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Report

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Usefulness of Icare Home in Telemedicine Workflow to Detect Real-World Intraocular Pressure Response to Glaucoma Medication Change

Based on knowledge that intraocular pressure (IOP) is a risk factor for glaucoma, clinical practice includes setting a target IOP and assessing treatment efficacy during clinic visits. There is growing appreciation that IOP fluctuation may contribute to progression.[1] Although many unanswered questions remain to define IOP fluctuation,[2] IOP measurements are limited to clinic hours and incompletely characterizes diurnal variations.

Technologies such as the Sensimed Triggerfish (Sensimed AG, Lausanne, Switzerland) and rebound tonometry (Icare HOME; Icare USA, Raleigh, NC), acquire real-world data.[3] This technology can assess fluctuation and treatment efficacy outside of the office. Given the current health environment, such technologies provide IOP data collected outside of the clinic and facilitate data-driven telemedicine.

The feasibility of acquiring real-world IOP with the Icare HOME showed that 84% of patients (n = 144/171) use this technology.[4] The precision of measurements by intraclass correlation coefficient was 0.92 comparing the Icare HOME and Goldman applanation tonometry. However, the caveat is that 1 in 6 patients fail to certify because of large IOP differences comparing Goldman applanation tonometry and Icare HOME. Huang et al[5] showed that 70% of patients could perform self-tonometry and studied diurnal and nocturnal curves in patients with newly diagnosed glaucoma and the treatment effects of initial therapy.

We present 2 patients whose cases support using such technology to obtain IOP data collected in the real world. Both patients were trained, certified, and motivated to use the Icare HOME to characterize IOP data under treatment and after changing treatment. With the current instrument design, all IOP measurements were obtained in the upright position and not supine. Both patients gave informed consent as part of an IRBMED protocol following the tenets of the Declaration of Helsinki.

A 72-year-old white man, who is an active general surgeon, transferred care for pseudoexfoliation glaucoma. He had a family history of glaucoma (mother and maternal uncle). His left eye was progressing with an IOP range of 10 to 16 mmHg. Ocular medications included fixed-combination dorzolamide 2% plus timolol 0.5% twice daily in both eyes and travoprost 0.004% every night at bedtime in both eyes. Corrected acuities were 20/30 in the right eye and 20/20 in the left eye, and IOPs were 11 mmHg in the right eye and 12 mmHg in the left eye. Central corneal thickness was 545 μm in the right eye and 551 μm in the left eye. Iridocorneal angles were open on gonioscopy. Global retinal nerve fiber layer (RNFL) thickness was 91 μm in the right eye and 75 μm in the left eye (Fig S1, available at www.ophthalmologyglaucoma.org), which corresponds to a vertical cup-to-disc ratio of 0.85 in the right eye and 0.95 in the left eye. Humphrey visual fields (24-2 Swedish interactive threshold algorithm standard) were reliable, with full-field in the right eye and superior and inferior arcuate defects in the left eye (Fig S1).

During the shortage of dorzolamide 2% plus timolol 0.5%, he was administered individual medications. Timolol 0.5% was stopped because of bradycardia. Brimonidine 0.2% was added, but then stopped because of orthostatic hypotension and blepharoconjunctivitis. His office-based IOPs were 12.7±2.25 mmHg in the right eye and 12.1±2.11 in the mmHg left eye, with a peak IOP of 16 mmHg in both eyes with treatment. A left disc hemorrhage was noted, so selective laser trabeculoplasty was performed in the left eye but did not lower the IOP. Therapy was escalated with the addition of latanoprostene bunod every night at bedtime in both eyes, stopping travoprost and continuing brinzolamide 1.0% thrice daily in both eyes.

He was motivated to capture real-world IOP variability because he was symptomatic as a result of vision loss in the left eye and was hesitant to undergo surgical intervention, given his hobbies of scuba diving and basketball. He was instructed to measure 6 times throughout the day and to add measurements during the night if he woke up over a 1-week period. However, he was committed to gathering more data and captured IOP peaks of 28 mmHg in the right eye and 43 mmHg in the left eye that occurred outside of clinic hours while taking latanoprostene bunod every night at bedtime in both eyes and brinzolamide thrice daily in both eyes (Fig 1, Top). After this 1-week period, he was instructed to stop latanoprostene bunod, to start netarsudil 0.02% every night at bedtime in both eyes, and to continue brinzolamide 1% thrice daily in both eyes. His peak IOPs were 19 mmHg in the right eye and 17 mmHg in the left eye.

A 63-year-old black man who is a computer programmer with primary open-angle glaucoma was managed since 1993 initially with timolol and then switched to latanoprost 0.005% every night at bedtime in both eyes in 1998. Risk factors included family history (mother and maternal aunt), central corneal thickness of 538 μm in the right eye and 538 μm in the left eye, and maximum IOP of 36 mmHg in the right eye and 38 mmHg in the left eye. Best acuities were 20/20 in both eyes. He was highly adherent for 20 years with a target IOP of in the "teens." His iridocorneal angles were open by gonioscopy. Office-based IOPs were 15.4±5.67 mmHg in the right eye and 16.4±5.90 mmHg in the left eye with treatment. Global retinal nerve fiber layer thickness was 86 μm in the right eye and 77 μm in the left eye (Fig S2, available at www.ophthalmologyglaucoma.org), which corresponded clinically to a vertical cup-to-disc ratio of 0.85 in the right eye and 0.95 in the left eye. Humphrey visual fields (24-2 Swedish interactive threshold algorithm standard) were reliable, with a full-field right eye and progression in the left eye with superior nasal step and inferior arcuate defects (Fig S2).

Given excellent adherence and clinic-based IOPs at target, there was concern for IOP fluctuation not being captured during office hours. His IOP data showed peaks to 23 mmHg in the right eye and 24 mmHg in the left eye while taking latanoprost every night at bedtime in both eyes (Fig 1, Bottom). After adding to brimonidine 0.2% twice daily in both eyes, his IOP peaks were 14 mmHg in the right eye and 15 mmHg in the left eye.

In summary, these carefully selected cases demonstrate that rebound tonometry can evaluate IOP modulation under treatment and changing treatment for patients with glaucoma.[6] The data captured by these individuals identified peak IOPs over a 1-week

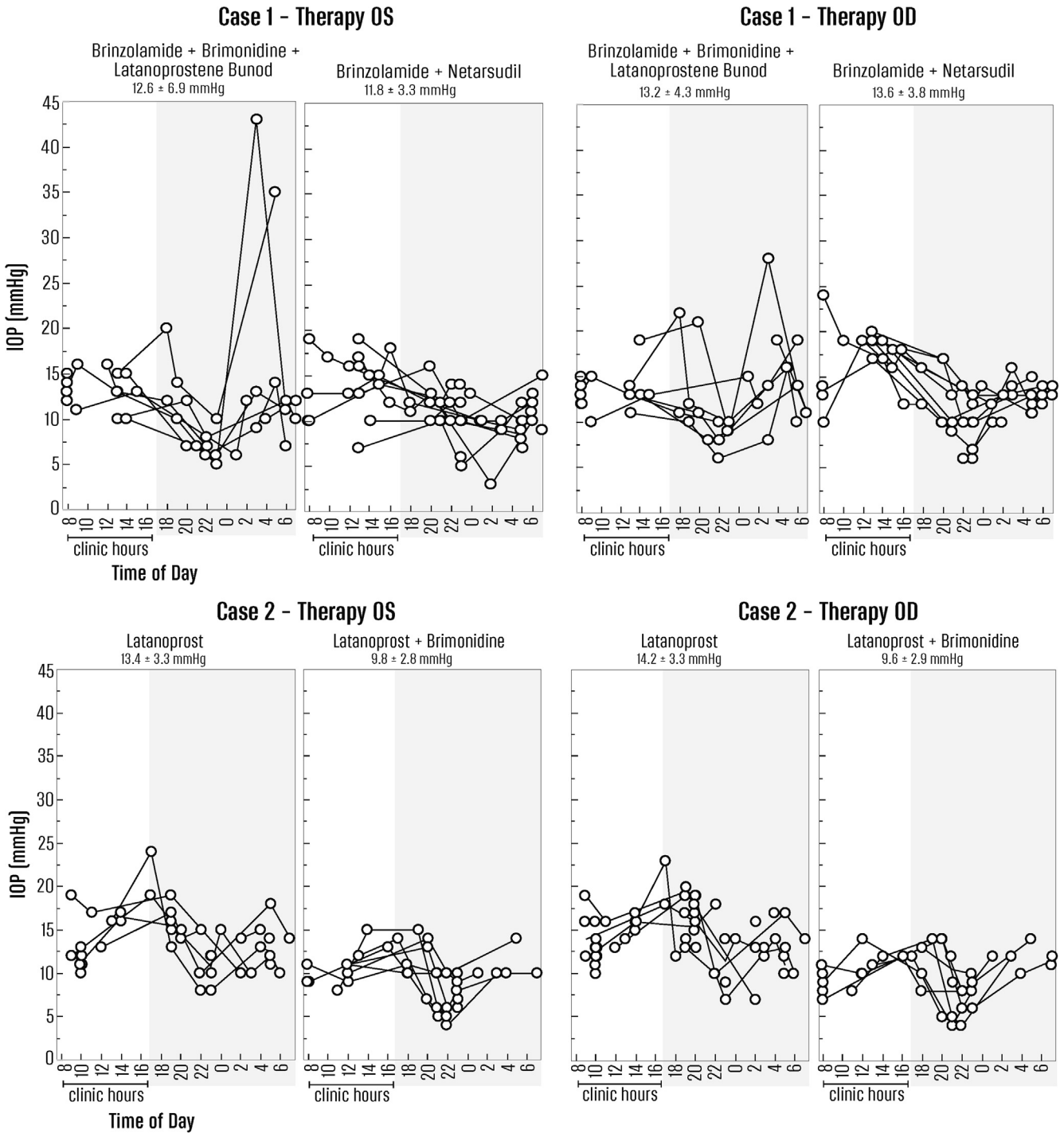

Figure 1. **A**, Patient 1: graphs showing intraocular pressure (IOP; mmHg) in the (left) left eye and (right) right eye measured by the individual throughout the day relative to the time of day. The real-world hours and homology to clinic hours (8 AM–5 PM) is on the left side of each graph. The real-world IOPs and homology to outside of clinic hours is on the right and shaded in light gray. The glaucoma medications are described at the top of each graph. The mean ± standard deviation of the IOP measurements are at the top of each graph over that measurement period. **B**, Patient 2: graphs showing the same information as in (**A**) for patient 1.

period and the effect of added glaucoma therapy to modulate these IOP peaks over a repeat 1-week period. However, not all patients can perform rebound tonometry at home.[4,5] Another limitation is the instrument cost as a barrier for general use, which we addressed by using a library-like check-out and return model for the instrument. Future studies are needed to determine both the number of IOP measurements over 24 hours and the number of repeated days sufficient to characterize the IOP curves, to capture IOP peaks, and then to understand the individual data and risk for glaucoma progression. Given current health circumstances, such

technology provides a telemedicine workflow for glaucoma management to assess the risk factor of IOP.

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No animal subjects were included in this study.

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