# A case of nephrotic syndrome associated with hydatiform mole

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## Abstract

The present case study is on a 16-year-old woman who was suffering from nephrotic syndrome after recovery from complete type of hydatiform mole. She was admitted in hospital because of proteinurea and hematuria. Then she was showing a generalized edema compatible with neprhotic syndrome. In her past medical history she had a suction curettage for hydatiform mole. After she received 4 courses chemotherapy, she completely recovered and βhCG has fallen from 12127 IU/L to under 10 IU/mL. Then she showed generalized edema, proteinurea and hematuria compatible with nephritic syndrome. After six courses chemotherapy the symptoms of nephrotic syndrome and invasive mole diminished, she released from hospital and scheduled for follow-up.

## Introduction

During normal pregnancy, the maximum of urinary protein excretion ranges from 200-300 mg per day. Nephrotic syndrome in pregnancy is very rare.[1] The most common cause is preeclampsia associated with preeclamptic nephropathy. Preeclampsia may have a relation to the molar pregnancy. Twelve percent of molar pregnancies are associated with preeclampsia.[2] We report a case of nephrotic syndrome associated with complete type of hydatiform mole.

## Case Report

### Diagnosis and treatment of the hydatiform mole

A 16-year-old Iranian woman, gravid 1, para 1 was admitted to the Educational hospital of Razi in gynecologic section because of molar pregnancy in 4/2/2010. In admission time uterine size was 16 weeks of pregnancy, uterine sonogram showed enlarged uterus contained 400 mL cystic tissue compatible with molar pregnancy or missed abortion.

Suction curettage was done and vesicular tissue has sent for pathologic study. Pathology result revealed molar pregnancy (6/2/2010) (Figure 1). At that time laboratory investigation showed: βhCG: 1980 IU/mL, total blood count showed; hemoglobin (Hb): 10.8 g/dL, white blood cell (WBC): $10.8\times10^9$/L, platelets: $247\times10^9$/L, blood glucose: 82 mg/dL, liver function and thyroid function tests were normal. Two days after suction curettage the patient was discharged and scheduled for follow-up of molar pregnancy (weekly measurement of βhCG).

### Diagnosis and treatment of the nephrotic syndrome

About two months later in 28/3/2010 the patient admitted in the nephrology ward in the educational hospital of Imam Khomaini because of generalized edema (she did not measure βhCG in a regular time). On examination blood pressure was 120/60 mmHg with a regular heart rate of 80 per minute, a paratibia pitting edema (2+) was noted. The

**Table 1. Paraclinical tests in the patient suspected to the nephrotic syndrome.**

| Tests | Results |
|---|---|
| Urinalysis | |
| Proteinuria | 4+ |
| Red blood cell in high power fields | 10-12 |
| Hemoglobin | 1+ |
| WBC in high power field | 8-10 |
| 24 urinary protein exertion | 9400 mg |
| Liver function tests | |
| SGOT | 24 µg/L |
| SGPT | 22 µg/L |
| Bilirubin | 0.8 mg/dL |
| Partial thromboplastin time | 30 sec |
| Prothrombin activity | 81% |
| Total blood count test | |
| Hemoglobin | 13 g/dL |
| WBC $\times1000/mm^3$ | 11.6 |
| Platelets $\times1000/mm^3$ | 270 |
| Blood urea nitrogen | 7 mg/dL |
| Creatinin | 0.7 mg/dL |
| Sodium | 139 µg/L |
| Potassium | 4.2 µg/L |
| Calcium | 8/9 mg/dL |
| Phosphorous | 4.6 mg/dL |

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Key words: nephrotic syndrome, hydatiform mole, chemotherapy.

Contributions: RM was the doctor in charge for the patient in the study; SB was a nephrologist who has consulted about patient; TH was an assistant of Dr Mohammadjafari who was responsible for the hospital daily care for the patient; TR is a gynecologist and oncologist who has consulted about patient; PA was responsible for gathering information and writing the paper in English.

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**Table 2. Paraclinical tests in the patient suspected to the nephrotic syndrome.**

| Blood tests for lipid and antibodies | Results |
|---|---|
| Total cholesterol | 322 mg/dL |
| Triglycerids | 393 mg/dL |
| High density lipoprotein | 33 mg/dL |
| Low density lipoprotein | 270 mg/dL |
| Very low density lipoprotein | 18 mg/dL |
| International ratio | 1.1 |
| Erythrocyte sedimentation rate | 52 mm/hr |
| C-reactive protein | 3+ |
| Complement component 3 | 150 mg/dL (86-184 mg/dL) |
| Complement component | 20.3 mg/dL (20-57 mg/dL) |
| The dose of complement that lyses 50% of a red cell suspension | 94 U/mL (63-184 U/mL) |
| Glomerular basement membrane | Negative |
| Antinuclear antibody test | 1/40 |
| Protoplasmic-staining anti-neutrophil cytoplasmic antibodies | <1.4 U mL |
| Classical antineutrophil cytoplasmic antibodies | Negative (normal <2.8 U/mL) |
| Antids-DNA | 1/10 |
| Antiphospholipid (immunoglobolin G antibodies) | 5.1 mg/dL |
| Immuunoglobolin M antibodies | 3 mg/dL (0-15 mpl) |

results of paraclinical tests are presented in Table 1 and 2. Pelvic sonogram was normal. In ultrasound scan the size of the kidneys was 111 mm with normal echo texture. The patient did not get consent for kidney biopsy.

Treatment started with oral prednisolon 50 mg, oral calcium daily, omperazol cap 20 mg/day, frusemide 40 mg daily. The low salt diet and restriction of fluid have chosen for her. In respect to past medical history, gynecology consultation has done and she referred to gynecologic section.

The urinalysis showed; proteinuria (3+), 10-12 red blood cells in high power field, WBC 30-35. βhCG titer raised to 12127 U/mL (21/3/2010), nephrotic syndrome associated with invasive mole was suggested and chemotherapy was started at (25/3/2010) with methotrexate (MTX). After she took six courses of chemotherapy, βhCG decreased to the normal range. The process of reduction of βhCG is demonstrated in Table 3. After chemotherapy, 24 h urinary protein exertion decreased from 9400 mg to 380 mg. At this time pelvic ultrasound scan was normal. Six weeks after treatment the patient was well enough to discharge from hospital and schedule for follow-up.

**Table 3. Reduction of βhCG after chemotherapy.**

| Weeks of treatment | βhCG (U/mL) |
|---|---|
| In time of diagnosis of nephrotic syndrome | 12127 |
| 1st week after chemotherapy | 17124 |
| 2nd week after chemotherapy | 4370 |
| 3rd week after chemotherapy | 687 |
| 4th week after chemotherapy | 67 |
| 5th week after chemotherapy | <10 |
| 6th week after chemotherapy | <10 |

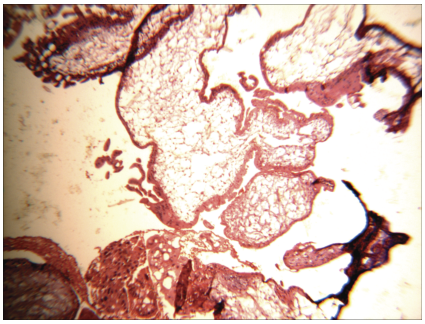

**Figure 1. Photomicrograph of complete mole; multiple large villi show stromal edema and marked trophoblastic proliferation.**

## Discussion

In this young patient with generalized edema, history of hydatiform mole and high βhCG, treatment with chemotherapy was started. There was no evidence of recurrence or metastasis of mole and she remained in complete remission of nephrotic syndrome after chemotherapy. Nephrotic syndrome occurs in 0.012-0.025% of all pregnancies.[3] The usual causes are preeclampsia, glomerulonephritis, diabetes, renal vein thrombosis, amyloidosis and hereditary nephritis. Occasionally it is necessary to treat the nephrotic syndrome with steroids. There is no proper response to steroids which can aggravate the problems related to nephrotic syndrome. Thus, it is important to know about histology before starting treatment.[2] Urinary protein excretion 200-300 mg per day is normal during pregnancy.[4] Preeclamptic nephropathy is about 80% in nephrotic syndrome during pregnancy. Other cases occur because of membranous nephropathy, focal glumerulosclerosis, minimal change nephropathy, diabetic nephropathy, systemic lupus erythematosus and other renal diseases.[4] The renal pathologic feature in preeclamptic nephropathy is bloodless glomerular enlargement and the narrowing the capillary lumen due to swelling of the endothelial, mesential and epithelial cells with an expansion of the mesential matrix. The glomerular capillary walls may be thickened but hypercellular change rarely occurs.[5-9] Akhtars case was a preeclamptic nephropathy associated with a partial mole with a coexistent fetus.[3] In the Cohen's case they did not performed renal biopsy but the nephritic syndrome was clinically related to a preeclamptic nephropathy. In this case, the hydatiform mole was incomplete type coexisting fetal tissue.[10] Komatsuda reported an older patient revealed a membrano proliferative like lesion by renal biopsy. His case was a nephrotic syndrome associated with a complete type of hydatiform mole.[11] Prior to this report, there was a similar case reported in Korean journal.[12] Han reported a 54-year old patient with membrano proliferative glumeronephritis associated with a complete type of hydatiform mole that patient remained renal symptom free for 2 year after the removal of the tumor.[5] In our case, the hydatiform mole was a complete type and renal biopsy was not performed. The precise relationship between the hyditiform mole and nephrotic syndrome is not clear, because the reported cases were extremely rare. The production of immune complexes and the activation of intravascular coagulation by the hydatiform mole are the supposed pathogenic mechanism.[3] These several interesting cases linking the pathogenesis of the glomerulonephritis directly to the gestational trophoblastic disease provide a challenge for future research.

## Conclusions

The hydatiform mole might be a cause of the nephrotic syndrome in some cases. Precise follow-up after molar pregnancy may help the specialists for early reorganization of rare situations.

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