

Pain management with morphine: variation in analgesic response secondary to genetic polymorphisms

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To the editor

The recent article by Villesen et al in a recent issue of your journal was most interesting.¹ Recent studies show that the potency of morphine in efficacious pain control may be influenced greatly by polymorphisms of certain genes.

For instance, Klepstad et al have recently shown that cancer patients on opioid maintenance therapy who are homozygous for the variant G allele of the 118 A > G polymorphism of the mu opioid receptor (OPRM1) gene require higher doses of morphine for efficacious pain control in comparison with patients who are heterozygous.² In fact, the morphine requirement is almost 93% less in AA genotypes in contrast with morphine requirements in cancer patients who carry the GG genotype of the OPRM1 gene.³ Similar pain modulation variation is seen with polymorphisms of the OPRM1 gene and perioperative fentanyl administration.⁴ Furthermore, more profound CNS depressant side effects after morphine administration are noticed in cancer patients with certain polymorphisms of the multidrug resistance-1 gene.⁵

Similarly, the potency of morphine in pain management in cancer patients is influenced and varies greatly with polymorphisms of the catechol-O-methyl transferase gene.⁶ For instance, individuals with the Met/Met genotype of the catechol-O-methyl transferase gene require 63% less morphine in comparison with those who have the Val/Val genotype of the catechol-O-methyl transferase gene.³ More profound central nervous system side effects are seen following morphine administration in cancer patients with single nucleotide polymorphisms in intron 1 of the catechol-O-methyl transferase gene.⁵

The above examples clearly illustrate the variation in adequate pain control with morphine secondary to genetic mutations. Further research is needed to identify other similar gene polymorphisms that may affect opioid requirements in patients being managed with other nonmorphine narcotics.

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