

PREGNANCY IN RENAL FAILURE

by

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Renal failure can occur due to previous obstetric complications or renal pathology. Four such cases with previous renal failure are presented in whom pregnancy was continued under strict supervision. The pregnancy outcome was gratifying but control of hypertension in 2 cases was exasperating.

The assessment of renal function in these patients during pregnancy is difficult and mostly a clinical approach was helpful.

These patients are prone to develop toxæmia of pregnancy and also intra-uterine growth retardation. Hence they need a long duration of hospitalization.

A critical study of the following four cases could give a lead to this important question of pregnancy in patients with previous renal failure.

CASE REPORTS

Case 1

Mrs. B., 25 years, was diagnosed to have renal hypertension following chronic nephritis at the age of 17. Her hypertension was controlled with antihypertensive drugs (methyl-dopa and hythaltan). She married at the age of 23, and discontinued the therapy for a few months.

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She came to the hospital for a check-up only during the early weeks of pregnancy.

On admission the B.P. was 180/110 mm Hg., and lying down 160/100 mm Hg. Decision was made to continue the pregnancy and therapy was started with methyl dopa 1-1-1 and hythaltan.

Investigations: Hb—8.25 g., blood urea—32 mg. Creatinine—1.6 mg., Total Proteins 4.7 g.

Alb: 2.4, Glb 2.3, Uric acid—6.4 mg.

Urinary proteins—1.2 G/24 hours. E.C.G.—LAD.

Fundus: Grade I Hypertensive retinopathy.

She was maintained on methyl dopa 1-1-1 and hythaltan throughout pregnancy. Blood pressure was maintained at 160/100 mm Hg. Foetal monitoring was carried out with weekly urinary oestrol levels.

Induction of labour was done at 36 weeks of gestation as there was a drop in the urinary oestrial level. Blood pressure during labour rose to 210-200/110-112 mm Hg. which was easily controlled with pethidine 100 mg., and chlorpromazine 50 mg. Lower segment caesarean section was done for foetal distress and a live male child weighing 2400 G. was delivered. At discharge the dosage of antihypertensive drugs were reduced. Mother and child are well. Followed up for one year.

Case 2

Mrs. S. P. 22 years, gravida 3, para 0 was admitted at the 16th week of pregnancy with hypertension. During the first pregnancy at 20-24 weeks of gestation she had renal failure following severe toxæmia of pregnancy and papilloedema, which was treated conservatively. Intrauterine death of the foetus resulted.

The second pregnancy was terminated at 10 weeks of gestation as the blood pressure was

210/130 mm. Hg., with Grade 1 retinopathy and also to investigate the cause of hypertension. The hypertension was controlled with hypotensive drugs.

However, the patient was irregular in her anti-hypertensive treatment and came only for check-up during the early weeks of her third pregnancy when she developed pyrexia due to urinary infection. She was adequately treated and pregnancy was continued along with anti-hypertensive drug therapy.

Induction of labour was done at 38 weeks of gestation and she delivered a live baby weighing 2450 grams. In spite of antihypertensive drug cover her blood pressure rose up to 200/120 mm. Hg., during labour, which was controlled with pethidine 100 mg. and Chlorpromazine 50 mg. Mother and child are doing well.

Case 3

Mrs. S., 19 years, gravida 3, para 0, Obstetric history.

1st pregnancy: Spontaneous abortion at 2½ months, 2nd pregnancy: accidental haemorrhage at 7 months severe haemorrhage. Anuria for 2 days. Brought to hospital (28-9-1975). On examination extremely pale with a blood pressure of 160/100 mm. Hg.

Investigations: Hb 4.25 G., Blood urea, 182 mg., Creatinine—2.1 mg., Uric acid—5.2 mg., Urine—Alb. ++, RBC, E.C.G.: Sinus tachycardia.

Conservative treatment: Mannitol, Sodab carbonate, fluid and diet restriction. Improved. At discharge blood pressure was 130/80 mm. Hg., Hb 9.5 G., B. Urea—35 mg., S. Creatinine—1.2 mg., K⁺ — 4.5 mEq., Urinary output good. No albuminuria.

3rd pregnancy: She came early in pregnancy when the renal function were within normal limits. Blood pressure was 120/80 mm. Hg., Hb—10 G.; B. Urea—20 mg., Uric acid—5.2 mg., Regular antenatal check-up was given and the pregnancy was uneventful.

Induction of labour was done at 38 weeks of gestation and a male baby of 2780 g. was delivered by outlet forceps. Puerperium was normal. Mother and child doing well.

Case 4

Mrs. H., 19 years, gravida 3, para 3, was admitted to the hospital 1 week after her first full normal delivery at home as she developed

puerperal pyrexia for 5 days and anuria of 1 day duration. She had a temperature of 40°C and passed a few ml. of urine, after 500 ml. of glucose infusion. She was anaemic but conscious, with a blood pressure of 160/110 mm. Hg.

Investigation: Blood Urea—158 mg., S. Creatinine—1.8 mg. Hb—8 G., Urine—Alb. +++, RBC. ++, Blood culture—Pseudo monous seroginosa++.

D & C was done under local analgesia on 2nd day after admission as there was bleeding and few bits of retained placenta were removed. Following this, patient developed convulsions and became semiconscious. Blood pressure was 160/110 mm. Hg. with B. Urea of 332 mg. K⁺ 5.1 mEq., C.S.F.—WBC. ++, E.E.G.: Abnormal diffuse changes both hemispheres. Fundus Retinopathy Grade I. Diagnosis—Uraemic convulsions.

She improved with conservative treatment. On discharge. Normal urinary output. Blood pressure was 150/90 mm Hg., B. Urea—15 mg. S. Creatinine—1.2 mg. IVP—normal.

The second pregnancy was uneventful except that she developed mild pre-eclamptic toxemia. She was admitted and labour induced at 38 weeks. Investigations: S. Creatinine—0.66 mg., K⁺ 4.0 mEq. Hb—11 G., B. Urea—12 mg. Blood pressure during labour was 160/110 mm. Hg., which was easily controlled with pethidine and Chlorpromazine. Mother and child well. Puerperal sterilization done under local anaesthesia. On discharge: B. Urea—35 mg., S. Creatinine—0.8 mg., Hb—11 G.

1 year later she sought recanalization operation as second child died. She had an uneventful third pregnancy. Mother and child did well.

Discussion

In the past renal failure was a contra-indication for pregnancy (Browne and McLure-Browne, 1955). But in recent years, an increased understanding of the renal function in renal disorders, improved diagnostic techniques and advances in the management of renal failure have led to a rethinking in the management of such patients (Knapp and Burden, 1977). However McGeown even as recent as 1977, is of the opinion that renal disease

not only affects the outcome of pregnancy but may even threaten the patient's life.

Thus there is still a certain measure of uncertainty as to whether pregnancy should be continued or not in patients with previous renal failure.

Analysis of the 4 cases presented here, the first 2 cases (Case 1 and 2) the renal failure was due to pathology unrelated to obstetric causes while in the latter 2 (Case 3 and 4) the cause could be directly attributable to pregnancy.

In this series, Case 1 and 2 had renal dysfunction due to renal pathology, exhibiting proteinuria and or arterial hypertension requiring therapy. In Case 1 the serum albumin was quite low, the urinary loss being at the rate of 1.2 G. per 24 hours. These were corrected.

The most trying aspect in the management of the first 2 cases (Case 1 and 2) was the control of hypertension.

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