that 26% of the enterococci in this study were highly resistant to both gentamicin and streptomycin. Aminoglycosides have no therapeutic benefit in infections involving such strains, and unnecessarily expose patients to possible ototoxic or nephrotoxic side effects. A further 28% of the isolates were highly resistant to either gentamicin or streptomycin, emphasising the value of testing both of these compounds in determining appropriate treatment.

Current guidelines recommend that endocarditis caused by enterococci with high level resistance to aminoglycosides should be treated with high dose amoxycillin or ampicillin for 6-12 weeks.[2 3] However, 11% of the enterococci were *E faecium*, which typically is resistant to ampicillin. Moreover, amoxycillin or ampicillin would be unsuitable for patients allergic to penicillin. This latter constraint applies to other proposed regimens that combine ampicillin with imipenem or ciprofloxacin. Although glycopeptides may be considered in place of penicillin, the finding of glycopeptide resistance in several isolates, including three of the *E faecium* isolates, means that their efficacy cannot be guaranteed.

The picture revealed is disturbing, with frequent resistance to the recommended synergistic combinations. Evaluation in endocarditis of unconventional regimens—for example, ampicillin plus carbapenems, ampicillin plus ciprofloxacin, or ciprofloxacin plus co-trimoxazole—is desirable, although the use of such broad spectrum agents may risk selecting resistance in the body microflora. Also desirable is early evaluation, in endocarditis, of novel narrow spectrum anti-Gram positive agents, such as streptogramins, oxazolidinones, and everninomycins.

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Multidrug resistant tuberculosis in France 1992-4: two case-control studies

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Since 1988 several outbreaks of multidrug resistant tuberculosis have occurred in the United States and Europe. We surveyed the national network of laboratories serving 80% of public hospital beds in France to measure the prevalence of multidrug resistant tuberculosis during 1992-4.[1]

Subjects, methods, and results

Annual prevalence of multidrug resistance was calculated by dividing the number of cases of multidrug resistant tuberculosis—patients who had at least one isolate resistant to isoniazid and rifampicin in the calendar year—by the total number of cases with tuberculosis confirmed by culture that the laboratories reported. Multidrug resistant tuberculosis was defined as secondary in patients who had been treated for 1 month or more before the first known multidrug resistant isolate, and as primary in all other cases. DNA fingerprinting was performed on multidrug resistant strains sampled in 1993 and 1994.[2] Factors associated with multidrug resistant tuberculosis were analysed by comparing cases of multidrug resistant tuberculosis reported by the laboratories with cases that were notified for the same period by 69 (of 100) French districts where HIV infection was consistently monitored. We compared primary and secondary cases of multidrug resistant tuberculosis in two case-control studies with all notified new cases and all notified cases with a history of previous tuberculosis respectively. We performed multivariate analysis by logistic regression.

In 1992, 48 out of 8521 cases of tuberculosis confirmed by culture were multidrug resistant (0.6% (95% confidence interval 0.4% to 0.7%)); in 1993, 40 out of 8539 (0.5% (0.3% to 0.6%)); and in 1994, 58 out of 7752 (0.7% (0.5% to 0.9%) ($P = 0.10$ for trend). Prevalence did not vary significantly between the 22 administrative regions.

The 146 cases occurred in 125 patients, of whom 116 (93%) had pulmonary tuberculosis (70 had a positive sputum smear test). Of 122 patients with information on previous treatment, 31 had primary and 91 secondary multidrug resistant tuberculosis. Overall, 91 out of 122 (74%) of the patients were men and 58 out of 122 (49%) were born outside Europe, without significant difference between primary and secondary cases. Primary cases were significantly younger than secondary cases (median age 35 years *v*

Case-control studies on primary and secondary multidrug resistant tuberculosis, France, 1992-4

Characteristic	No (%) of patients with primary multidrug resistant tuberculosis (n=31)	No (%) of new cases of tuberculosis	Adjusted odds ratio (95% CI)	No (%) of patients with secondary multidrug resistant tuberculosis	No (%) of patients with recurrent tuberculosis	Adjusted odds ratio (95% CI)
Sex:						
Female	10 (32)	4 161 (37)	Reference	21 (23)	740 (35)	Reference
Male	21 (68)	6 994 (62)	1.0 (0.5 to 2.0)	70 (77)	1382 (65)	1.5 (0.9 to 2.5)
Age group (years):						
45+	8 (26)	4 910 (44)	Reference	37 (41)	1492 (70)	Reference
0-44	23 (74)	6 235 (56)	1.5 (0.6 to 3.4)	54 (59)	627 (30)	2.2 (1.4 to 3.5)
Geographic origin:						
Europe	17 (55)	8 012 (76)	Reference	46 (51)	1688 (84)	Reference
North Africa	5 (16)	1 057 (10)	2.5 (0.9 to 6.8)	16 (18)	186 (9)	2.7 (1.5 to 4.9)
Sub-Saharan/other	9 (29)	1 468 (14)	2.4 (1.0 to 5.5)	28 (31)*	142 (7)	4.8 (2.8 to 8.2)
HIV status:						
Negative/unknown	20 (64)	10 267 (92)	Reference	79 (83)	2015 (95)	Reference
Positive	11 (36)	893 (8)	5.6 (2.6 to 12.1)†	12 (17)	107 (5)	1.6 (0.8 to 3.1)‡
Total	31 (100)	11 160 (100)		91 (100)	2122 (100)	

*Country of birth was unknown for one patient.
†When only patients with known HIV status were included (29 multidrug resistant tuberculosis, 5864 new cases), the adjusted odds ratio for HIV positivity was 3.3 (1.5 to 7.3).
‡When only patients with known HIV status were included (69 multidrug resistant tuberculosis, 868 recurrent cases), the adjusted odds ratio for HIV positivity was 1.0 (0.5 to 2.0).

40 years, P = 0.02) and were more likely to be infected with HIV (35% (11/31) *v* 13% (12/91), P < 0.01).

We analysed DNA fingerprints for 66 of the 88 patients whose cases were reported on in 1993 and 1994. Only two patients had identical fingerprints. One was a French citizen resident in New York City who tested positive for HIV and returned to France after multidrug resistant tuberculosis was diagnosed. During his stay in hospital, where he had respiratory symptoms and a positive sputum smear test, he came in contact with the other patient, who was also HIV positive and developed multidrug resistant tuberculosis 2 months later. The strain was the "W" strain first recognised in several outbreaks in New York City.[3]

The only factor associated with primary multidrug resistant tuberculosis in multivariate analysis was infection with HIV (table). Secondary multidrug resistant tuberculosis was independently associated with young age and non-European origin but not with HIV infection (table). These results were unchanged when analysis was restricted to patients with known HIV status.

Comment

Our results do not show an epidemic of multidrug resistant tuberculosis in France. However, although HIV infection was not associated with secondary multidrug resistant tuberculosis, it was an independent risk factor for primary multidrug resistant tuberculosis. The increased risk of primary multidrug resistant tuberculosis in people infected with HIV has recently been shown by the nosocomial outbreaks reported in London and Madrid.[4 5] To assess failures in tuberculosis control, the prevalence of multidrug resistance should be monitored throughout Europe.

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Contributors: VS designed the study protocol, analysed and interpreted the data, and had overall responsibility for preparation of the paper; BD coordinated the data management and participated in protocol design and data analysis; A-CdeB collected the data and participated in data analysis; SH collected the data and participated in data analysis; GT performed the DNA fingerprinting; VV coordinated the performance and interpretation of DNA fingerprinting; JG initiated and coordinated the surveillance of multidrug resistant tuberculosis, the protocol design, and the data analysis. All authors contributed to interpreting the results and to writing the paper.

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Conflict of interest: None.

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Corrections

Confidential inquiry into quality of care before admission to intensive care

An authors' error occurred in this paper by P McQuillan and colleagues (20 June, pp1853-8). Dr C H Collins, consultant anaesthetist at the Royal Devon and Exeter Hospital, should have been included as an author. Dr Collins was a member of the team that originally set up the protocol, and he helped organise the assessments.

Randomised controlled trial of laparoscopic versus open mesh repair for inguinal hernia: outcome and cost

An error occurred in the labelling of one of the figures in this paper by Wellwood and colleagues (11 July, pp 103-10). In figure 3 the blue solid line represents bilateral laparoscopic repair and the purple dotted line represents unilateral open repair.