

recessive inheritance. The condition has been assigned the McKusick catalogue number 206700.⁵

Several authors have added single case reports to the literature.^{6,7,8} Sibling pairs have also been reported^{9,10}; in neither case was consanguinity noted. Crawford *et al*¹¹ reported a family with three affected members; a brother and sister had Gillespie syndrome; the sister later married an unrelated healthy male and had an affected son. The authors suggested that the sister had married a heterozygote carrier and that provisionally the disorder should still be regarded as autosomal recessive. An alternative explanation of autosomal dominant plus reduced penetrance, with most affected individuals not reproducing, cannot be totally excluded.¹⁰

In conclusion, parents of an affected child should still be advised of a one in four recurrence risk. Further cases should continue to be described in the literature.

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Reply

EDITOR.—We are grateful to Dr Quarrell for suggesting a more specific diagnosis of Gillespie's syndrome in the case we reported. She presented with developmental delay, partial aniridia, and was late in achieving her motor milestones. Cerebellar ataxia was not confirmed on clinical examination and it was felt she did not fulfil the characteristic triad of Gillespie's syndrome. An atrophic vermis and dilated fourth ventricle was reported by Nevin and Lin¹ in a case of Gillespie's syndrome. These changes were present in our case, supporting Dr Quarrell's argument.

The syndrome is rare but should be considered in the differential diagnosis of cases presenting with congenital aniridia to allow genetic counselling.

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- Nevin NC, Lin JHK. Syndrome of partial aniridia, cerebellar ataxia and mental retardation in Gillespie syndrome. *Am J Med Genet* 1990; 35: 468-9.

An intralenticular foreign body and a clear lens

EDITOR.—We would like to draw your attention to the following case which we feel is of interest.

A 21-year-old mechanic presented with a red, uncomfortable eye and blurred vision, 3 days after noticing an object hit his left eye while working in a slate pit. There was a sealed, laceration of the central cornea, and a puncture wound of the overlying anterior lens capsule. A large intralenticular slate fragment was observed towards the lens equator (Fig 1). The



Figure 1 Slit image photograph demonstrating the intralenticular particle of slate at 1 year.

posterior capsule and the retina were unaffected. The fragment was left in situ, since slate is chemically inert.¹ At 1 year, his visual acuity was 6/6. The anterior capsule wound had healed (Fig 2), but the lens has not opacified (Fig 3).

In 5% of cases of perforating ocular injuries with retained intraocular foreign bodies, the foreign body lodges in the lens,² which usually becomes opaque and requires cataract extraction for visual rehabilitation.^{1,3} Documented

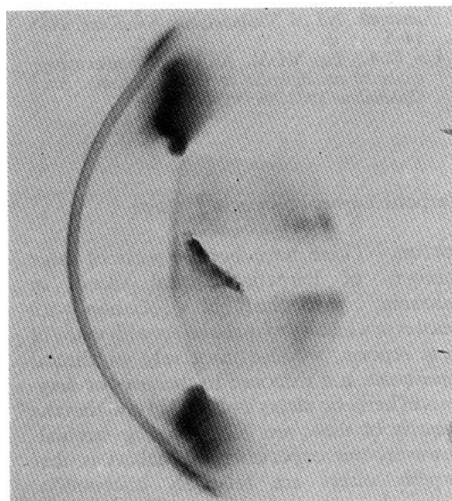


Figure 2 Scheimpflug photograph demonstrating the reformation of the anterior lens capsule with the intralenticular foreign body in the anterior cortex at 3 months.

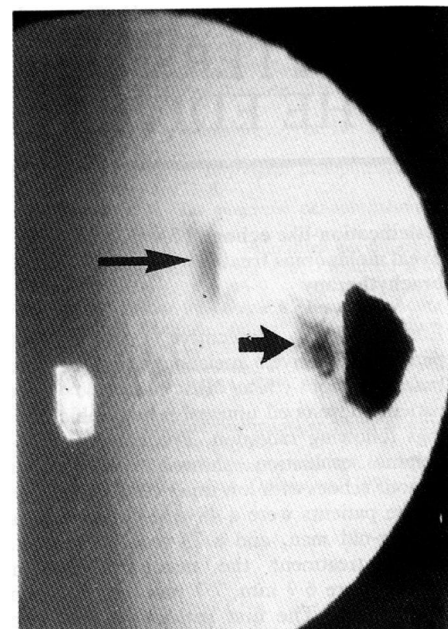


Figure 3 Retroillumination photograph showing the slate particle against the red reflex at 1 year. The thin arrow shows the corneal scar and the thick arrow shows the anterior capsule scar, but the red reflex is otherwise clear.

cases of retained intralenticular foreign bodies are either associated with mature cataracts, or localised lens opacities.^{1,4} Unprogressive, localised lenticular opacities, such as capsular scars, opacities along the track of the injury, or posterior subcapsular opacities have also been described following penetrating injuries with small sharp objects such as needles.¹ The healing capacity of the anterior lens capsule, in contrast to the posterior capsule, is well documented and is thought to be due to the presence of the subcapsular epithelium.^{1,5} Epithelial proliferation creates a plug which seals the wound. The plug reduces as new capsule is formed, and reconstituted lens fibres fill in the track. We believe that our patient did not develop a significant opacity because the posterior capsule was intact, and the entry site was small and linear, allowing the breached capsule to seal itself rapidly. We believe that this case is of interest since it is extremely rare to find reports of intralenticular foreign bodies with minimal opacification,⁶ and it is normal practice to remove such lenses. By electing to wait and observe the outcome, we have avoided surgery with the subsequent refractive problems that can occur in young patients.

We thank Mr Awdry and Mr Cheng for allowing us to report this case.

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