Opioid Safety: Is Your Patient at Risk for Respiratory Depression?

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Case Study: S.J. is a 42-year-old woman with stage III ovarian cancer who underwent a total abdominal hysterectomy and bilateral oophorectomy today. Other than ovarian cancer, she has no other significant medical history except sleep apnea. She is five feet, two inches tall and weighs 200 pounds. Her serum creatinine and liver function tests are within normal limits. She was taking no medication prior to admission. Her medications now include hydromorphone (1 mg IV push every three hours as needed for pain) and promethazine (25 mg IV push every four hours as needed for nausea). At 1 am, she puts on her call light. "I really have a lot of pain. It is a 9/10, and I feel sick to my stomach. Could you get me something?" she says. Her last hydromorphone was four hours earlier, and she is due for promethazine. She is alert, and her respirations are 12. The nurse gives her hydromorphone and promethazine. One hour later, the nurse returns to the room and finds S.J. difficult to arouse. S.J.'s pupils appear constricted, and her respirations are 7 and shallow. The nurse notifies the resident on call and obtains an order for naloxone. The nurse administers the naloxone and oxygen and monitors the patient's vital signs. S.J.'s respirations quickly return to normal, and she is alert and oriented.

Fear of respiratory depression is one of the major barriers to the effective use of opioids to manage pain. According to the American Pain Society (1996), withholding appropriate opioids based on respiratory concerns is unwarranted and leads to unnecessary suffering. Although death or neurologic injury for patients with otherwise treatable illnesses is tragic, serious complications from respiratory depression are not common. In most instances, clinically significant respiratory depression can be prevented by identification of high-risk patients, individualization of analgesic regimens, and close monitoring of respiratory and sedation status (Institute of Safe Medication Practices, 2002).

Pathophysiology

Most opioids commonly used in the clinical setting work primarily by binding to Mu receptor sites to produce analgesia. Mu receptors are located throughout the body, including the cerebrum and medulla (parts of the brainstem), which play an important role in respiration. Chemoreceptors in the medulla and other parts of the body detect low levels of oxygen (hypoxia) and high levels of carbon dioxide (hypercapnea). The body responds by increasing the rate and depth of respiration. Opioids bind to Mu receptor sites in the medulla and can cause respiratory depression. Naloxone, an opioid antagonist, is believed to bind to Mu receptors and reverse analgesia and other side effects of opioids, including respiratory depression (Sargent, 2002).

Definition

Clinically significant respiratory depression has been defined differently in the literature. McCaffery and Pasero (1999) defined it as a decrease in the rate and depth of respirations from a patient's baseline. A meta-analysis examining postoperative pain management found that 70 study groups defined respiratory depression as fewer than 10 respirations per minute and 24 study groups defined respiratory depression as less than 90% oxygen saturation (Cashman & Dolin, 2004). Others have defined respiratory depression as fewer than eight breaths per minute (Sidebotham, Dijkhuizen, & Schug, 1997). A lack of correlation between respiratory rate and oxygen saturation level also has been reported (Hauer, Cram, Titler, Alpen, & Harp, 1995; Overdyk, Carter, & Maddox, 2006; Sidebotham et al.; Tsui et al., 1997).

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