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Pregnancy Related Renal Failure

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SUMMARY

Six cases with pregnancy related renal failure are presented. All these patients except one presented with abortion after 28 weeks and with retention of fetus for variable periods. In five cases three had post partum haemorrhage and in one case there was severe infection. All these patients underwent peritoneal dialysis alongwith conservative management of renal failure.

Three patients out of six had improvement in their kidney function after 8-12 weeks, three patients had not shown any recovery in the renal function and are on chronic peritoneal dialysis even after five months. Thus incidence of irreversible renal failure in our small number of patient is higher than 10-20% reported in the literature. This is probably due to the fact that these patients developed acute cortical necrosis because of massive heamorrhage, prolonged intrauterine death and septicemia.

INTRODUCTION

Renal failure associated with pregnancy and parturition can occur with the underlying risk factors or without the underlying risk factors. The underlying risk factors can be shock, trauma, ischemic insult, sepsis, antibiotics, analgesic, heavy metals, solvent, radio contrast media, septic abortion, intrauterine fetal death, ergotamine compounds, oxytocic agent and oral contraceptive[1]. This can also occur within 48 hours to 10 weeks after apparently normal pregnancy. Different names have been given to it including postpartum renal failures, late postpartum intravascular coagulation with ARF and postpartum Haemolytic Uremic syndrome[2]. This syndrome is regarded as distinct from acute renal failure occurring in third trimester of pregnancy or 48hrs of delivery secondary to obstetric complications. The term of idiopathic postpartum ARF was first recognized by Robson et al in 1968[2]. Oliguria, anuria, rapidly developing azotemia, microangiopathic haemolytic anemia are usual presenting features of this disease process.

CASE REPORT

We present our experience with 6 patients who were admitted at Shaikh Zayed Hospital, with pregnancy related acute renal failure. Clinical features of these patients are shown in Table-1 and laboratory reports in Table-2a & 2b.

1. A.B. 35 years old multipara,G7, Para 5, A2 had still birth at 28 weeks associated with severe postpartum bleeding. The intervention was carried out by a dai and complete evacuation of placenta was done in the hospital. She developed severe bleeding, high grade fever and oliguria. Following these symptoms the patient was admitted in another hospital, where acute peritoneal dialysis(APD) was done but neither oliguria nor renal function improved and further deterioration occurred. She was shifted to Shaikh Zayad Hospital where APD was done again. The renal function on admission stabilized but oliguria presisted even after 10 days. At that time she was put on eontinous ambulatory peritoneal dialysis(CAPD) and was discharged from the hospital. She is presently on CAPD and the renal function as well as urine out put has improved very little.
2. N.B., 35 years multipara, G3, Para2, A1, with history of home delivery, 10 days prior to admission, which was done by a dai complete evacuation of placenta was done in the hospital. She developed fits and became drowsy 2 days prior to admission she became deeplly comatosed 12 hours prior to admission. She also had low grade fever and oliguria. The renal function on admission showed raised Bun and creatinine. Patient was admitted in ICU and APD was done but no improvement was noticed. On the 5th day

Table-1

Name	Age	Past Obsetetric	Present Pregnancy	Peri Partum	Associated Risk	Urine Output
Azra Bibi	35	G.7 Para.5, A2.	Intra uterine death after 28 weeks	Intervention done by a Dai and followed by eli bration of rest of placenta in the hospital.	Post partum bleeding	< 400 cc in 24 hours.
Nazira Bibi	35	G.3 Para.2, A2.	Intra uterine death after 28 weeks.	Intervention done by a Dai and followed by evacuation in hospital.	Post partum haemorrhage associated with retained placental tissue.	< 100 cc in 24 hours.
Khurshid Bibi	35	G.11, Para 10, A1.	Abortion was done by manipulation on the 54 days of the LMP appeared.	Intervention was done by a Dai followed by evacuation in hospital.	Severe haemorrhage after abortion.	1300 cc in 24 hours.
Andaz Begum	30	G3, Para 2, A1.	Still birth after 28 weeks.	No intervention done after primary intervention in the hospital.	Severe infection. No haemorrhage.	< 100 cc in 24 hours.
Sabira Bibi	32	G6, Para 4, A2.	Still birth after 28 weeks.	Done by Dai. placental debris present on intervention in the hospital.	Severe Pre/Post partum haemorrhage.	< 400 cc in 24 hours
Shagufta Bibi		G1, Para 0, A1.	Abortion after 17 weeks. Dr. in a private hospital.	Intervention done by a haemorrhage.	Severe Pre/Pose partum	Nil.

Table-2a

					On Admission						URINALYSIS				
Patient	HB g/dl	ESR 1/hr.	WBC	FDP	Na. mmol/l	K. mmol/l	Ca. mg/dl	P. mg/dl	BUN mg/dl	CREAT md/dl	PH.	Protein mg/dl	WBC. HPF	RBC. HPF	Urine Output in 24 hours
1.	-	100	8.0x10 9/1	x	1.25	5.1	8.5	4.4	106	23.2	1025	500	25	Many	300 cc.
2.	4.2	90	17x10 9/1	>10ug/ml <20g/ml	155	32	7.5	8.2	130	13.9	1015	100	10	Many	300 cc.
3.	7.2	45	16.8x10 9/1	x	126	2.8	6.4	10.3	175	18.8	1035	100	10-15	Nil	600 cc.
4.	6.6	117	6.0x10 9/1	x	127	6.1	7.8	15	90	19.9	1035	100	Many	Many	100 cc.
5.	5.7	67	-	Normal	141	5.2	7.6	4.9	70	8.2	-	Nil	4	2	500 cc.
6.	5.9	117	-	>10ug/ml140	140	32	-	-	98	9.8	-	500	-	Many	Ni.

Table-2b

					ON DISCHARGE								
Patient	HP g/dl	Platelet	PT	APTT	Na mmol/L	K mmol/l	Ca mg/dl	P mg/dl	BUN mg/dl	Creat mg/dl	Protien g/dl	Albumin g/dl	Urine out put in 24 hours.
1.	8.4	213.000	Sec	Sec	132	6.2	80	6.9	92	20.0	3.4	6.0	500 cc.
2.	9.6	264	16 Sec	36 Sec	141	4.2	8.2	7.3	78	18.4	3.2	6.4	450 cc.
3.	9.2	x	-	-	138	3.0	5.4	7.2	92	8.2	3.4	6.4	1300 cc.
4.	6.2	200,000	12 Sec	38 Sec	130	5.4	6.8	9.7	80	17	2.3	5.2	2800 cc.
5.	9.3	x	13 Sec	34 Sec	134	2.3	7.3	5	30	2.5	x	x	2800 cc.
6.	7.5	x		29 Sec	145	4.4	x	x	82	9.4	x	x	Nil

haemodialysis was done. She remained oliguric. She was kept under observation for the next 4 days during which the blood urea and cretinine went up. On the 13th day of admission APD was done again. No marked change was noticed in renal function nor in oliguria. She had the last APD on the 21st day after admission following which there was improvement in renal function and oliguria. She was discharged from the hospital on her request.

3. K.B., 35 years multipara G11, Para10, A1, was admitted in Shaikh Zayed Hospital after abortion and manipulation done by a Dai on the 54th day of last menstrual cycle with history of excessive bleeding, dizziness and vomiting and profound weakness 5 days prior to admission. On admission the patient had deteriorated renal function. APD was done following which the condition of the patient improved and was discharged on 11th day with a stable renal function and good urine output. On her revisit after 2 weeks her condition was found to be stable with normal urine out and normal renal function.
4. A.B., 30 years, multipara G3, Para2, A1, was admitted with history of still birth after 28 weeks. She suddenly developed high grade fever with chills followed by bilateral lumbar pain radiating to groins for the last 1 month. 15 days prior to admission she became edematous and oliguric associated with nausea and vomiting. There was no history of pre or postpartum haemorrhage. The renal function had deteriorated. BUN and cretainine were found to be raised. APD was done twice and hemodialysis once during her stay in the hospital.Her renal function did not improve. She was discharged on request but after 4 weeks she was readmitted and CAPD was started.

5. S.B., 32 years, multipara, G 6, Para 4. A 2, admitted with history of still birth after 28 weeks of apparent normal pregnancy. The delivery was done by a dai, following which she developed high grade fever and postpartum haemorrhage. On examination she had placental debris. During her stay in the hospital she developed oliguria and hypertension with nausea and vomiting. The BUN and cretainine were found to be raised APD was done. During next few days her urine output increased and blood urea and cretinine started comming down. She was discharged from the hospital with normal renal function after one week.
6. S.R., Primigravida admitted with history of bleeding p/v for 5 days prior to admission at Shaikh Zayed Hospital. She had evacuation done at another hospital. At admission here she was febrile, jaundiced and anemic. She remained anuric for three days in hospital. Her blood urea and cretinine continued to rise. She underwent APD once and hemodialysis twice during next two weeks. By that time her urine output increased and she was discharged with improving kidney function.

Comments

There has been a marked decrease in the acute renal failure in pregnancy since 1958. This probably is due to legalization of abortion and secondly due to earlier detection of underlying obstetric complication and the management. A report published by Jean Pierre Grunfield the incidence fell from 40% in 1958, to 4.5% in 1978[3] In another report by P Stratta et al in successive ten years period (1958-67, 1968-77,) the incidence of pregnancy related acute renal failure fell from 43% to 2.8%[5].

Acute renal failure associated with pregnancy and parturition can occur with or without factors related specifically to pregnancy.

Factors which are specifically related to pregnancy include.

a. Acute pyelonephritis is a common complication of pregnancy. It is an extremely rare cause of acute renal failure in non-pregnant subject, but their have been several reports showing occurance of acute renal failure in gravid whomen due to aucte pyelonephritis[4,6,7].

b. Hyperemesis gravidarum and late onset of vomiting of pregnancy which leads to sodium and water loss leading to prerenal azotemia[5].

c. Eclampsia, preeclampsia and its associated hypertension can lead to acute renal failure. In different studies it have been shown that features of pre-eclampsia were present in upto 62% of patients with acute renal failure associated with pregnancy[4,9,14]].

d. Abruptio placentae is another known factor associated with acute renal failure due to acute bilateral cortical necrosis. Grumfield reported that 50% of gravida with abruptio placenta had acute renal failure[4].

e. Uterine haemorrhage if massive can lead to acute renal failure. In different series it has been

reported in 7 to 58% of pregnant women [4,14].

f. There is another disease entity which has been just recognized by Robson et al in 1968, and has been named as idiopathic postpartum renal failure[2]. The idiopathic post partum renal failure occurs within 48 hours to 10 weeks after apparently normal pregnancy and delivery or it can appear in the third trimester of pregnancy. The patient can present with oluguria and at times with anuria and rapidly developing azotemia. Initially the blood pressure remains normal or slightly elevated. The blood picture reveals microangiopathic haemolytic anemia, numerous schistocytes thrombocytopenia, raised serum fibrin degradation product (FDP) normal prothrombin and activated partial thromboplastin time.

In pathogenesis of renal failure the most common factor is that of hypovolemia. Commonly this hypovolemia is due to uterine haemorrhage. It is necessary that blood loss should be replaced as a protective measure against hypovolemia and preventing deterioration in toxemic patient. These patients are highly sensitive to blood loss as noted by Smith et al.[14]. Pre eclamptic women have lower plasma volume and renal blood flow had higher pressor responsiveness to norepinephrine and angiotension II than normotensive gravidas. It has been shown that placenta of pre-eclamptic patient releases less prostaglandin than those in normal gravidas and relative prostaglandin deficiency may be involved in hypersensitivity to haemorrhage. Coagulation change can facilitate acute renal failure.

Hypertension was noted in course of postpartum renal failure[9,10,11]. One of our patient out of six had hypertension as well. Extra-renal involvement of central nervous system like lethargy, coma with gradnal seizures were present in one patient. Oral contraceptive can produce hemolytic uremic syndrome[12] and severe hypertension. None of our six patient had any previous history of oral contraceptives.

In normal pregnancy there is enhanced capacity to produce fibrin and decreased fibrinolytic activity returns to normal in less than one hour after delivery. In abruptio placenta and prolonged intrauterine fetal death the fibrinogen concentration at term is below the normal value, and Factor V+VIII level are markedly reduced, FDP level are elevated. Postpartum haemorrhage and aminotic fluid embolism may lead to defibrination syndrome[13].

Prognosis

In our study five out of six patients had postpartum haemorrhage and one was suffering from eclampsia. 4 patients also had intrauterine fetal death. Three patients recovered completely. Three patients are on chronic intermittent peritoneal dialysis or CAPD without any significant recovery even after 5 months. The three patients out of six who did not recover had massive haemorhage, prolonged intra-uterine death and septicemia. These problems usually lead to acute cortical necrosis rather than acute tubular nerosis. This was most probably the reason of higher incidence of irreversible renal failure in our patients as compared to other reports. Where it has been reported to be 10-38% of all cases of obstetrical renal failure[9,15,16].

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