

Disorders of uric acid metabolism / Questions and case reports

Questions

1. In humans, carbamoyl phosphate is a precursor of biosynthesis:
 - A – uridine monophosphate
 - B – Inosine monophosphate
 - C – urea
 - D – glutamine
2. vitamin B₁₂ metabolites play a role in
 - A – Catabolism of fatty acids with an odd number of carbon atoms
 - B – In the formation of acetyl-CoA from pyruvate
 - C – In the transfer of the CH₃- group from the tetrahydrofolate coenzyme to homocysteine
 - D – In the synthesis of palmitate
3. All of the following statements relating to purine nucleotide biosynthesis are correct except:
 - A – PRPP is a substrate in this metabolic pathway
 - B – Glutamine forms 2 nitrogen atoms of the purine cycle
 - C – Formation of N-glycosidic bond only after completion of the base structure
 - D – Folate cofactors are involved in the carbons of the purine cycle
 - E – Inosine monophosphate is a precursor of both AMP and GMP.
4. Gout is caused by an excessive increase in the concentration of uric acid in the blood. The cause can be both overproduction and insufficient excretion. A 15N-labeled amino acid can be administered to recognize this situation. Which is best for this purpose?

Answers

Case reports

A patient being treated for acute leukaemia

A 3-year-old girl is diagnosed with acute lymphocytic leukaemia. She received an infusion, allopurinol, day 2 therapy vincristine, prednisone, methotrexate, etc. In 5 days released home. She continued therapy at home (prednisone, allopurinol. Chemotherapy added again in a month. Then she got soor (candidosis) in her mouth, she couldn't eat.

Laboratory results (gradually during the month):

S-urea	4,0	5,0	1,3	0,7 (mmol/l)		
S-creatinine	62	88	62	62 (μmol/l)		
S-uric acid	714	547	238	113	137	184 (μmol/l)
white blood cells	56 300	3 700	2 800	3 700 (no./ml in blood)		

Questions:

1. How do you explain the high level of uric acid (1st examination performed after 5 days of hospitalization, after discharge)?
2. Why was uric acid already normal in future examinations?
3. Why was the urea level 0.7 mmol / l?
4. What other tests will confirm this finding?

Answers

References

Related articles

- Disorders of ureagenesis
- Antiuratics
- Arthritis uratica

Source

- MASOPUST, Jaroslav – PRŮŠA, Richard. *Patobiochemie metabolických drah*. 1. edition. Praha : Univerzita

Karlova, 1999. 182 pp. pp. 113- 114. ISBN 80-238-4589-6.