JCSM
Journal of Clinical Sleep Medicine

PODCAST TRANSCRIPT

Podcast of the Journal of Clinical Sleep Medicine

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Welcome to the regular Podcast of the Journal of Clinical Sleep Medicine. I am Dr. Stuart Quan, Editor of the Journal. These Podcasts are a regular feature of each issue of the Journal and can be downloaded at the Journal's website. Each Podcast features summaries of important articles published in the current issue of the Journal, as well as occasional interviews with authors of these papers.

The lead article of the August 15, 2007, issue of the Journal is entitled, "Chronic Opioid Use Is a Risk Factor for Development of Central Sleep Apnea and Ataxic Breathing." The authors are James M. Walker, Robert J. Farney, Steven M. Rondo, Kathleen M. Boyle, Karen Valentine, Tom V. Croward and Kevin C. Shilling, from the Intermountain Sleep Disorders Center in Salt Lake City, UT. In their introduction to this study, the authors note that chronic opioid therapy is increasingly used for management of chronic pain, with relatively little attention paid to any respiratory consequences. In their study, they retrospectively selected 60 patients taking chronic opioids who had been referred to their sleep disorders center and compared them to the same number of non-opioid using patients matched on the basis of age, gender and body mass index to determine the effect of chronic opioid use on breathing patterns during sleep. They found that the apnea-hypopnea index was significantly greater in the opioid group as a result of an increased number of central apneas. In addition, arterial oxygen saturation in the opioid group was significantly greater during wakefulness and non-REM sleep, but not during rapid-eye-movement sleep. Furthermore, there was a dose-response relationship between opioid dosing and indices of sleep-disordered breathing. An interesting finding was that the body-mass index was inversely proportional to the apnea-hypopnea index in the opioid group, suggesting that the effect of opioids was most significant in those who were less obese. They also noted ataxic or irregular breathing during non-REM sleep in those who used chronic opioids. The significance of this study is to highlight for sleep practitioners the importance of chronic opioid use in worsening sleep-disordered breathing, especially central sleep apnea, in their patients. However, if opioid use cannot be reduced or discontinued in these individuals, it remains unclear as to the best way to approach treatment.

Another study in this issue of the Journal also is on the topic of central sleep apnea. It is entitled, "Central Sleep Apnea on Commencement of Continuous Positive Airway Pressure in Patients with a Primary Diagnosis of Obstructive Sleep Apnea-Hypopnea." The authors of this paper are Sanaz Lehman, Nick A. Antic, Courtney Thompson, Peter G. Catchside, Jeremy Mercer and R. Doug McEvoy, from the Adelaide Institute for Sleep Health, Repatriation General Hospital, Daw Park, South Australia and the Department of Medicine, Faculty of Health Sciences, Flinders University, Bedford Park, South Australia. In the introduction to their paper, the authors note that central sleep apnea is frequently noted during the commencement of continuous positive airway pressure therapy for the treatment of obstructive sleep apnea-hypopnea. It is also noted that the presence of central sleep apnea may limit the effectiveness of CPAP therapy. The purpose of their study was to determine the prevalence of central sleep apnea amongst patients starting CPAP therapy for obstructive sleep apnea and determine possible predictors of this occurrence. They reviewed the polysomnograms and clinical records of 99 consecutive patients who had an in-laboratory CPAP titration for obstructive sleep apnea. Patients were divided into those who had a central sleep apnea index of 5/hr or greater at their prescribed CPAP level, and those who had less than this amount of central sleep apnea at their prescribed CPAP level. They found that 13.1%, or 13 subjects, had central sleep apnea with CPAP. In comparison to those without central sleep apnea, there were no differences with respect to age or body mass index. However, almost half of the patients who had central sleep apnea with CPAP also were noted to have central sleep apnea on their baseline polysomnogram. Conversely, only 8% of those without central sleep apnea had central sleep apnea on their baseline polysomnogram. Central sleep apnea patients also had a higher apnea-hypopnea index, arousal index and mixed apnea index. They also noted that a history of ischemic heart disease or heart failure was more frequent among those who had central sleep apnea with CPAP. The authors concluded that a significant minority of individuals who undergo CPAP titration for obstructive sleep apnea will have central sleep apnea at their prescribed CPAP pressure.

Next is an interview with Dr. Ronnie Fass, who is senior author of the paper in this issue of the Journal entitled, "Relationships Between Sleep Quality and pH Monitoring Findings In Persons with Gastroesophageal Reflux Disease."

SQ: Ronnie, thank you for agreeing to be interviewed for this Podcast. What do you believe is the relationship between GERD and insomnia?

RF: I think that, based on our article as well as some previous work that we and other investigators have done, there is no question that gastroesophageal reflux disease may lead to problems due to sleep abnormalities and it appears to occur through two important mechanisms. The one that is by far the most common is the impact of reflux disease on patients' sleep through multiple awakenings. The multiple awakenings during the night lead to sleep fragmentation. The other one, which is less common but

more commonly discussed, is the fact that patients wake up during the night with heartburn and this leads to sleep abnormalities. But, as we've shown in this study, it appears to that there is also an effect of abnormal sleep or poor sleep quality on patients' gastroesophageal reflux disease and we were able to show there is an impact on esophageal acid exposure. However, we do believe that the impact is much greater and it includes more than just abnormal acid exposure but also exacerbation of GERD related symptoms through brain gut mechanisms--which is that poor quality sleep or abnormal sleep may lead to decreased perception thresholds for pain and increased symptoms report.

SQ: What do you think is the clinical significance of the findings of this paper with respect to what clinicians might take from it?

RF: I think that there are two important messages. The first one is the fact that the more severe the GERD, the more severe is the sleep abnormalities you will get and the more severe the sleep complaints you will have. But at the same time comes the second message of this study--that is poor quality of sleep per se or sleep abnormalities may also affect GERD through its impact on esophageal acid exposure. So, this bilateral relationship between GERD and sleep is extremely important for the understanding of how one can affect the other, and I think it is something that still remains unknown for the practicing clinician. They should use it in practice when they evaluate patients, even though they just come with sleep abnormalities and do not complain from the beginning about gastroesophageal reflux disease.

SQ: Having said that, do you think that patients with both GERD and insomnia should be treated for their GERD before treatment of their insomnia, realizing that perhaps the GERD is causing the insomnia?

RF: I think that that would be my first choice. If somebody comes in and they have GERD and insomnia, if there is a suspicion that GERD is the underlying cause of their insomnia, I would definitely treat the gastroesophageal reflux disease to see if I can improve their sleep. There is also some early evidence in several studies, preliminary studies, showing that if you take patients with insomnia and gastroesophageal reflux disease, and these patients have evidence of GERD then their insomnia improves.

SQ: What additional studies do you think need to be performed in this area?

RF: I think that there are several other studies that should be looked at. First of all, we are trying to establish a bi-directional relationship between sleep and GERD. So, it would be very interesting to see what would happen in patients with GERD and insomnia if we address the insomnia first and see if it has any impact on GERD from a objective or subjective point of view--for example, treating patients for their insomnia to see if it improves their GERD-related symptoms as well as potentially their physiologic esophageal acid exposure. If therapy makes any impact, it will be a very important study and very intriguing. It will clearly show that this bi-directional relationship created a vicious cycle and that we need to break it. One way which I think will establish it will be by treating GERD. But what would be very interesting is if were to treat insomnia, and it makes an impact on GERD. The other study which we have embarked on already is to look at the impact of quality of sleep per se on esophageal perception of GERD patients--if poor quality of sleep is impacting how patients perceive symptoms or from a physiological point of view how they perceive intraesophageal stimuli in the form of acid perfusion, for example.

SQ: That sounds very interesting and I look forward to seeing further studies in this area. Thank you very much for agreeing to be interviewed.

This concludes the Podcast for the August 15, 2007, issue of the Journal of Clinical Sleep Medicine.