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A review on the effect of CGRP and anti-CGRP monoclonal abs in migraine

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Migraine is a common neurovascular primary headache disorder. CGRP activates the trigeminovascular system, is a 37-amino acid neuropeptide and has two α and B isoforms. In the plasma, the half-life of CGRP is ~ 10 min. The human trigeminal ganglia accounts for up to 50% of all CGRP-immunoreactive neurons. The main pharmacological features of anti-CGRP (CGRP-receptor) mAbs are a large molecular size, a prolonged T1/2, slow distribution and target specificity, inability to cross the blood-brain barrier, regarding to not related to cytochrome P450 isoenzymes resulted a decreased potential for drug-drug interactions and liver toxicity via binding to specific oligosaccharides. The large size, the relatively poor membrane permeability and gastrointestinal degradation of the mAbs mean that they can be administered only parenterally. Binding of CGRP to an antibody reduces the free ligand that is available to interact with the receptor and efficacy is driven by the magnitude and duration of the reduction in free ligand concentration. Fremanezumab comes in subcutaneous prefilled syringes of 225 mg. Galcanezumab comes both in subcutaneous prefilled syringes of 120 mg and autoinjectors of 120 mg. Galcanezumab is effective in preventing episodic cluster headache as well. Erenumab is a fully human anti-CGRP receptor mAb and is available in autoinjectors with monthly subcutaneous doses of 70 or 140 mg. The most common adverse events are injection site reactions (6%); upper respiratory infection (6.4%). The study of eptinezumab is ongoing and has not approved by FDA yet.