, where the level of use of these drugs is lower, except for chloramphenicol.

Discussion

Comparison of the prevalence of antimicrobial resistance in countries from different regions of the world presents some difficulties. First, there is sample

bias in selecting the type and severity of infections to be studied. The species of bacteria that are the dominant cause of infection may vary, and the species may acquire resistance to different antimicrobials with greater or less ease. Also, if the prevalence of resistance for a single species of bacteria is compared, the identification criteria for that species may vary from study to study. Even more difficult to control is the significance of different methods of testing susceptibility and the frequent lack of documentation on quality control. Finally, there is no universally accepted standard for defining susceptibility categories. Despite these difficulties, antimicrobial resistance has been reported to be more prevalent in developing than in developed countries (4).

In the present study we have attempted to overcome some of the biases by examining urinary tract infections only and by comparing only the susceptibility of *E. coli*, the dominant causative agent of such infections in both hospitals. The identification of the strains involved and the susceptibility testing were carried out simultaneously in one laboratory (at KH) using the same criteria for both sets of strains. The

strains isolated at TAH were considerably more resistant to all seven antibiotics tested than were those isolated at KH.

A high level of antibiotic resistance has been detected at TAH for many years. Comparison of the results of routine susceptibility testing of *E. coli* strains isolated in 1986–87 from inpatients treated for urinary tract infections at TAH with those treated in 1976–81 (5) indicates that there was an overall high level of resistance during both periods. The only significant increase in resistance was towards trimethoprim–sulfamethoxazole and gentamicin. Our analysis shows that the increase in resistance to trimethoprim–sulfamethoxazole was caused by the increase in that to trimethoprim alone. This is not surprising, considering the high level of resistance to sulfonamides that prevailed when the drug combination was first introduced. The presence of the sulfonamide has therefore not prevented development of resistance to trimethoprim, which was not used on its own in TAH. The increase in resistance to trimethoprim is unfortunate, since this relatively recently introduced drug is an inexpensive oral antibiotic that could have been useful in the control of infectious diseases.

The increase in the resistance to gentamicin at TAH is also alarming. Since 1976–81, during the years shortly after the drug was introduced, resistance to it has increased from 1% to 18%. This is particularly serious, since in life-threatening bacterial infections gentamicin is often the drug of choice. Along with resistance to gentamicin, the *E. coli* strains were also resistant to tobramycin and netilmicin, either through a common mechanism or because they have acquired resistance markers to more than one aminoglycoside-degrading enzyme.

The following important questions arise:

- Why is there such a great difference in the level of resistance between the two hospitals?
- What can be done to regain the usefulness of some of the antibiotics?
- What can be learned from our findings to help prevent a similar development when new antibiotics are introduced?

The use of antibiotics provides an environment for the selection of resistant bacteria. The more antibiotics that are used, the more likely it is that resistant strains will be selected and maintained in the environment; however, the relationship between resistance rates and usage of such drugs is complicated. The use of any one antibiotic can contribute to resistance to another if there is a common cross-resistance mechanism or if the resistance genes are linked on the same transferable genetic element. The direct relationship between the use of an antibiotic and resistance to it may be difficult to establish since

the resistance may reflect not only those antibiotics that are being currently employed but also those that are no longer in common use.

There was a large difference in the resistance rates at the two hospitals, but this cannot be explained by the difference in the amounts of antibiotics used in the hospitals at present. If the anti-tuberculosis drugs isoniazid, ethambutol, and rifampicin are disregarded (they probably do not contribute to the selective pressure on *E. coli* because of their particular antibacterial spectrum), the total amount of antibiotics used was 54.5 DDD per 100 bed-days at TAH compared with 49.2 at KH. Also, if the aminoglycoside antibiotics are examined separately, 3.7 DDD per 100 bed-days were used at KH, with no resistant strains, and 2.6 DDD per 100 bed-days at TAH, with 15% of the strains being resistant to these drugs (streptomycin was not included in the analysis since there was no cross-resistance between it and the other aminoglycosides tested). However, there are differences in the way antibiotics are used in the two hospitals. At KH use of antibiotic prophylaxis is restricted to certain types of surgery, and then only for a short perioperative period; in contrast, at TAH about 60% of surgical and gynaecological patients received such prophylaxis, usually with more than one antibiotic and for a period of a week or longer (6–8). At KH the antibiotic selected for individual patients is usually determined from the results of susceptibility tests on the infecting micro-organism. If empirical therapy is started before the results of such tests are available, the medication given is corrected within 1–2 days, if needed. Furthermore, there are good epidemiological data available on the level of resistance in the hospital to guide clinicians in the choice of empirical therapy. In contrast, at TAH the laboratory facilities are less extensive than at KH, and epidemiological data, when available, provide little guidance in selecting an antibiotic because of the high level of multiresistant bacteria.

Nosocomial infections influence the level of antibiotic resistance since resistant bacteria are likely to be selected in the hospital environment. A difference in nosocomial strains could account for some of the variation in the rates of resistance between TAH and KH, for the following reasons. First, the large proportion of strains at TAH that exhibited multiple resistance produced a small number of different antibiograms, which is consistent with many of the strains being of nosocomial origin. Second, if the definition of nosocomial urinary tract infection is based on the length of time patients spent in hospital before a positive urine culture was made (16), there was a substantial difference between the hospitals in that the majority of such infections at TAH were of

nosocomial origin. Third, determination of the strain biotypes indicates that there was a higher level of homogeneity and lower number of phenotypes at TAH than at KH, which suggests that nosocomial strains were more frequent at TAH. The majority of *E. coli* strains isolated at TAH were therefore from infections of nosocomial origin, which accounts for the observed high levels of resistance.

Measures can, however, be taken to improve the situation at TAH. For example, the facilities at the bacteriological laboratory could be strengthened. It is, however, not easy to gain support for such investments in a hospital with limited resources, but such a measure would be economically favourable because of the reduction it would bring in the use of ineffective antibiotics, together with the higher cure rates leading to shorter periods of hospitalization. Improved laboratory facilities in TAH are also required as a back-up for a much needed infection control programme to reduce the rate of nosocomial infections.

Only a few compounds exhibit antibiotic activity and it is therefore important that their efficacy be extended for as long as possible. The lower bacterial resistance to such drugs in many developed countries could be due to the existence there of infection control programmes and the extensive use of the services of bacteriological laboratories, which permit microorganisms to be treated according to their characterized susceptibility.

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Résumé

Sensibilité d'*Escherichia coli* aux antibiotiques chez des sujets hospitalisés atteints d'infections urinaires à Addis-Abeba et Stockholm

La résistance des bactéries aux antibiotiques est un problème mondial, plus particulièrement répandu dans les pays en développement. Plusieurs rapports font état d'un fort degré d'antibiorésistance à l'Hôpital Tikur Anbessa d'Addis-Abeba. Ce taux est en revanche faible à l'Hôpital Karolinska de Stockholm. On a étudié la corrélation entre l'emploi des antibiotiques et la résistance en com-

parant, dans ces hôpitaux, la résistance des souches d'*Escherichia coli* isolées chez des sujets atteints d'infections urinaires.

Les souches d'*E. coli* isolées à Addis-Abeba étaient beaucoup plus résistantes que celles de Stockholm à chacun des sept antibiotiques étudiés, la proportion de souches résistantes étant 6,2 fois plus élevée pour l'ampicilline, 3,7 fois pour la céfalotine, 4,9 fois pour la tétracycline, 8,7 fois pour le chloramphénicol, 4,2 fois pour la sulfisomidine, et 3,4 fois pour le triméthoprime. Dans cet hôpital, 15% des souches étaient résistantes à la gentamicine, alors que ce type de résistance n'existait pas à Stockholm. L'emploi d'antibiotiques, exprimé en nombre de doses standards quotidiennes pour 100 lits-jour, n'était pas sensiblement différent dans les deux hôpitaux (à l'exception des antituberculeux, qui ont été exclus de cette étude). La quantité totale d'antibiotiques utilisée était de 54,5 doses pour 100 lits-jour à Addis-Abeba et 49,2 doses à Stockholm.

Les souches d'*E. coli* isolées à Addis-Abeba ne présentaient qu'un petit nombre d'antibiogrammes différents. De même, les résultats de l'étude biochimique informatisée de ces souches montrent qu'à Addis-Abeba, il existait une plus grande homogénéité et un plus faible nombre de phénotypes. D'après ces deux observations, les souches présentes à Addis-Abeba dérivent d'une source d'infection commune. Si l'on base la définition des infections urinaires nosocomiales sur la durée du séjour à l'hôpital avant obtention d'une culture urinaire positive, la plupart des infections trouvées à Addis-Abeba entrent dans cette catégorie. L'ensemble de ces résultats semble donc indiquer que la majorité des souches d'*E. coli* trouvées à Addis-Abeba sont d'origine nosocomiale, ce qui expliquerait leur degré élevé d'antibiorésistance, car les bactéries résistantes se sélectionnent plus facilement en milieu hospitalier.

Il est recommandé de renforcer les ressources du laboratoire bactériologique de l'Hôpital Tikur Anbessa d'Addis-Abeba, d'une part pour faciliter le choix des antibiotiques convenables à administrer aux malades et d'autre part pour qu'il puisse fonctionner comme service d'appui dans le cadre d'un programme de lutte contre les infections.

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