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Incidence of acute symptomatic toxoplasma retinochoroiditis in south London according to country of birth

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Abstract

Objective—To determine the incidence of acute symptomatic toxoplasma retinochoroiditis presenting to ophthalmologists for patients born in Britain and elsewhere.

Design—Population based, cross sectional study.
Setting—11 districts in south Greater London.

Subjects—All patients presenting to NHS ophthalmologists with symptoms due to acute toxoplasma retinochoroiditis in 1992-3.

Main outcome measure—Intraocular inflammation in association with a retinochoroidal scar, active adjoining retinitis, and IgG serum antibodies to toxoplasma.

Results—The estimated incidence of acute symptomatic retinochoroiditis for all people born in Britain was 0.4/100 000/year and for black people born in west Africa 57/100 000/year. If a mean of two symptomatic episodes per lifetime is assumed, 100 people born in Britain may be affected each year, about a fifth of the estimated 500-600 congenitally infected people born each year.

Conclusions—A substantial proportion of people with acute symptomatic toxoplasma retinochoroiditis were born outside the country, and the number born in Britain was smaller than the number previously estimated to develop retinochoroidal lesions due to congenital toxoplasmosis. These findings suggest that prenatal screening for toxoplasmosis in Britain may be of limited benefit.

Introduction

Evidence from clinic based follow up studies suggests that retinochoroidal lesions occur in over 80% of people with congenital toxoplasmosis.¹⁻³ The proportion of affected people who develop ocular symptoms in the long term, however, is not known. Retinochoroidal lesions may be asymptomatic for long periods of time until reactivation of latent toxoplasma cysts in the retina leads to symptoms of acute retinochoroiditis, which usually requires urgent ophthalmological attention. Acute toxoplasma retinochoroiditis is diagnosed clinically by a characteristic appearance.^{4,5} In some people retinochoroidal lesions may be present from infancy and, if the macula is affected, can lead to permanent visual impairment in early childhood, whereas symptoms of acute retinochoroiditis may not

occur until lesions reactivate in the second, third, or four decades.⁶

Toxoplasma retinochoroiditis is generally attributed to congenital infection but cannot be distinguished from lesions due to acquired infection unless there is clinical or serological evidence of congenital or recently acquired infection.^{3,6-8} If we assume that people with early macular lesions eventually experience symptoms due to reactivation of retinochoroidal lesions, the incidence of acute symptomatic retinochoroiditis should represent an upper limit for the birth prevalence of congenital toxoplasma infection resulting in symptoms due to toxoplasma retinochoroiditis in the long term. A recent report from a Royal College of Obstetricians and Gynaecologists' working party on prenatal screening for toxoplasma infection recommended that more information on the prevalence of long term effects in those congenitally infected was required to determine the balance of potential risks and benefits of screening.⁹

We established a reporting system to identify all cases of acute symptomatic toxoplasma retinochoroiditis in south London. The incidence of acute toxoplasma retinochoroiditis was calculated according to country of birth using the 1991 census, and the likelihood of long term effects was estimated in congenitally infected people born in Britain.

Subjects and methods

All adults and children who attended any of the nine NHS ophthalmology units (including eye clinics and eye casualty departments) within the study area (see figure) were identified prospectively for one year, October 1992 to September 1993.

Ophthalmologists at all nine units agreed to participate, and the study protocol was discussed personally and in detail with consultants and all middle grade medical staff at each of the units before the start of the study. Throughout the study two of us (MRS and HJ) were in regular clinical contact with all the units. As some residents may have been seen at tertiary referral centres in north London, consultants specialising in uveitis at Moorfields Eye Hospital, the Western Ophthalmic Hospital, and Great Ormond Street Hospital for Children were included in the study protocol.

Ophthalmologists were asked to report all cases

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presenting with symptoms of uveitis and signs suggestive of acute toxoplasma retinochoroiditis. All patients with suspected acute toxoplasma retinochoroiditis were reviewed clinically by one of us (MRS or HJ): a clinical history and demographic details were recorded by interview questionnaire and a blood sample was taken for serological investigation for toxoplasma infection according to a standard protocol.¹⁰ Toxoplasma retinochoroiditis was defined as the presence of intraocular inflammation in association with a retinochoroidal scar and an active adjoining retinitis.

The incidence of symptomatic toxoplasma retinochoroiditis was calculated separately for all patients born in Britain and for black patients born in west Africa. The denominator was estimated from the local base statistics from the Office of Population Censuses and Surveys 1991 census for country of birth and ethnic group and from the greater London census data for age group by country of birth.¹¹

The estimation of the risk of developing acute toxoplasma retinochoroiditis in congenitally infected offspring born in Britain was based on three assumptions. Firstly, about 600 congenitally infected children born in Britain survive to adulthood each year.⁹ This calculation was based on an incidence of maternal infection of 2/1000 pregnancies,¹²⁻¹⁴ maternal-fetal transmission of infection in 40% (2/1000 × 40%),^{8,15} and a mean of 800 000 births/year in Britain between 1948, when the oldest patient was born, and 1992.

Secondly, patients experience a lifetime mean of two acute symptomatic episodes that require ophthalmological attention.^{7,16,17}

Thirdly, all toxoplasma retinochoroiditis is due to congenital infection.

Results

In 1992-3, 32 patients who lived within the study area (figure) were reported with symptoms of reactivation of toxoplasma retinochoroiditis. Two patients were seen at one of the tertiary referral centres outside the area and the 30 remaining were reported from six ophthalmology units within the area. At the three centres at which no cases had been reported, further inquiry confirmed that none had been seen.

Four patients were excluded from further analysis: two because of incomplete data; one patient had evidence of acute primary toxoplasmosis characterised by a systemic illness, retinal lesions typical of primary acquired infection (distinct from retinochoroidal lesions due to reactivation of latent infection), and IgM antibodies in serum specific for toxoplasma; and one patient had sarcoidosis.

Of the 28 patients remaining, 15 were male; six were white Europeans born in Britain; three were black west Africans born in Britain; two of whom had spent most of their childhood in west Africa; 16 were black and had been born in west Africa; one was black and had been born in the United States but had lived mostly in west Africa; one was mixed race and had been born in

Central America; and one had been born in the Middle East. No patients had been born in the West Indies. The median (range) age at entry to Britain for those born or living outside the United Kingdom (21/28) was 22.2 (5.0 to 37.1) years.

The diagnosis was confirmed by clinical examination by HJ or MRS in all 28 patients. The median (range) age at examination was 28.8 (13.6 to 44.4) years. All presented with symptoms attributable to reactivation of toxoplasma retinochoroiditis. No systemic symptoms indicative of acquired or congenital infection were reported and all had IgG specific for toxoplasma (dye test ≥ 2 IU) but no IgM detected in their serum. Sixteen patients had previously experienced similar attacks which required ophthalmological attention: six had experienced their first episode during childhood (age unspecified); for the remainder, the median (range) age at first attack was 23.4 (12.2 to 37.4) years.

Retinochoroidal lesions due to toxoplasmosis were present in both eyes in seven of the 28 patients and lesions were present inside the macular arcade in 13. During the episode under study, vision was reduced to 6/12 or less in 23 patients: visual impairment was unilateral in 22 and bilateral in one, six having unilateral visual impairment of 6/60 or less.

The incidence of acute symptomatic toxoplasma retinochoroiditis among all people born in Britain and resident in the study area was 0.4/100 000 population/year (9/2 314 027 (95% confidence interval 0.1 to 0.7/100 000/year with Poisson distribution)). If we assume two acute episodes per lifetime and apply this rate to the native born population of Britain (51 million), we would expect 200 episodes per year from 100 patients with congenital toxoplasmosis, equivalent to 20% of the 500-600 infants believed to be born each year with congenital toxoplasmosis.

The incidence of symptomatic retinochoroiditis for black people born in west Africa was 57/100 000/year (16/27 922 (29 to 90/100 000/year)). No patients were reported on who had been born in the West Indies or in east Africa, although 10 Asian east Africans, two black east Africans, and 16 black West Indian patients would have been expected if the rate for black west Africans was applied.

Discussion

The annual incidence of acute symptomatic toxoplasma retinochoroiditis of 0.4/100 000 subjects born in Britain was much lower than expected from the predicted prevalence of retinochoroidal lesions. Of the estimated 600 congenitally infected subjects born each year, less than one fifth would develop such ocular symptoms long term. This proportion contrasts with results of two clinic based studies, in which 18 out of 22 initially asymptomatic or mildly affected, congenitally infected neonates eventually developed retinochoroidal lesions after 20 years of follow up.¹² The proportion who developed symptoms associated with reactivation of toxoplasma lesions is not known, but six (27%) had permanent visual impairment (none were blind). Three factors could explain the disparity between the proportion of patients developing acute retinochoroiditis and the predicted prevalence of retinochoroidal lesions. Firstly, not all patients with retinochoroidal lesions experience symptoms due to reactivation. Secondly, previous studies may have overestimated the prevalence of retinochoroidal lesions. Finally, underascertainment may have occurred in our study.

SYMPTOMS DUE TO REACTIVATION

Firstly, information is lacking on the proportion of subjects with retinochoroidal lesions who experience symptoms due to reactivation, often delayed until the

Geographical distribution of patients with acute symptomatic retinochoroiditis in south London by place of residence. Four patients are excluded (see text)

second, third, or fourth decade of life.⁶ Lesions which affect the macula and cause visual impairment, however, may be clinically important even if acute retinochoroiditis never occurs. In childhood, amblyopia affects approximately 2.5% of preschool children.¹⁸ According to a recent study in west Lambeth (see figure) toxoplasma retinochoroidal lesions were found in only one out of 92 children with confirmed amblyopia between 1992-4 (E Tomlin, personal communication). Adults with unilateral visual impairment may not come to ophthalmological attention, but toxoplasma retinochoroiditis seems to be a rare cause of bilateral impairment of vision. In England and Wales for the year 1990, 30 000 registrations for partial or complete blindness have been reviewed, and toxoplasma infection was mentioned in only 24 cases (C Rooney, Office of Population Censuses and Surveys, personal communication). The proportion of those patients born in Britain or with experience of symptoms of reactivation of retinochoroidal lesions is not known.

PREVALENCE OF RETINOCHOROIDAL LESIONS

Secondly, previous follow up studies^{1,2} may have overestimated the risk of clinical effects as children who seem to be unaffected are more likely to be lost to follow up.^{19,20} Evidence to suggest that the prevalence of retinochoroiditis may be lower is provided by a recent report of population based, neonatal screening for congenital toxoplasmosis²¹: of 600 000 Guthrie cards tested for IgM specific for toxoplasma, 50 cases of congenital toxoplasmosis were identified (birth prevalence < 1/10 000 live births) and 12 out of 48 cases (25%) had retinochoroiditis after one to six years of follow up, of which seven (15%) showed unilateral visual impairment due to macular lesions.

A disparity between the expected number of affected people and the reported number had previously been found for severely affected, congenitally infected children. In previous studies, between 3% and 11% of congenitally infected infants were severely affected, and some died perinatally or during infancy.²²⁻²⁴ A national surveillance study, however, identified only 14 cases in England and Wales in 1989-90, not all of whom were severely affected, compared with 50-70 predicted.²⁵

UNDERASCERTAINMENT

Thirdly, the number of patients with symptoms due to reactivation of toxoplasma infection who were missed in this study is probably small: symptoms associated with reactivation of toxoplasma retinochoroiditis typically include sudden deterioration of vision, a painful red eye, and floaters,⁴ which would usually result in patients presenting directly to eye accident departments, followed by review in an ophthalmology clinic. If seen by a general practitioner or optician, such patients would be referred directly to the ophthalmology clinic. Ophthalmologists may have failed to report cases, but in view of the rarity and characteristic appearance of toxoplasma retinochoroiditis and the need for clinical review of cases this is unlikely. The absence of any subjects with the condition in three districts in this study probably reflects the small number of black residents who had been born in west Africa (n=2616).

Some children with symptoms may not have been seen by an ophthalmologist, either because of lack of recognition in preverbal children or because of the presence of severe congenital disease managed by paediatricians. No children were seen from the south London study area by the ophthalmologists at the tertiary referral centre for children during the study year, although one was seen in the subsequent year.

PROPORTION ATTRIBUTABLE TO CONGENITAL PLASMOSIS

Estimates of the proportion of congenitally infected subjects who develop acute symptomatic retinochoroiditis also depend on the accuracy of the estimated incidence of congenital toxoplasmosis and whether all toxoplasma retinochoroiditis is attributable to congenital rather than acquired infection. On balance, 20% is likely to be an overestimate: the incidence of toxoplasma infection during pregnancy may have been higher 30-50 years ago,²⁶⁻²⁸ it may have been underestimated in previous studies,¹²⁻¹⁴ and fetal infection may have occurred more commonly in untreated women.²⁰ Most importantly, not all toxoplasma retinochoroiditis is due to congenital infection. Retinochoroiditis due to acquired toxoplasma infection has been well documented^{4,6,17} and, in the absence of clinical or serological indicators of recent infection, is indistinguishable from congenital infection. Outbreaks of toxoplasma retinochoroiditis have been reported in siblings,^{29,30} and in a population based study in southern Brazil, where toxoplasmosis is common, a fifth of adults had toxoplasma retinochoroidal lesions.³¹ Most toxoplasma retinochoroiditis was due to acquired infection as only 1% of cord blood samples in that population were positive for IgM. Retinochoroiditis has been estimated to occur after 2% of cases of symptomatic acquired toxoplasma infection,⁶ although up to 90% of acquired infections may be asymptomatic.¹⁷

EFFECT OF COUNTRY OF BIRTH

The high incidence of symptomatic toxoplasma retinochoroiditis in black subjects born in west Africa, compared with no cases among black people born in the West Indies or in east Africa, reflects previous findings that country of birth is a key determinant of previous exposure to infection.³² When the denominator population was limited to those aged 10-44 years the rate among black people born in west Africa was still nearly 100 times higher (77/100 000/year, (38 to 120); 16/20 884) than the rate among those born in Britain (0.8/100 000/year, (0.3 to 1.4); 9/1 116 321).

Risk factors for exposure seem to have changed: 30 years ago Chesterton and Perkins found that most patients seen in north east London were black and had been born in the West Indies.⁵ In our study the high incidence of symptomatic retinochoroiditis among west Africans compared with east Africans, West Indians, and subjects born in Britain may reflect a higher incidence of congenital or acquired infection, an increased prevalence of retinochoroiditis in infected subjects, or a higher incidence of symptomatic episodes in subjects with toxoplasma retinochoroidal lesions. Such differences may be associated with greater exposure to *Toxoplasma gondii* because of diet or living conditions, exposure to a more virulent strain, or, possibly, genetic differences in susceptibility to infection or disease manifestation.

IMPLICATIONS FOR PRENATAL SCREENING

Advocates of prenatal screening have cited the high risk or long term effects which are assumed to be preventable if infection could be identified and treated during pregnancy as a major benefit of screening in both clinical and economic terms.³³⁻³⁵ In contrast, our results suggest that only one fifth of congenitally infected people born in Britain would be expected to develop symptoms of reactivation of toxoplasma retinochoroiditis in the long term. Moreover, the most severe symptom documented in 21% of cases was unilateral visual impairment (6/60 or less): this is likely to have been temporary in most cases as visual acuity improves as inflammation subsides.

More information is needed on the prevalence of visual impairment due to toxoplasma retinochoroidal

Key messages

- Toxoplasma retinochoroiditis usually develops in the second and third decades of life and is generally attributed to congenital infection
- Prevention of toxoplasma retinochoroiditis is considered to be a major benefit of prenatal screening as previous studies have suggested that 80% of congenitally infected children would eventually develop retinochoroiditis
- This report shows that the incidence of symptomatic toxoplasma retinochoroiditis is nearly 100 times higher in black people born in west Africa than in subjects born in Britain
- In subjects born in Britain acute symptomatic toxoplasma retinochoroiditis is much less common than expected, affecting about one fifth of the estimated number of congenitally infected people
- These findings suggest that the potential benefits of prenatal screening for toxoplasma infection in Britain are limited

lesions and on the proportion of retinochoroiditis attributable to acquired rather than congenital infection. These factors could have led to an underestimate or overestimate, respectively, of the proportion of congenitally infected subjects who develop ocular symptoms in the long term.

The benefits of potentially preventing ocular symptoms in the long term need to be balanced against the risks of repeated testing of all pregnant women, the participation of a large number of women falsely identified as infected and those truly infected in unnecessary invasive investigations and treatment, and unnecessary terminations of pregnancy.^{9 15 36 37} Evidence that symptomatic retinochoroiditis is less common than suggested by previous estimates of the prevalence of retinochoroiditis implies that prenatal screening for toxoplasmosis may be of limited value in Britain.

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Randomised clinical trial of ultrasound treatment for pressure ulcers

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One in four Dutch doctors and supervisory nurses in nursing homes thinks that ultrasound treatment is effective for pressure ulcers.¹ McDiarmid *et al* found positive results only in a subgroup of patients with infected ulcers.² We performed a placebo controlled trial of ultrasound treatment for pressure ulcers.

Patients, methods, and results

We randomly assigned 88 patients (median age 81) in 12 nursing homes, to genuine or sham (detuned) ultrasound treatment for stage II pressure ulcers (partial thickness skin loss or worse). Randomisation was done in blocks of four after stratification by nursing home and according to whether muscle was affected (16 patients, yes; 72, no) and ascorbic acid supplements were being given (43 patients, yes; 45, no).

The ultrasound devices had 20 codes randomly divided over the two treatment options. Each patient was assigned a treatment code and treated five times weekly over 12 weeks, or until complete healing of the ulcer. The manufacturer checked the power output of the ultrasound devices every three months. The main-