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Perspective

Identifying Drug-Related Respiratory Adverse Events in Sleep Apnoea

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DESCRIPTION

The increasing prevalence of sleep-related breathing disorders, particularly Obstructive Sleep Apnoea (OSA) and Central Sleep Apnoea (CSA), has drawn growing attention to the influence of pharmacological agents on respiratory patterns during sleep. While OSA is primarily characterized by upper airway obstruction, CSA arises from diminished or absent respiratory effort due to impaired central regulation. Emerging evidence suggests that certain drugs, including both prescription and over-the-counter medications, may contribute to the onset or worsening of these conditions. Therefore, systematic signal detection for pharmacovigilance has become critical in identifying drug-associated risks and guiding safe prescribing practices.

Traditionally, the adverse effect profiles of medications are evaluated during clinical trials, yet many sleep-related complications, especially those of insidious onset like sleep apnoea, may go undetected until post-marketing. The low frequency and delayed manifestation of sleep apnoea syndromes make them particularly challenging to associate with specific drug exposures without advanced data mining and signal detection methodologies. With the growing availability of real-world data from Spontaneous Reporting Systems (SRS), Electronic Health Records (EHRs) and large-scale adverse event databases, signal detection algorithms are increasingly being applied to explore these relationships.

Recent pharmacovigilance analyses using disproportionality methods such as the Reporting Odds Ratio (ROR) and the Bayesian Confidence Propagation Neural Network (BCPNN) have identified several classes of medications that may contribute to the development or exacerbation of sleep apnoea. Among these, opioids, sedative-hypnotics and certain antipsychotics have emerged with noteworthy signals. Opioids, for example, are well known for their depressant effects on central respiratory drive and have been implicated in CSA. Sedatives, such as benzodiazepines and barbiturates, can promote upper airway collapse and reduce excitement level, potentially aggravating OSA.

Antipsychotics, particularly those with strong antihistaminergic or anticholinergic properties, are associated with weight gain and muscle relaxation, both of which are risk factors for OSA. Meanwhile, agents like gabapentinoids, which modulate synaptic neurotransmission, have also been observed in case reports to worsen sleep-disordered breathing. However, a strong causal inference remains elusive due to confounding variables such as underlying comorbidities, concurrent medication use and preexisting sleep disorders.

The complexity of identifying true drug-induced sleep apnoea events is further amplified by the underreporting and misclassification of symptoms. Many patients and clinicians may not recognize subtle changes in sleep quality or daytime somnolence as drug-related, particularly in the context of polypharmacy. Furthermore, diagnostic confirmation of sleep apnoea typically requires polysomnography, which is not routinely performed in pharmacovigilance contexts. This underscores the importance of integrating multiple data sources and leveraging machine learning algorithms that can detect latent patterns and subtle correlations across large populations.

The perspective moving forward is clear: There is a need to establish structured approaches for detecting and validating drug-sleep apnoea associations. These should include harmonized definitions of sleep apnoea adverse events, incorporation of natural language processing to extract symptom data from clinical notes and prospective cohort studies to substantiate findings from retrospective analyses. Moreover, there is an opportunity to develop predictive risk models that account for patient-specific factors such as age, BMI, gender, genetic predisposition and concomitant medications to identify individuals at higher risk for drug-induced apnoea.

Signal detection efforts must also be translated into clinical action. Regulatory agencies and healthcare systems should consider issuing alerts or guidelines when a consistent

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association is identified. Clinicians, especially those managing patients with multiple comorbidities or on chronic sedative therapy, should be aware of the potential for drug-induced worsening of sleep-disordered breathing. In high-risk populations, baseline and follow-up screening for sleep apnoea may be warranted when initiating medications known to affect respiratory control or upper airway tone.

In conclusion, signal detection for drugs associated with obstructive and central sleep apnoea represents an evolving frontier in pharmacovigilance and sleep medicine. While challenges remain in distinguishing causation from correlation, ongoing advancements in data analytics, integration of sleep-specific health records and collaborative reporting initiatives will enhance our ability to uncover hidden safety signals. Recognizing and minimizing the respiratory risks associated with certain medications can lead to improved patient outcomes, better sleep health and a more informed approach to personalized pharmacotherapy.