NEPHROTIC SYNDROME ASSOCIATED WITH THE TREATMENT OF TOXOPLASMOSIS IN A PREGNANT WOMAN: A CASE REPORT

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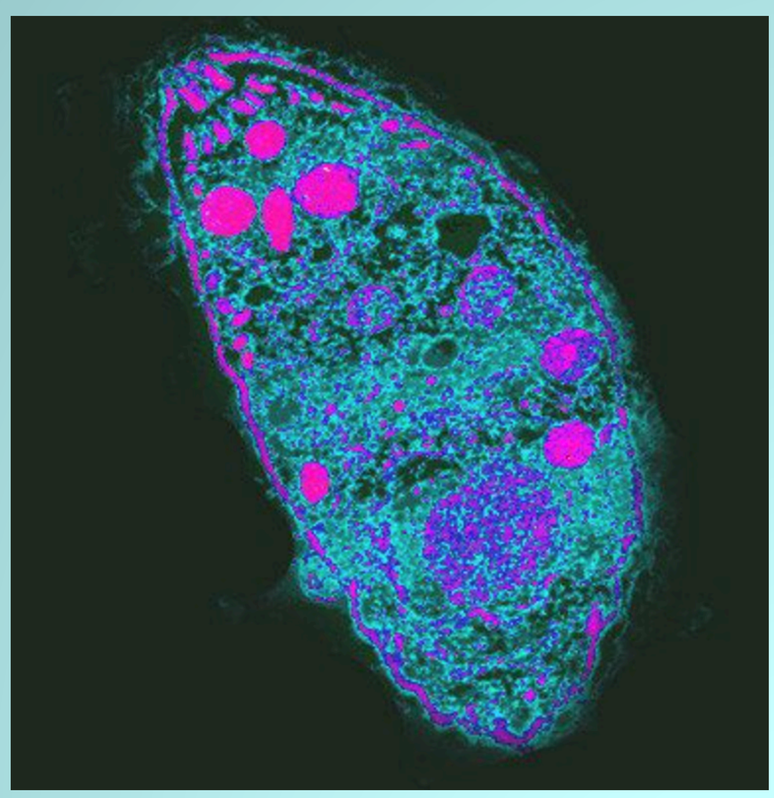
INTRODUCTION

Proteinuria in pregnancy is common and can achieve nephrotic range. In early pregnancy, nephrotic proteinuria with hypertension suggests intrinsic renal disease rather than pre-eclampsia. Nephrotic syndrome in pregnancy is rare, the causes being preeclampsia, glomerulonephritis, diabetes, renal vein thrombosis, amyloidosis, hereditary nephritis, drugs and infections. Parasitic infections as toxoplasmosis can also be associated with nephrotic syndrome. In general nephrotic syndrome in pregnancy causes few problems, if there is no hypertension or abnormal renal function.

Placental transmission of Toxoplasma gondii (picture 1) results in congenital toxoplasmosis, which may lead to fetopathy, hydrocephalus, and death. The brain and retina are frequently affected.

Therefore prompt diagnosis and proper management are crucial for maternal and fetal health.

We present a case of nephrotic syndrome in a pregnant woman, associated with the treatment of toxoplasmosis.



Picture 1. Toxoplasma gondii.

CONCLUSIONS

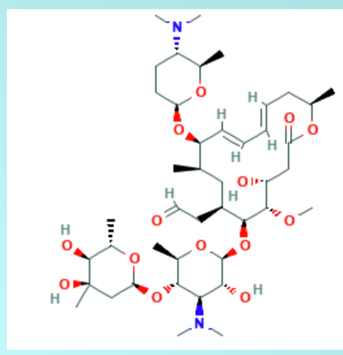
Nephrotic syndrome in primigravid with no previous history of kidney disease, spontaneously resolved after discontinuation of spiramycin and pregnancy termination. Renal biopsy and steroids were not indicated. It is known, that spiramycin can be associated with acute interstitial nephritis, but to our knowledge there are no reports about the association with nephrotic syndrome.

CASE

A 23-year-old healthy primigravid was hospitalized in 33rd week of gestation because of oedema and proteinuria. On the first gynaecologist visit, there was no proteinuria present. Since the serology results raised the suspition of acute toxoplasmosis, spiramycin (picture 2) was started in the 9th week of gestation with the aim of reducing the risk of mother-to-child transmission. Approximately one month before the conception she also noticed bilaterally enlarged cervical lymph nodes.

During the pregnancy proteinuria gradually developed and in 32rd week of gestation she also noticed lower limb oedema. She was normotensive. Laboratory investigations upon admission showed urea 2,4 mmol/l, creatinine 46 µmol/l, albumin 24 g/l, haemogram and haepatogram were within normal limits, cholesterol 14,7 mmol/L, HDL-cholesterol 3,0 mmol/L, LDL-cholesterol 11,1 mmol/L, triglycerides 5,4 mmol/L. Urine sediment was normal, 24 hour proteinuria was 7,42 g. Imunoserology was negative. Since PCR for Toxoplasma gondii was negative, spiramycin was discontinued. Preeclampsia was ruled out, the condition of fetus was stable.

She delivered a healthy 4500 g baby in 38th week of gestation. Six weeks after the labour the 24 hour proteinuria has fallen to 0,88 g and serum albumin was 43 g/L. Six months after the labour the 24 hour proteinuria was estimated to 0,165 g/day/1,73m2.



Picture 2. Spiramycin, a macrolide.

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