INTERNATIONAL MULTIDISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
SJIF 2019: 5.222 2020: 5.552 2021: 5.637 2022:5.479 2023:6.563 2024: 7,805
eISSN :2394-6334 https://www.ijmrd.in/index.php/imjrd Volume 11, issue 03 (2024)

MEDICINES AFFECTING ADRENORECEPTORS

Egamberdiyev Jasurbek Jumanazar o'g'li

Andijan State Medical Institute

Abstract: Adrenoceptors, generally called adrenergic receptors, are a class of G protein-coupled receptors that are centers for some solutions. These receptors are tracked down in various tissues generally through the body and expect an urgent part in the rule of physiological cycles, for instance, beat, circulatory strain, and processing. Drugs that target adrenoceptors can essentially influence these cycles, making them central gadgets in the treatment of different afflictions. In this article we will give information about adrenoceptors and their effects and types.

Keywords: Adrenoceptors, types, receptors, medical diagnose, drugs, chemical substances.

Introduction: During the 1930s, Ahlquist determined that the actions of noradrenaline were mediated by two different types of receptors which he designated alpha and beta. This was an important conceptual breakthrough, since it became clear subsequently that there were several subtypes of each of these types of receptors and it was feasible that drugs might be developed which would either selectively activate or block a single subtype of receptor. Beta-adrenoceptors were named as such because isoprenaline (isoproterenol), a substance which is a potent bronchodilator and which leads to tachycardia and an increase in the force of cardiac contraction, was found to be a very effective tool for activating the heart muscles in vitro without having a significant effect on the heart when it is beating normally in vivo. Subsequent research has shown that isoprenaline is a non-selective agonist which activates both beta1 and beta2-adrenoceptors and it was found that the inotropic, chronotropic, and dromotropic effects of adrenergic stimulation on the heart were all mediated through activation of the myocardial beta1-adrenoceptors.

Adrenoreceptors are activated by the endogenous agonists, adrenaline and noradrenaline, which are the principal hormones of the sympathetic nervous system. These receptors are located on the effector organs and are responsible for the various actions of the sympathetic nervous system. When the adrenal glands are stimulated by the sympathetic nerves, they discharge adrenaline into the bloodstream where it can then activate adrenoreceptors on a variety of organs. The general effect of activation of alpha-adrenoceptors is to promote a sympathetic-like response in the effector organ, since they lead to contraction in most smooth muscle and glands and cause an increase in the metabolic activity of the tissue. Beta-adrenoceptors have an overall effect which is to increase the contractile force of the heart and cause glycogenolysis because the cAMP which is generated by activation of these receptors can lead to increased contractility of the myocardium and release of stored glucose in the form of glycogen from the liver.

Adrenoreceptors and Their Role

Adrenoreceptors are receptors for epinephrine and norepinephrine. Endogenous catecholaminergic agents exert their physiological effects through the interaction with adrenoreceptors. There are 2 major groups of adrenoreceptors, alpha and beta, which in turn have subgroups. Stimulation of alpha adrenoreceptors results in vasoconstriction and relaxation of the gut, stimulation of beta adrenoreceptors results in an increase in cardiac output from the heart and the relaxation of smooth muscle. The density of adrenoreceptors can change according to disease states, such as congestive heart failure, and various medications can modulate receptor activity. Various agonists and antagonists of adrenoreceptors are used in clinical medicine,

particularly in the field of cardiology. Knowledge about the distribution of adrenoreceptors within the body and their physiological effects is helpful in understanding how these medications work and in predicting and explaining their effects. Labetalol is a competitive inhibitor of alpha and beta adrenoreceptors which is used in the treatment of hypertension. Labetalol reduces systemic blood pressure by reducing the peripheral vascular resistance and has no effect on plasma volume. This is because it blocks beta adrenoreceptors which normally mediate the catecholamine-induced increase in cardiac output. Labetalol also blocks the alpha adrenoreceptors causing a short-lasting decrease in blood pressure due to the drop in peripheral resistance. Knowledge that the blood pressure lowering effect is solely due to the reduction in peripheral resistance is helpful because if labetalol is used in a patient with impaired cardiac function and there is a decrease in blood pressure, the drug can be ceased without the fear of precipitating overt cardiac failure.

Importance of Medicines Affecting Adrenoreceptors

Their importance stems from the afferent signals that trigger proper and balanced response to medical need, stressful response, repair, and maintenance of homeostasis. The drugs that affect adrenoceptors are termed sympathomimetic if they enhance adrenergic transmission and sympatholytic if they inhibit adrenergic transmission. Adrenoceptor modulating drugs have been valuable in management of diverse conditions such as hypertension, angina, asthma, nasal congestion, excessive secretions, premature labor, glaucoma, and CNS disorders. Their importance in maintaining physiological and psychological well-being has translated into major pharmaceutical advances and in some cases spurned development of more selective agents, which sometimes have been molecular chimeras from diverse groups.

Importance is also underscored since endogenous (nor)adrenaline is not an ideal drug for the appropriate receptor on all occasions. It's a good general rule of thumb to remember that refined regulation and precision intrinsic to the system itself is always superior to that which can be externally imposed by the drug. This is an encouraging notion for some who wish to see disease management with minimal drug intervention and maximum environmental/behavioral change. However, the fate of medical management for a particular health issue lies in the quality-of-life impact with the juxtaposition between general quality and length of life for the individual concerned. This is to say that it's one thing to cure a condition and another to restore a patient's (or doctors?!) faith in the cure! Disease states almost invariably represent imbalance from normal/a greater defense against a greater attack. For better or worse, the efficacy of a drug is often measured in terms of how quickly it can restore patient to normal/if not more effective function.

Classification of Adrenoreceptor Medicines

For example, congestive heart failure or myocardial damage are associated with an increase in β1 receptor density, and HIV patients often exhibit orthostatic hypotension due to a destruction of sympathetic nerves caused by a viral infection and resulting in the antibodies specific for adrenergic neurotransmitter degrading enzymes. In these cases, selective β adrenoceptor agonist administration and/or inhibition of adrenoceptor degradation may be useful therapeutic strategies, and theoretical understanding of drug to receptor interaction in these cases could predict a more successful therapeutic strategy.

Adreno mimetic drugs are used to treat conditions in which deficiency of sympathetic nervous activity results in pathophysiologic states. Weight is given to α or β selectivity rather than

consideration of the specific receptor subtype, as in general therapeutic measurement of receptor subtype function has not been clearly defined. This is important because many pathologic states are associated with an alteration in receptor subtypes.

Adrenoreceptor medicines, also known as sympathomimetic drugs, can be categorized by the type of adrenoreceptor they activate or inhibit. Drugs that activate adrenoreceptors are called Adreno mimetic. They are classified by the receptor type and specificity on which they act, α or β, and whether they exhibit specificity for a receptor subtype. Drugs having both α and β effects are considered to be non-selective.

Adrenergic medications are an expansive class of prescriptions that tight spot to adrenergic receptors all through the body. These receptors include: alpha-1, alpha-2, beta-1, beta-2, beta-3. Adrenergic medications will tie straightforwardly to at least one of these receptors to instigate different physiologic impacts. A few medications in a roundabout way act at these receptors to prompt specific impacts.

Adrenergic medications should be arranged in light of the particular receptors they tie. Direct-acting medications, which are the essential focal point of this article, incorporate vasopressors, bronchodilators, and different medications. Instances of roundabout medications are amphetamines and cocaine.

The significant impacts of agonist restricting rely upon their adrenergic receptors. The general classifications are:

Alpha-1 receptor: Smooth muscle compression, mydriasis

Alpha-2 receptor: Blended smooth muscle impacts

Beta-1 receptor: Expanded cardiovascular chronotropic and inotropic impacts

Beta-2 receptor: Bronchodilation

Beta-3 receptor: Expanded lipolysis

Instances of adrenergic medications which specifically tie to alpha-1 receptors are phenylephrine and oxymetazoline. Specific alpha-2 receptor drugs incorporate methyldopa and clonidine. The key beta-1 specific medication is dobutamine. In conclusion, beta-2 particular medications are bronchodilators, like albuterol and salmeterol.

Adrenergic medications can likewise be non-particular and tie to a blend of adrenergic receptors. Norepinephrine ties to the alpha-1, alpha-2, and beta-1 receptors. Dopamine ties to the alpha-1, alpha-2, beta-1 receptors, and furthermore dopamine receptors. Epinephrine ties to the adrenergic receptors in general. These medications tie to additional adrenergic receptors when controlled at higher portions, i.e., they can lose selectivity.

Coming up next are key clinical signs of different adrenergic medications:

Specific Medications

Alpha-1 Receptor Agonists

Phenylephrine: FDA-supported as a decongestant and vasopressor. It has utility in instances of hypotension because of shock, for example, septic shock.[6] Olson C. et al. provided details regarding a patient who created ischemic priapism from high portions of hydroxyzine

hydrochloride (200 to 600 mg) for sleep deprivation treatment. The patient got treatment with yearning and 560 micrograms of intracorneal phenylephrine, which prompted detumescence.

Oxymetazoline: FDA-supported as a decongestant and to treat rosacea. A patient with ptosis of the right eye brought about by myasthenia gravis was treated with one eye drop of oxymetazoline (0.1%). The ptosis was dispensed with following 2 hours, and the impact endured seven hours. This is an expected clinical utilization of oxymetazoline.

Alpha-2 Receptor Agonists

Methyldopa: FDA-endorsed for hypertension and gestational hypertension.

Clonidine: FDA-endorsed for treating hypertension and consideration shortage hyperactivity jumble (ADHD). Non-FDA-supported signs incorporate rest problems, post-awful pressure issue (PTSD), uneasiness, a propensity to fidget, hot glimmers related with menopause, and different diseases.

Dexmedetomidine: This medication is shown for sedation in the emergency unit doesn't cause respiratory melancholy. It causes an arousable condition of sedation which is useful during cerebrum growth medical procedure and implantation of profound mind triggers in light of the fact that the patient can answer neurological tests.

Beta-1 Receptor Agonists

Dobutamine: Demonstrated for the treatment of cardiogenic shock and cardiovascular breakdown. Patients in shock need hemodynamic help. Vasopressors (e.g., norepinephrine), inotropes (e.g., dobutamine), and IV liquid help are utilized for treatment. In many patients with shock (e.g., cardiogenic or septic), norepinephrine is usually used to accomplish fitting blood vessel pressure. If the patient keeps on having low tissue and organ perfusion, dobutamine can be added to increment heart yield.

Beta-2 Receptor Agonists

Bronchodilators: Showed for treating obstructive lung illness, like asthma.

Beta-3 receptor Agonists

Mirabegron: This specialist is demonstrated for treating overactive bladder, e.g., urinary incontinence and recurrence. It is likewise shown to treat pediatric (more prominent or equivalent to 3 years of age) neurogenic detrusor overactivity.

Conclusion

All in all, drugs that target adrenoceptors assume an essential part in the therapy of different ailments. By understanding the different subtypes of adrenoceptors and how drugs cooperate with them, medical services suppliers can actually deal with these circumstances and work on persistent results. In any case, it is critical to painstakingly consider the possible results of these prescriptions and screen patients near guarantee their security and prosperity.

References:

1. Giovannetti JA, Thoms SM, Crawford JJ. Alpha-2 adrenergic receptor agonists: a review of current clinical applications. Anish Prog. 2015 Spring;62(1):31-9.

2. Ciccarone D. Stimulant abuse: pharmacology, cocaine, methamphetamine, treatment, attempts at pharmacotherapy. Prim Care. 2011 Mar;38(1):41-58.
3. Piascik MT, Perez DM. Alpha1-adrenergic receptors: new insights and directions. J Pharmacal Exp Ther. 2001 Aug;298(2):403-10.
4. Madden CJ, Tupuna D, Canoe G, Morrison SF. α2 Adrenergic receptor-mediated inhibition of thermogenesis. J Neurosis. 2013 Jan 30;33(5):2017-28.
5. Wachter SB, Gilbert EM. Beta-adrenergic receptors, from their discovery and characterization through their manipulation to beneficial clinical application. Cardiology. 2012;122(2):104-12.
6. Hatton RC, Winterstein AG, McKelvey RP, Shuster J, Hendel's L. Efficacy and safety of oral phenylephrine: systematic review and meta-analysis. Ann Pharmacotherapy. 2007 Mar;41(3):381-90.
7. Olson C, Jhawar A, Ellesse Z, Doyle R. Hydroxyzine-induced priapism. Am J Emerg Med. 2021 Oct; 48:375. e5-375.e6.