Choroidal Folds in Astronauts

We read with interest and enthusiasm the recent article by Sibony and colleagues1 which accurately described the biomechanical etiology of peripapillary wrinkles, retinal folds, and choroidal folds caused by idiopathic intracranial hypertension (IIH). The authors correctly state that chorioretal folds have been documented in a variety of ophthalmic disorders including the microgravity environment of prolonged space flight. We would like to emphasize two main theories that might explain the optic disc edema, globe flattening, and choroidal folds observed in astronauts during and after long-duration space flight. The first theory proposes that these changes result from a rise in intracranial pressure (ICP) similar to that seen with terrestrial IIH.2 In this case, the rise in ICP would presumably be propagated from the cerebral spinal fluid (CSF) surrounding the brain down the optic nerve (ON) sheaths to the posterior globe. This could result in a posterior pressure within the optic nerve sheath as well as an anteriorly directed pressure on the posterior globe and optic nerve head. As nicely described by the authors, this would result in biomechanical stress on the ON head and load bearing structures and ultimately produce retinal and choroidal folds. The second possible explanation, in some astronauts, is that these changes are the end result of localized events occurring at the level of the intraorbital ON with or without a rise in intracranial pressure.2,3 In this scenario, prolonged microgravity fluid shifts may cause alterations in CSF flow dynamics in the intraorbital portion of the subarachnoid space (SAS) such that CSF enters the SAS but outflow may become impeded. Thus, CSF within the SAS of the ON may gradually become sequestered with a resulting local elevation of optic nerve sheath (ONS) pressures capable of producing the anatomic changes observed. In a previously affected astronaut, following long-duration space flight, we documented asymmetric disc edema in conjunction with a normal lumbar puncture opening pressure 1 week after space flight4 as well as the unilateral loss of previously visible spontaneous venous pulsations during space flight in the eye with disc edema.5 These continued to be absent 21 months following his return to Earth with an otherwise normal disc.6 Also, a more recent report of lumbar puncture opening pressures of 22 cm and 16 cm H2O, obtained at 1 week and 1 year post mission respectively, in the presence of asymmetric disc edema still present 6 months following long-duration flight, suggests optic nerve sheath compartmentation as the etiology (Lee A, unpublished observations, 2016).

We hypothesize that, although the retinal and choroidal folds in papilledema could result from elevated ICP, these changes could also be consistent with a rise in ON sheath pressure with or without a commensurate elevation in ICP. This may be the case in some of the affected astronauts as described above.

It is interesting to note that there was only a 1% and 10% frequency of choroidal folds, by photos and OCT, in three of five astronauts (60%) with disc edema.2 One additional astronaut had choroidal folds with no visible disc edema by slit lamp fundus examination.2 All astronaut choroidal folds were noted in the right eye only. Finally, in your study, choroidal folds were the only type of folds associated with higher levels of ICP and perhaps this suggests that the relatively high frequency of choroidal folds in astronauts may also have resulted from correspondingly high intracranial or localized ON sheath pressures. Alternatively, choroidal expansion during prolonged microgravity exposure2 may make the choroid more susceptible to mechanical folding in response to lower pressure gradients, compared with terrestrial IIH or other etiologies. We commend the authors on their outstanding work and we would be interested in their additional comments in comparing and contrasting their findings with our prior work in astronauts after long-duration space flight.

Thomas H. Mader1
C. Robert Gibson2
Andrew G. Lee3

1COL (R) US Army, Moab, Utah, United States; 2Coastal Eye Associates, Webster, Texas, United States; and the 3Department of Ophthalmology, The Methodist Hospital, Houston, Texas, United States.

E-mail: tmader84@gmail.com

Acknowledgments

The authors alone are responsible for the content and writing of the paper.

References


Citation: Invest Ophthalmol Vis Sci. 2016;57:592. doi:10.1167/iovs.15-18720