Clinical characteristics of unilateral myopic anisometropia in a juvenile optometric practice population

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Abstract

Purpose: A retrospective study of longitudinal case histories, undertaken to establish the clinical and statistical characteristics of unilateral myopic anisometropia (UMA) amongst the juvenile and adolescent population at an optometric practice, is reported. UMA was defined as that specific refractive state where an unequivocally myopic eye is paired with a ‘plano’ (spherical equivalent refraction, \( \text{SER} = \text{+0.25 Dioptres (D)} \)) companion eye.

Methods: The clinical records of all patients aged <19 years on file at an established independent optometric practice were categorised as ‘myopic’ (\( \text{SER} \leq \text{+0.50 D} \)), ‘hypermetropic’ (\( \text{SER} \geq \text{+0.75 D} \)) or ‘emmetropic’ (\( \text{SER} \geq \text{+0.37} \) to \( \text{+0.62 D} \)). Subsequently all juvenile patients matching the UMA criterion, together with a case-matched group of bilaterally myopic individuals, were selected as the comparative study populations.

Results: A total of 14.4% (\( n = 21 \) of 146) of the juvenile myopic case histories were identified as cases of UMA. More than half of these UMA cases emerged between the ages of 11.5 and 13.5 years. There was a marked female gender bias. The linear gradient of the age-related mean refractive trend in the myopic eye of the UMA population was not statistically significantly different (\( p > 0.1 \)) to that fitted to the ametropic progression recorded in either eye of the case-matched population of young bilateral myopes; uniquely the slope associated with the companion eye of UMA cases was statistically significantly (\( p < 0.025 \)) less steep. Compared with bilateral myopes fewer cases of UMA required a refractive correction to relieve visual or asthenopic symptoms, and this initial correction was dispensed on average 1 year later (at age 12.7 years) in UMA patients.

Conclusions: Individuals identified as demonstrating clinically-defined UMA can be considered as distinct but functionally normal cases on the continuum of human refractive error. However, any unilaterally-acting determining factor(s) underlying the genesis of the condition remain obscure.

Keywords: adolescent, juvenile, myopia, optometric practice population, unilateral myopic anisometropia

Introduction

Anisometropia, by definition, exists whenever a difference in refractive status is present between an individual’s pair of eyes. Any refractive combination and degree of imbalance is possible: the specific clinical condition under examination here – unilateral myopic anisometropia (UMA) – is that situation where an unequivocally myopic eye is paired with one having a plano refraction.

The existence of any determining factor(s) for anisometropia remains obscure. A genetic basis is a possibility (Sorsby, 1972), although for low degrees (e.g. < 1.00 D) and in isolated cases (i.e. where no other family members are affected) the condition can be regarded as sporadic (Lyle, 1990). Specifically in myopic cases, it is recognised that it is the relative axial length of the posterior vitreous chamber that constitutes the
primary physical basis for the inter-eye refractive imbalance (Sorsby et al., 1962; Logan et al., 1995).

Unfortunately, with the possible exception of two studies on the prevalence of anisometropia in school children (Pärssinen, 1990) and adults (Weale, 2002), statistics relating to the distribution of the several varieties of anisometropia tend to be confused or even contradictory (for example, see Hirsch, 1967, and the literature surveys of Duke-Elder and Abrams, 1970, and Laird, 1991). Much of this uncertainty can be attributed either to a lack of agreement on the criterion to be adopted for the inter-ocular refractive difference taken to define ‘anisometropia’ or to the decision on whether or not to include pathological or amblyopic cases in the study population.

As a precursor to another study, a retrospective analysis of longitudinal sequences of refractive case records was undertaken to document the clinical characteristics and ‘natural history’ of UMA in a juvenile and adolescent optometric population.

Methods

The clinical files at an independent optometric practice established for nearly 15 years were surveyed in the late summer of 2003. The refraction records of all patients (Caucasian) who were aged <19 years (typically about 25% of the practice’s patient base) were identified, producing a total of 724 case histories. The upper age limit was specified because the refraction should have stabilised in the majority of individuals by the mid-to-late teens (Goss and Winkler, 1983). Refractive corrections were maximally ±6.00 DS with astigmatism no greater than ±2.00 DC.

All refractions had been undertaken in the same test location by author JSP. Routine non-cycloplegic procedures were the norm, using a standard spectacle trial lens set graded in 0.25 D intervals to achieve best subjective acuity on the Snellen chart at 6 m: the refraction end-point was monitored using the duochrome test to achieve equality or a just-on-red bias.

Based on an overview of their available series of bilateral refractions, these juvenile and adolescent individuals were classified as follows: ‘myopic’ (spherical equivalent refraction, (SER) ≤−0.50 D: 146 or 20.2% of cases), ‘hypermetropic’ (SER ≥+0.75 D: 72 or 9.9%) and ‘emmetropic’ (SER ≥−0.375±0.62 D: 506 or 69.9%).

Within the myopic refractive classification n = 21 individuals (14.4% or around 1-in-7 of the juvenile myopes) were, at two or more consecutive examinations, myopic in one eye and ‘plano’ (SER = ±0.25 D) in their companion eye: this group of case histories became the clinically-defined population of UMA cases. The remainder of the myopic classification recorded consistent bilateral ametropia: from this pool of material a second group of n = 21 individual case histories was assembled. Selection for this group was on a case-matched basis (gender, age, and magnitude of the refraction in the more-myopic eye) against the same characteristics of the n = 21 UMA patients.

There was no clinical difference between the Snellen acuity of the two eyes of these unilaterally and bilaterally myopic individuals. For either eye it was recorded as being at least 6/9 (with a refractive correction if necessary), the majority attaining 6/6 or better in their pre-teen and adolescent years.

The distance and near oculo-motor balance of all individuals in these two study groups was recorded as being within normal clinical limits at each of their sight tests, with no evidence of amblyopia or manifest ocular deviation: standard screening with the Lang Stereotest II at 40 cm had also indicated stereopsis better than 200 s arc in all individuals at their initial eye examination at the practice. The gender, age and refractive details of all subjects were entirely non-attributable in subsequent analysis thus preserving patient confidentiality.

Results and analysis

For either group, for each of the n = 21 contributing cases, a longitudinal refractive history was available from the clinical records. The mean ± S.D. (standard deviation) number of sight tests was 5.67 ± 3.02 per UMA patient and 5.14 ± 2.24 per bilateral myope: the mean interval between examinations was 1.16 ± 0.67 years for UMA cases and 1.13 ± 0.59 years for the bilateral myopes.

The refractive material of the UMA patients, summarised as the inter-ocular refractive difference ΔSER (=consistently myopic eye SER minus (initially) plano companion eye SER, D], is displayed vs patient age in Figure 1a. The point where each of these subjects first met the specific clinical definition of UMA adopted here is indicated by a circular symbol. The mean age when UMA was first identified in this group was 12.37 ± 2.27 years: 52.4% (n = 11 of 21) of these cases emerged between the ages of 11.5 and 13.5 years, with the remainder arising in equal numbers before and after this 2-year span.

The availability of these series (n = 21) of refractive histories also permitted a cross-sectional investigation of any age-related refractive trend in this material. The calculation (after Harris, 1991) was undertaken of the mean of each set of sphero-cylindrical prescriptions across all available individuals of a given age (increments of 1 year: ≥6 < 19 years): also the mean SER was established across contributing prescriptions for both the myopic and the companion eyes at each age grouping. The results of this procedure are given in
Table 1 and are illustrated in Figure 1b. The broad picture is of an increasingly negative (i.e. myopic) refractive trend in either eye across the 13 year age span studied. An initial steady rise is checked briefly around the age of 9–10 years, before continuing at a steeper rate for a couple of years before that increase is also reined in, subsequently to be maintained at a plateau from the mid-teens onwards. The similarity of pattern between the two eyes is striking, with the eye that was initially approximately plano (unfilled symbols in Figure 1b) itself becoming myopic with advancing adolescence but at a lesser rate and to a lower degree than its continuously myopic companion eye (solid symbols).

Regression analysis provided good linear fits (dashed lines) to these series of age-related refractive data:

![Figure 1](image.png)
myopic eye ($y = 0.60 - 0.10x$, $r = -0.94$, $r^2 = 0.88$) and companion eye ($y = 0.56 - 0.06x$, $r = -0.91$, $r^2 = 0.83$). Also shown on Figure 1b are the linear regression fits (lightly dotted lines) to the age-related refractive trends of each eye of the case-matched bilaterally myopic population: most myopic eye (M': $y = 0.55 - 0.09x$, $r = -0.93$, $r^2 = 0.86$) and least myopic eye (L': $y = 0.53 - 0.08x$, $r = -0.92$, $r^2 = 0.85$).

Nonparametric statistical testing (Kolmogorov–Smirnov) confirmed that the dependent variable SER was normally distributed in all of this material. Consequently the same statistical technique (two-sample testing) was then employed to assess the extent of any difference between the individual linear fits to these four sets of age-related refractive material.

The linear gradient fitting the distribution of the (initially) plano companion eye of the UMA cases was shown to be statistically significantly different to the slopes fitting the other three classes; $p < 0.025$ vs myopic UMA eye data or most myopic eye data of the bilaterally myopic matched cases; $p < 0.03$ vs least myopic eye data of the bilaterally matched cases. However, no statistically significant difference was indicated between the linear regression gradient through the myopic UMA eye data and that through either the most or the least myopic eye of the bilaterally myopic population: $p > 0.1$ in each instance. In addition, there was no statistically significant difference ($p > 0.1$) between the slopes fitting the two eyes of the bilaterally myopic cases.

The clinical impression stated above is thus substantiated statistically: the (initially) plano companion eye in UMA cases does indeed manifest a uniquely lesser average rate of myopic change with increasing age, not only in comparison to its fellow consistently myopic eye but also vs the age-related refractive trend in visually normal bilaterally myopic patients.

Reviewing other comparative clinical aspects of the two juvenile myopic optometric populations, it was found that overall two-thirds ($n = 14$ of $21$) of the UMA patients required a refractive correction before the age of 19 years for the relief of visual/asthenopic symptoms. The mean age at which this first correction was dispensed was $12.72 \pm 3.12$ years: at this point the mean prescription (methodology after Harris, 1991) across the myopic eyes was $-0.87/-0.12 \times 78.35$ and for the companion eyes was $-0.15/-0.16 \times 86.29$. Compared with the UMA cases, a much larger proportion (90.5%; $n = 19$ of $21$) of the bilaterally-myopic patients required a refractive correction before the age of 19 years. While clinically significant, this difference in prescribing rate was only of borderline statistical significance ($\chi^2 = 3.53$; $p = 0.06$). The mean age at which this first prescription was dispensed was exactly 1 year in advance of the UMA patients, being $11.70 \pm 2.80$ years. The mean prescription was $-0.95/-0.18 \times 89.62$ and $-0.79/-0.16 \times 81.52$ for the greater and lesser myopic eyes, respectively.

In $57.1\%$ ($n = 12$ of $21$) of UMA cases the left eye was the consistently (more) myopic one. The laterality distribution of the more-myopic eye in the bilaterally myopic patients was similar to that found for the UMA cases, namely the left eye in $61.9\%$ of cases ($n = 13$ of $21$): $\chi^2 = 0.10$; $p = 0.75$.

Female UMA cases ($n = 16$ of $21$; $76.2\%$) greatly outnumbered males. This gender imbalance of 3:1 is substantially larger than the $22\%$ female excess (i.e. $55\%$ of patients) found across all patients aged $<19$ years at this optometric practice, and is a statistically significant feature ($\chi^2 = 9.76$; $p = 0.002$). In this regard it might be remarked that to case-match individuals between the uni- and bilaterally myopic patient groups, the marked (3:1) female gender bias uncovered in the UMA subjects had to be sustained in the bilaterally myopic comparison group. While a female bias in optometric patient numbers is a feature which has been remarked upon previously (Pointer, 1996, 2000) – and was indeed manifest at this practice – it must be acknowledged that this imbalance may have had an influence on the absolute refractive and other values derived here for these clinical comparisons between divisions of the myopic classification.

**Discussion**

On the basis of the analysis of the clinical material assembled here, it seems reasonable to suggest that cases of UMA can be regarded as a subset of bilateral myopia. More specifically, ametropia in the consistently myopic eye of juvenile UMAs progresses at a similar rate to that observed in visually normal young bilateral myopes, whereas the (initially) plano eye shows a lesser myopic change with advancing age.

This clinically-derived conclusion fits well with the sparse anato-physiological evidence available with regard to anisometropia. The consensus is that a structural difference underpins the myopic inter-ocular imbalance, the refractive difference being correlated with an inter-globe difference in axial length of the posterior vitreous chamber (Sorsby et al., 1962; Fledelius, 1981; Logan et al., 1995; Choi et al., 2003), an independent change (increase) in the corneal power being most unlikely (Ooi and Grosvenor, 1995; Zadnik et al., 2003 with the CLEERE Study Group). In addition, inter-ocular differences in fundus morphology (including the presence of optic disc crescents and increased tessellation: Curtin and Karlin, 1971) are often present, underlining the anatomical basis advanced to account for the anisomyopic condition.

However, the enduring paradox centres on why it is that anisometropia (especially in its more extreme forms)
should arise in a pair of eyes that have presumably hitherto experienced the same environmental, hormonal and nutritional conditions. While it may be reasonable to postulate that a difference in the rate of emmetropisation may account for anisomyopia in an infant, this explanation is surely untenable in the context of a healthy and visually normal adolescent who first manifests UMA when (s)he enters the second decade of life.

It is difficult to contemplate any exclusively unilateral influence or bias affecting agreed and accepted consensual neuro-physiological processes. Thus, for example, the possibility of any unilaterally-directed increase in parasympathetic or sympathetic activity or tone specifically at the level of the ciliary body would be most difficult to reconcile with the known facts regarding function of the autonomic nervous system (see Gilmartin, 1998). An investigation of tonic accommodation (or possibly of pupillary function) in a defined group of UMA subjects might give the lie to such suggestions.

Animal studies have suggested that direct, spatially-local retinal-scleral communication regulates axial length of the globe. In this regard, Norton and Siegwart (1995) have speculated that if such a neural linkage is present in man, successful childhood emmetropisation must involve not only an intact and functional emmetropisation mechanism but also guaranteed exposure to a non-compromised visual environment. Again, the assumption is that any external influence will be bilateral and that any internal effect will invariably affect the neuro-physiology of both eyes.

It is similarly difficult to envisage hereditary influences having anything other than a bilateral effect upon refractive development. Edwards (1998) has speculated that there might be ‘... a genetic programming which affects the two eyes differently, or which affects the susceptibility of the two eyes to some environmental factor(s) differently’. It remains to be seen whether the burgeoning genetic research directed at the decoding of the human genome throws any light on this specific developmental conundrum. However, with regard to the clinical material presented here, it must be remembered that a substantial proportion of UMA cases emerge not in infancy but in young persons entering adolescence (refer to Figure 1a).

Conclusions

Defining UMA as a myopic vs a plano refraction, the incidence of this specific refractive type amongst healthy and normally-sighted optometric patients aged <19 years was 2.9% [which proportion equated to approximately 0.7% of the total (all ages) patient base at this practice].

A unique clinical feature of UMA cases identified in this present study is the slower age-related ametropic progression documented in the (initially) plano eye of such individuals compared with not only the trend observed in the consistently myopic companion eye but also to that recorded in visually normal bilaterally myopic young persons.

The clinical evidence discussed here implies that cases of UMA should be regarded as a subset of the myopic population, i.e. as functionally normal but distinct points on the continuum of human refractive error. Either eye, but specifically that which manifests consistent myopia, displays refractive and other characteristics typical of a visually normal developing myopic eye. Any treatment applied to an approximately ‘plano’ eye which concurrently acted as an inherent control for its fellow myopic eye would have obvious experimental utility in trials employing either optical (bi-/multifocal spectacle lenses: Gwiazda et al., 2003), assumed-physical (rigid gas-permeable contact lenses: Walline et al., 2001) or pharmacological [e.g. Pirenzepine (a relatively selective M1-muscarinic antagonist) 2% ophthalmic gel b.i.d.: Tan et al., 2003; Siatkowski et al., 2004] therapeutic methods of inhibiting myopic progression.

References


