

# Disorders of uric acid metabolism/Questions and case studies

## Questions

1. In humans, carbamoyl phosphate is a precursor for the biosynthesis of:
  - A – uridine monophosphate
  - B – Inosine monophosphate
  - C – urea
  - D – glutamine
2. The metabolites of vitamin B<sub>12</sub> 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 – During the transfer of the CH<sub>3</sub>- group from tetrahydrofolate coenzyme to homocysteine
  - D – In the synthesis of palmitate
3. All of the statements below regarding purine biosynthesis nucleotides are correct except:
  - A – PRPP is a substrate in this metabolic pathway
  - B – 2 nitrogen atoms of the purine cycle are formed from glutamine
  - C – Formation of N-glycosidic bond only after completion of base structure
  - D – Folate cofactors are involved in the carbons of the purine cycle
  - E – Inosine monophosphate is a precursor to 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. To recognize this situation, <sup>15</sup>N-labeled amino acid can be administered. Which one is best for this purpose?

## Answers

## Case reports

### Female patient treated for acute leukemia

A 3-year-old girl was admitted with a diagnosis of acute lymphocytic leukemia. She received IVs, allopurinol, 2nd day of vincristine therapy, prednisone, methotrexate, etc. Discharged home in 5 days. She continued therapy at home (prednisone, allopurinol). Added chemotherapy again in a month. Then she got thrush 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 (count/ml of blood)		

### Questions:

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

## Answers

## Links

### Related articles

- Ureagenesis disorders
- 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.

