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Cataract surgery

Do waiting times really matter?

Melissa M Brown

Shortening the waiting time for cataract surgery improves patients' quality of life

Mojon-Azzi and Mojon¹ have performed a superb analysis (see pages 282) which demonstrates that the waiting time for cataract surgery in 10 European countries is influenced primarily by the total expenditure on health ($p < 0.01$). Of note is the fact that public expenditure on health, physician density and acute hospital bed density did not significantly influence waiting times.

Since cataract waiting times differ significantly among countries ($p < 0.001$), a reasonable question to ask is, “do waiting times really make a difference for the average patient?”. Fortunately, a value-based medicine analysis sheds some light on this issue.

Excellent evidence-based data come from the PORT study, in which the average person who underwent cataract surgery had a visual acuity of 20/83 in the affected eye.^{2,3} The average postoperative visual acuity, factoring in the complications of posterior capsular opacification, endophthalmitis, loss of lens particles into the vitreous cavity, intraocular lens dislocation, retinal detachment, cystoid macular oedema and bullous keratopathy, was 20/27.³

Utility values allow us to reproducibly quantify the quality of life associated with a health state.⁴ Utilities also allow us to calculate the total value (improvement in

quality of life and length of life) conferred by virtually all interventions. For ocular procedures, the value gain is typically conferred by improvement in quality of life rather than improvement in length of life.

With vision, utility values decrease as the corresponding visual acuity in the better-seeing eye decreases.⁴ Assuming that patients undergoing surgery have cataracts that are equal in both eyes, the utility value associated with 20/83 vision preoperatively is 0.71 and the utility value associated with 20/27 vision postoperatively is 0.858.³ This results in a 0.148 (0.858–0.71) utility gain conferred by the cataract surgery.

The mean age of SHARE patients waiting for cataract surgery was 73.8 years.¹ The average life expectancy for a person of this age is approximately 13 years.^{5,6}

The mean waiting time in the SHARE study was 3.3 months, but the wait to see an ophthalmologist can be up to a year.¹ Therefore, a total of 15.3 months could be necessary from the time a patient notes disabling visual loss until the responsible cataract is removed.

To calculate the quality of life lost in this instance (in quality-adjusted life-years, QALYs)^{3,4} by delaying cataract surgery, the utility gain conferred by cataract

surgery is multiplied by the time (in years) from visual loss until the cataract is removed. Thus, there is a (0.148 utility gain \times 1.275 years) = 0.19 QALY gain.

Looked at in another way, the average patient waiting 15.3 months for cataract surgery has a 21% diminution in quality of life, or life's value, during this time. This is a dramatic diminution in quality-of-life, equivalent to having an amputation versus no amputation or having clinically relevant coronary artery disease versus having none.⁷ Averaged over the remaining lifetime of the patient, the 15.3 month wait for surgery results in a 1.6% diminution in quality of life on a daily basis. This latter percentage is not as severe as the diminution during the waiting period but is still considerable when it occurs only secondary to waiting!

In essence, the quality of life associated with various health states includes more than just what happens on the days of surgery or another intervention. As healthcare providers, we should do our best to maximise the value we confer to our patients. Shortening the waiting time from the start of visual disability until the responsible cataract is removed, or alternatively the waiting time for many other healthcare interventions, is a good way to begin.

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rRNA-based tests for chlamydial infection in trachoma

rRNA-based tests for chlamydial infection in trachoma

Robin Bailey

Trachoma, the worlds leading cause of preventable blindness, is the subject of worldwide control efforts via the SAFE (Surgery, Antibiotic Treatment, Facial Cleanliness and Environmental Improvement) strategy. The “A” component of this strategy antibiotic treatment of the active disease has been supported through the large scale donation of millions of doses of the antibiotic azithromycin by its manufacturers, Pfizer, for distribution in trachoma endemic areas by the International Trachoma Initiative.¹ Azithromycin, as a single 20 mg/kg oral dose is effective against *Chlamydia trachomatis* infection.² In the field the diagnosis of active trachoma may be made simply by examining the surface of the everted upper eyelid for clinical signs of trachoma: lymphoid follicles and inflammatory thickening.³ Current recommendations are that communities in which the prevalence of active trachoma is greater than 10% of 1–9 year olds should be mass treated annually for three years.⁴ So far, so good. A problem, however is that the clinical signs of trachoma are quite poorly predictive of the presence of ocular chlamydial infection. Wherever tests for infection have been carried out, there have been significant rates of mismatch between infection and clinical signs: infection without disease and disease without infection are very common. There are also examples of whole communities with substantial rates of active trachoma in whom not a single individual has been found to harbour *C trachomatis* infection,⁵ and of communities where mass treatment has suppressed infection, but clinical signs of trachoma persisted at pre-treatment levels.⁶

Distributing azithromycin repeatedly to such communities must be considered wasteful of scarce resources.

Thus there are at least three reasons why testing for infection in trachoma may be informative. Firstly it may tell us how to prioritise individuals or communities for treatment. Secondly it may indicate when treatment, or distribution ought to be discontinued, or resumed. Finally we may learn something useful about the biology of trachoma. In their paper, Yang *et al* present the first data using a commercial assay which detects chlamydial ribosomal RNA (rRNA) in subjects with trachoma (*see page 293*).⁷ Because chlamydial rRNA, reflecting ribosomal activity, is typically present in infected cells at a multiplicity of hundreds to thousands of copies per chlamydial chromosome one would expect that, as demonstrated in their findings, rRNA based testing would be more sensitive than the more commonly applied AmpliCor PCR, which detects the common chlamydial plasmid pCT typically present at a median multiplicity of about six per chromosome in ocular infection.⁸

The advent of rRNA based tests raises more questions in need of answering. Does the detection of rRNA without chlamydial DNA really indicate an infectious reservoir of epidemiological significance? What is the prognosis for infection in these subjects? Does rRNA disappear before or after DNA following treatment?⁹ A previous study, albeit using a homebrew quantitative assay, found that high level rRNA expression was strongly predictive of clinical signs of active trachoma.¹⁰ It would be interesting to know whether quantitative estimation of rRNA in trachoma subjects

will reconcile these findings. Finally, trachoma habitually occurs in settings characterised by poverty and poor access to services and utilities. An ideal test for chlamydial infection would be specific, able to be performed at the point of care and to be interpreted by programme staff with minimal training, cheap and not requiring electricity or expensive technology. The high sensitivity conferred by nucleic acid amplification tests is likely not strictly necessary for community prioritisation and treatment-stopping decisions by programmes. A new test in dipstick format that detects chlamydial lipopolysaccharide antigen is currently undergoing evaluation and may fit the bill here.¹¹

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