SHORT REPORTS

Contamination of dropper bottles with tear fluid in an ophthalmic outpatient clinic

The isolation of the human immunodeficiency virus (HIV) from the tears of a patient with the acquired immune deficiency syndrome (AIDS)[1] has aroused concern about the risks of transmission of the virus during various ophthalmic procedures.[2] Current recommendations permit the use of multiple application dropper bottles in ophthalmic outpatient departments, though opened bottles should be discarded at the end of each day.[3] We performed a simple study to discover whether such bottles can become contaminated with tear fluid during normal use.

Methods and results

The study took place during a single ophthalmic outpatient clinic. Oxybuprocaine (Benoxinate) was supplied in glass multiple application dropper bottles incorporating a dropper in the screw cap. One bottle was provided for each of eight slit lamps. For tonometry we asked that a drop of oxybuprocaine should be instilled into the tear sac *after* 1% fluorescein, thus reversing the usual order of application. To eliminate bias and otherwise maintain normal practice we did not explain the purpose of the study to the medical staff until afterwards. At the end of the clinic the eight bottles were collected and examined for contamination with fluorescein by using a Perkin Elmer 3000 fluorescence spectrometer set for an excitation frequency of 440 nm and an emission frequency of 510 nm. The instrument was calibrated by using serial dilutions of 1% fluorescein. The volume of drops remaining in each bottle was also measured as a rough guide to the number of times each bottle had been used.

The table shows that six of the eight bottles were contaminated with fluorescein. The bottles that were used more frequently tended to have heavier contamination.

Contamination of dropper bottles with fluorescein

Bottle No	Volume (ml) (to nearest 0·25 ml)	Contamination (nl equivalent of 1% fluorescein)
Control	4·0	0
1	3·75	0
2	3·5	1
3*	2·75	10
4	3·0	5
5	3·25	6
6*	2·5	38
7	3·0	4
8	3·75	0

* Fluorescein contamination of these bottles was detectable with the naked eye.

Comment

Our study shows that dropper bottles can be contaminated with the content of tear fluid, in contradistinction to other sources, within a single ophthalmic outpatient clinic. There are various ways in which this could occur. If the drop is touched rather than dropped on to the conjunctiva there may be reflux into the dropper. This is particularly likely if the drop is applied while the patient's chin is resting on the slit lamp. If the dropper is held too low and the patient's head is not sufficiently far back the tip of the dropper may brush against the lashes of the upper lid.

The practical importance of this result relates to patients without obvious external eye disease who none the less have microbes in their tears. Commensal bacteria are present in the conjunctival sac, and bacterial contamination of dropper bottles may occur.[4] For this reason preservatives are used. Contaminated ophthalmic solutions have also been responsible for the spread of viral disease, as in an outbreak of infection with adenovirus type 8 in Alabama.[5]

HIV has been isolated from the tears of a patient with AIDS,[1] but the extent to which it is present and active in the tears of the much larger group of asymptomatic carriers is not known. We know of no evidence to suggest that HIV has been transmitted through contact with tears. If a dropper bottle did become contaminated and if virus was then instilled into the conjunctival sac of a patient it is still unlikely that infection would occur. An analogy would be contact with saliva, which also contains HIV. Studies of exposed hospital staff, family members, and sexual partners of infected individuals have failed to show transmission of the virus by any medium other than blood and semen.

Thus contamination of multiple application dropper bottles with tear fluid may occur even when the current recommendations are complied with. Greater care or the use of single application packs would eliminate the small but finite risk that such contamination represents.

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Limitations of direct ophthalmoscopy in screening for glaucoma

Primary open angle glaucoma is one of the most common causes of visual loss encountered in clinical practice. Some form of screening is needed so that ophthalmologists can see and treat patients before they develop symptoms and reach an advanced stage of the disease. At present, most screening is performed by opticians, who examine fundi and occasionally assess intraocular pressures and visual fields of patients. Theoretically, general practitioners are better placed to identify patients at risk of developing glaucoma; however, they rely solely on direct ophthalmoscopy in screening for glaucoma.[1] Stereophotographs of optic discs may be used to predict glaucomatous defects in the visual field, with a specificity and sensitivity of over 90%.[2] To find out whether direct ophthalmoscopy can match this we conducted a survey at a recent North of England Ophthalmological meeting in which we checked the ability of ophthalmologists to screen for glaucoma by direct ophthalmoscopy alone.

Method

Thirty four ophthalmologists participated in the study, 20 of whom were consultants. Twenty two patients aged between 32 and 75 were examined. One patient had a unilateral vitreous haemorrhage so there were only 43 discs to assess. Sixteen discs were in eyes which had definite early or moderate glaucomatous defects in the visual field. The other 27 discs were in eyes which did not have any demonstrable glaucomatous defect on Goldman perimetry. This group consisted of eyes of normal subjects or patients with ocular hypertension and the normal eyes of patients with unilateral glaucoma.

Patients' eyes were dilated with tropicamide 0·5% before examination in a darkened room. Doctors' assessments were independent of one another, each doctor filling in an answer sheet by ticking "yes", "no", or "unsure" for each disc. The scores for unsure were not included in either the false positive or the false negative percentages or the specificity or sensitivity percentages.

The junior doctors diagnosed more discs as glaucomatous (table), indicating not only a better detection rate for glaucoma but also a higher false positive score. In both groups the sensitivity and, to a less degree, the specificity were less than that reported with stereophotographs. In addition, there was considerable interobserver disagreement on the glaucomatous discs.

Comment

Direct ophthalmoscopy of the optic disc entails assessing a monocular view of a sometimes highly mobile target. This study has shown that, when

Results of study

	Consultants (n=20)	Junior doctors (n=16)
Glaucomatous discs (n=16)		
Doctors' mean scores (%)		
Correct (ie, sensitivity)	7·0 (44)	8·5 (53)
Unsure	2·4 (15)	1·5 (9)
Incorrect (false negative)	6·6 (41)	6·0 (38)
Scores on individual discs (no of discs)		
75-100% answers correct	2	4
50-74% answers correct	4	5
25-49% answers correct	5	3
0-24% answers correct	5	4
Non-glaucomatous discs (n=27)		
Doctors' mean scores (%)		
Correct (ie, specificity)	20·7 (77)	18·6 (69)
Unsure	2·5 (9)	3·4 (13)
Incorrect (ie, false positive)	3·8 (14)	5·0 (18)
Scores on individual discs (no of discs)		
75-100% answers correct	16	13
50-74% answers correct	8	7
25-49% answers correct	3	3
0-24% answers correct	0	4

used in isolation, direct ophthalmoscopy may be quite inaccurate. If ophthalmologists can miss 40% of cases of early to moderate glaucoma when examining patients under ideal circumstances it is likely that general practitioners miss many more cases, especially if they examine fundi without dilating the pupils. Not surprisingly, therefore, recent surveys of the referral source of patients with glaucoma have shown that nearly all early and moderate cases are referred by opticians, while general practitioners refer mainly advanced, symptomatic cases.[3,4] General practitioners do need further training in the use of the ophthalmoscope, but these findings suggest that when they identify patients at risk of developing glaucoma, as well as performing funduscopy themselves, they should also encourage these patients to be seen regularly by their optician.[5] Finally, ophthalmologists themselves should be aware of the potential inadequacy of direct ophthalmoscopy when examining patients with suspected or definite glaucoma.

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Changes in serum lipid concentrations during first 24 hours after myocardial infarction

The serum total cholesterol concentration falls after myocardial infarction, reaching its lowest value between the sixth and ninth days.[1,2] Two reports suggested, however, that total cholesterol concentrations measured in the first 24-48 hours after myocardial infarction are probably close to pre-infarction values.[2,3] Both studies assumed that the concentrations measured months before or after myocardial infarction accurately reflect the concentrations at infarction, but this may not be valid, particularly with the increasing use of dietary intervention in patients with symptoms of heart disease.

Although total cholesterol concentration cannot be measured in the hours before myocardial infarction, documentation of any changes in the early hours after infarction is likely to enable a more accurate estimate of pre-infarction concentrations to be made. We present data on changes in serum lipid concentrations during the first 24 hours after myocardial infarction in patients admitted to hospital within four hours after the onset of chest pain.

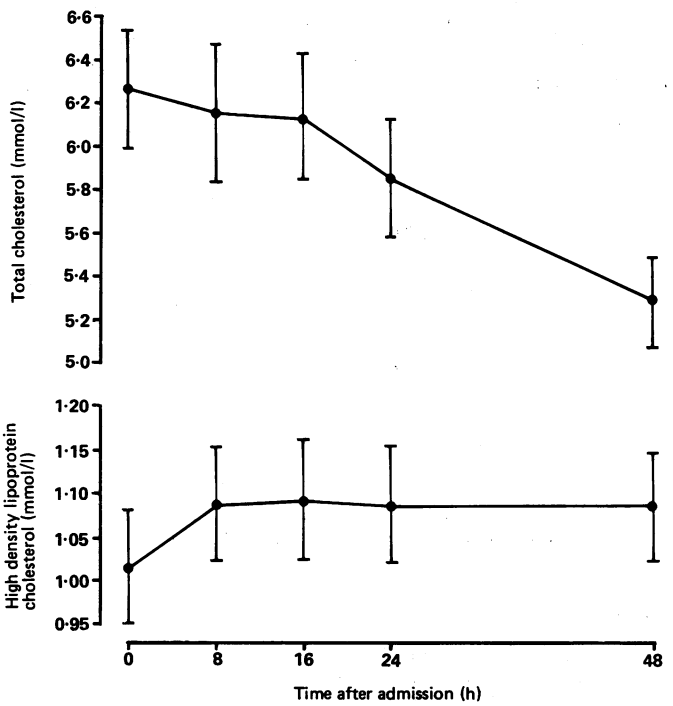
Patients, methods, and results

Non-fasting total serum cholesterol and high density lipoprotein cholesterol concentrations were measured immediately after admission to hospital and at eight, 16, 24, and 48 hours after admission in 25 patients with myocardial infarction admitted to Auckland coronary care units during 1985 within four hours after the onset of chest pain. The patients comprised 22 men and three women aged 35-69 (mean age 57·8 years). The mean time of the first sample was 2 hours 58 minutes after the onset of chest pain (range 1 hour 35 minutes to 3 hours 55 minutes).

Lipid estimations were done in a laboratory standardised according to the cholesterol standardisation programme at the Centers for Disease Control, Atlanta, Georgia. The between batch coefficient of variation for total cholesterol was 0·4% at 6·35 mmol/l; for high density lipoprotein cholesterol it was approximately 6% at 1·00 mmol/l. Paired *t* tests were used to assess the significance of differences between lipid concentrations. The level of significance was set at 5% (two sided). The study was designed to detect a 10% change in the concentrations of total cholesterol and high density lipoprotein cholesterol from 6·2 and 1·0 mmol/l, respectively, with a type II error of 0·05.

Eight hours after admission the total cholesterol concentration had fallen by 1·8% from the value on admission; at 16 hours it had fallen by 2·0%, at 24 hours by 6·6%, and at 48 hours by 15·8% (figure). There were no significant differences between the concentrations on admission and those at eight and 16 hours. At 24 hours the difference was significant (p=0·004) but remained small (0·4 mmol/l).

A simple linear regression model was fitted to the first four data points for each patient with time 0 corresponding to the onset of chest pain. A test for the homogeneity of the slopes between patients showed no significant differences, and the estimated common slope of the fitted model was 0·0166 mmol/l/h.



Trends in concentrations of total cholesterol and high density lipoprotein cholesterol after myocardial infarction. Values are means (SD).

Eight hours after admission there was a significant increase (p=0·025) in the concentration of high density lipoprotein cholesterol of 6·9% (figure). The subsequent measurements at 16, 24, and 48 hours showed almost no change from this value.

Comment

Our study confirms previous reports that the serum total cholesterol concentration in the first 24 hours after infarction is likely to reflect preinfarction concentrations. The relative change in the high density lipoprotein cholesterol concentration after myocardial infarction was greater than that of the total cholesterol concentration during this period, but this is probably of little clinical importance. As most patients with myocardial infarction are admitted within 24 hours after the onset of chest pain, and many patients are likely to miss screening scheduled for after their