Eicosanoids/Questions and case reports

Tip: Expand the author's answers!

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Tip: Expand the author's answers!

Questions

1. **What is the difference in the effect of aspirin and nonsteroidal anti-inflammatory drugs of the indomethacin type on cyclooxygenase activity?**
2. '*Some asthmatics experience an exacerbation of their symptoms when they take non-steroidal anti-inflammatory drugs. How can this be explained from a biochemical point of view?*
3. **Which pair has the opposite effect?**
   - A – cholic and lithocholic acid
   - B – 5-HPETE and leukotriene D4
   - C – Lactosylceramide and galactocerebroside
   - D – Thromboxane A2 and prostacyclin (PGI2)
   - E – Acetone and 3-hydroxybutyrate

Answers

Question 1.

- Aspirin blocks cyclooxygenase activity irreversibly by acetylating the enzyme molecule. This effect will disappear only with the disappearance of thrombocytes, i.e. in 7-10 days. Indomethacin only temporarily blocks receptors for prostaglandins (PGE, PGD and PGI series)

Question 2.

- NSAIDs inhibit cyclooxygenase but not lipoxygenase. Metabolism of arachidonic acid cannot continue in the presence of non-steroidal anti-inflammatory drugs via the cyclooxygenase pathway. Therefore, there is more substrate for the lipoxygenase pathway. This creates more leukotrienes, which induce bronchoconstriction. The bronchoconstrictive effect prevails over the bronchodilating effect of prostaglandins and thromboxane.

Question 3.

- A – wrong
- B – wrong
- C – wrong
- D – correct – thromboxane formed in platelets causes arteries to contract and induce platelet aggregation; prostacyclin has exactly the opposite effect
- E – wrong

Case reports

Patient with asthma

A woman, 46 years old, was treated on an outpatient basis for asthmoid bronchitis by triamcinolone acetonide inhalation. Before being admitted to the hospital, she had an upper respiratory tract infection, and her breathing difficulties significantly worsened, turning into an acute asthma attack with significant bronchospasm. glucocorticoids i.v.

Questions:

1. **What is the mechanism of action of glucocorticoids in the treatment of asthma?**

Answers

1. Glucocorticoids suppress some manifestations of the inflammatory reaction that induces bronchospasm and thus leads to an acute asthma attack. They inhibit the entry of leukocytes and monocytes/macrophages into areas affected by inflammation, thus reducing the production of some chemotactic and other substances, especially eicosanoids, which mediate an adverse reaction. Glucocorticoids support the synthesis of e.g. lipocortins and macrocortins, which inhibit the activity of phospholipase A_2. The consequence is that the production of pro-inflammatory prostaglandins and leukotrienes is suppressed.

Cardiovascular disease patient

A man with ischemic heart disease takes small doses of aspirin as a preventative measure.

Questions:

1. **What is the pathobiochemical mechanism of the beneficial effect of aspirin on the occurrence of acute myocardial infarction?**
2. **What adverse effect can the administration of acetylsalicylic acid have in some patients?**

Answers

1. Aspirin (acetylsalicylic acid) irreversibly inactivates cyclooxygenase by acetylating it. This blocks the formation of thromboxane A_2 from arachidonic acid. Thromboxane is a powerful vasoconstrictor and stimulant of thrombocyte aggregation (i.e. mechanisms that can significantly narrow or even block a coronary artery → acute myocardial infarction). Inactivation of cyclooxygenase is irreversible, and because mature platelets cannot make new proteins, inactivation takes 7–10 days, when a generation of new platelets is formed.
2. Long-term administration of aspirin can lead to the development of an acute stomach ulcer in some patients. By reducing the formation of prostaglandins, the dampening effect on the secretion of acid gastric juice induced by gastrin is removed.

Links

Related Articles

- Prostaglandins
- Prostaglandin E1
- Thromboxane A2
- Nonsteroidal antiphlogistics

Template:Pathobiochemistry of metabolic pathways (Masopust)

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