Motherhood becomes a reality: A case study

Abstract
This paper will present the case of a young type 1 diabetic female who presented with an unexpected pregnancy who had suffered the loss of a baby two years earlier due to preeclampsia. The paper will cover the literature, the guidance from the valuable shared experiences of other Australian units’ approaches to dialysis prescription, foetal monitoring during dialysis and the involvement of the multidisciplinary team. Our efforts in maintaining a continuously revised plan of care, while offering constant support and encouragement to our patient who so desperately wanted a baby, were favourably rewarded. Her pregnancy completely maintained on dialysis, Karen gave birth to a healthy baby girl at 34 weeks gestation. Motherhood became a reality for our patient. The intention of this paper is to contribute more experiential information to the literature available on pregnancy, diabetes and dialysis.

Key Words
Pregnancy, Dialysis, Diabetes, Pre-eclampsia, Foetal monitoring

Background
Conception is relatively uncommon in patients who are on dialysis. Pregnancy in dialysis occurs at a rate of 1-7% in childbearing age women on dialysis, (Holley & Reddy, 2003) with only 40% of these women will give birth to a live infant (Hou, 1998, Toma, et.al. 1999, European Dialysis and Transplant Association, cited in Chao et al, 2002).

The success of pregnancy outcomes is closely related to the time on dialysis, which should maintain relatively low urea and creatinine levels (Toma et al, 1999). Lowering the shifts in the mother's intravascular volume also helps to prevent hypotension which reduces the chance of diminished blood flow to the foetus (Giatras, Levy, Malone, Carlone & Jungers, 1998). Reduced blood flow to the foetus is thought to be associated with foetal distress and consequent onset of premature labour (Holley & Reddy, 2003; Giatris et al, 1998). Dialysis should be increased to between 18 – 24 hours per week in order to maintain a serum urea of < 21 mmol /L and creatinine <531 _mol/L (Holley & Reddy, 2003; Toma et al, 1999).

Frequent dialysis contributes to a less uraemic environment which permits the mother to have a much more liberal and well balanced diet, rich in protein and potassium. Fluid intake is more relaxed, further enhancing the optimal blood volume requirements of pregnancy while hypertension can be controlled (Giatras et al, 1998).

Dangers associated with pregnancy in the diabetic woman are not widely known, outside obstetric and midwifery circles. Reducing the risk of miscarriage, congenital malformations and perinatal mortality is dependent upon adequate metabolic control before conception (Evers, de Walk & Visser, 2004). Ideally folic acid (500 micrograms daily) should be commenced prior to conception to prevent neural tube defects (McElduff, Cheung, McIntyre, Lagstrom, Oats, Ross, Simmons, Walters & Wein, 2005).

Our Challenge
Our Renal Unit’s first experience with a pregnant woman on dialysis began when Karen our youngest female patient (32 yrs) with insulin dependent diabetes informed us that she was 5 weeks pregnant. Her risk factors were multiple. Apart from having diabetes, chronic renal failure and being on dialysis, she had suffered preeclampsia in her previous pregnancy, her baby had died. The current pregnancy had been conceived when Karen’s blood glucose levels (BGL) had been out of control.

The trained renal nurses, midwives, neonatal, intensive care and general nurses of the Royal Hobart Hospital Renal Unit rallied with disbelief, concern and excitement for our pregnant patient who at that stage, we instinctively knew was at significant risk. Although I was feeling slightly overwhelmed with the responsibility of both ‘at risk’ mother and potential baby it was overcome with the
Flux dialysis. High flux also enabled us to this reason change treatment to high
Peters, Neumayer & Budde, 2005). For Martini, Hocher, Diekmann, Dragun, (Haase, Morgera, Bamberg, Halle, supplemental vitamins and phosphate and that we would have to consider hypophosphataemia could develop, water soluble vitamins would be lost, regarding HDF indicated that valuable haemodiafiltration (HDF). Information prior to the pregnancy Karen received diluted as the pregnancy advanced. Normal in pregnancy, and became more albumin was mostly 34g/L, which is
and her creatinine 300-400 mol/L. Her dialysis urea ranged between 9-14mmol/L and the creatinine below 300 mol/L. We were unable to achieve the latter. With daily dialysis, Karen’s pre dialysis urea ranged between 9-14mmol/L and her creatinine 300-400 mol/L. Her albumin was mostly 34g/L, which is normal in pregnancy, and became more diluted as the pregnancy advanced.

Prior to the pregnancy Karen received haemodiafiltration (HDF). Information regarding HDF indicated that valuable water soluble vitamins would be lost, hypophosphataemia could develop and that we would have to consider supplemental vitamins and phosphate (Haase, Morgera, Bamberg, Halle, Martini, Hocher, Diekmann, Dragun, Peters, Neumayer & Budde, 2005). For this reason change treatment to high flux dialysis. High flux also enabled us to use blood volume monitoring (BVM®, Fresenius Medical Care) which we found increasingly valuable both as the pregnancy advanced and during the post partum period to assist in the fluid management. Karen’s post dialysis phosphate did drop (0.68mmol/L) quite soon after she changed to more frequent dialysis. Her phosphate binder (calcium carbonate) was stopped and she was commenced on oral phosphate replacement one month later.

Sodium bath management was evaluated. The normal pregnancy serum sodium value is approx 135mmol/L, the lower range of normal, due to increased plasma volume and naturally lower serum sodium. We decreased Karen’s sodium from 140 to 135, which quenched the thirst Karen was experiencing in the evenings after dialysis. Hou (2003) reported that sodium retention is thought to be the reason for intravascular expansion in pregnancy. We believed that maintaining a lower sodium in our patient contributed to the control of her blood pressure.

The bicarbonate (HCO3) bath was decreased from 32 mmol/L to 25 mmol/L during pregnancy. In an uncomplicated pregnancy HCO3 decreases as the kidney excretes more HCO3 to compensate for the drop in CO2 level due to a relative hyperventilation causing a decrease in PCO2, a relative respiratory alkalosis and decreased HCO3 level. This is due to the relaxant action of progesterone on the smooth muscle, in this case the lungs (Giatris, 1998). We maintained Karen’s HCO3 at 19-20 mmol/L using a HCO3 bath of 25.

Karen’s serum calcium remained within normal parameters using a 1.3 mmol/L bath. It is not clear whether the placental production of active vitamin D is of great clinical significance during pregnancy (Zerwekh, 1986, cited in Giatras, 1998). Calcification of the fetal skeleton requires 25-30g calcium (Giatras, 1998). During pregnancy in the dialysis patient, alterations to calcium metabolism can occur, but in Karen’s case her serum calcium was within normal range.

Anticoagulation
In pregnancy, there is marked increase in fibrinogen and a slight reduction in platelet count, but an increased proportion of larger, younger platelets (Baker et. al. 2006). We noticed clot formation during dialysis early in the pregnancy, which alerted us to the fact that pregnancy is a hypercoagulative state intended to meet the dangers of haemorrhage at placental separation (Miller & Callander, 1989). Heparin in higher doses than usual was used. An initial bolus of 2000 I/U and infusion rate of 1750 I/U per hour maintained Karen’s blood circuit clot free.

Heparin does not cross the placenta (Grossman & Hou, 2001). Although not considered dangerous to the pregnancy, in the event of any ante partum bleeding its effects can be catastrophic. Given the increased risks of miscarriage or ante partum haemorrhage occurring in a potentially hypertensive mother, our protamine sulphate protocol was reviewed, reinforced to all dialysis nurses and readily available (Holley & Reddy, 2003).

Frequent bloods including full blood count, creatinine, urea, electrolytes, parathyroid hormone, folate, liver function tests, and urates, a possible marker for preeclampsia (Wagner 2004), fructosamine and glycosylated haemoglobin (HbA1C) (Jovanovic & Nakai, 2005) (Table 2).

Anaemia management
Erythropoietin (EPO) was administered weekly intravenously (IV) during dialysis, and was increased during the course of the pregnancy. Karen’s haemoglobin varied from 110-130g/L. The dose was increased from 6000 –7000units at 30 weeks gestation. The evidence to date strongly suggests that EPO cannot cross the placenta (Holley & Reddy, 2003).

Karen remained on weekly IV Iron (Polymaltose 100mg) during dialysis throughout the entire pregnancy which maintained adequate iron transferrin saturation (22-39 %) and ferritin levels (756 – 1200 _mol/L). Iron losses are increased during daily dialysis and estimated to be 780 mg per year. An IV dose of 500mg
Therapeutic Indications

EPREX is indicated for the treatment of patients with symptomatic or transfusion requiring anaemia associated with chronic renal failure, anaemia of chronic disease, myelodysplasia, breast and other malignancies or chemotherapy-induced anaemia, congenital and acquired hypoplastic anaemia, aplastic anaemia, and secondary anaemia in cancer patients. EPREX is indicated in patients with moderate-to-severe anaemia (haemoglobin >10 g/dL) scheduled for elective surgery with an expected moderate blood loss (24 units or 900 to 1800 mL) to reduce exposure to allogeneic blood transfusion and to facilitate erythropoietic recovery.

EPREX is also indicated in patients with maintenance dialysis who are not yet on dialysis. EPREX is also indicated to augment autologous blood collection and to limit the decline in haemoglobin in anaemic adult patients who are scheduled for major elective surgery and who are not expected to complete their preoperative blood requirements.

Contraindications

Contraindicated in patients with:

1. Uncontrolled hypertension
2. Known sensitivity to mammalian cell-derived products
3. Patients scheduled for elective surgery, who are not participating in an autologous blood donation programme and who have severe coronary, peripheral arterial, or cerebrovascular disease, including patients with recent myocardial infarction or cerebrovascular accident.
4. Patients who developPure Red Cell Aplasia (PRCA) following treatment with any erythropoietin should not receive EPREX or any other erythropoietin.

Warnings

Hypertension

Hypertension develops or is aggravated in about 30% of patients with chronic renal failure (CRF) treated with EPREX while the haemoglobin is rising during the first 3 months.

The incidence of hypertension is not dose-related and Patients should be closely monitored for changes in haemoglobinolin and blood pressure in CRF patients, but especially during this period when such hypertensive episodes (in some cases with encephalopathy and seizures) are most likely to occur. Particular attention should be paid to sudden, stabbing, migrainous-like headaches and chest pain. Blood pressure should be monitored every 3 months. Patients should take ordinary precautions to avoid potentially hazardous activities such as driving or operating heavy machinery during this period.

Shunt Thrombosis

During haemodialysis, patients treated with EPREX may require an increase in dialysis heparin to prevent clotting the shunt.

Seizures

Seizures have occurred in patients with CRF receiving EPREX with a frequency of from 3 to 7% usually during the first 90 days of treatment. Seizures have been reported in approximately 15% of haemodialysis patients. The incidence of seizure in chronic renal failure patients, pure red cell aplasia (erythropoietin) has been rarely reported after months to years of treatment with erythropoietins. In most of these PRCA patients antibodies to erythropoietins have been reported.

Thrombocytopenia

In patients studied to date, EPREX has been generally well tolerated. Reactions attributable to EPREX were flu-like symptoms, bone pain and chills (incidence 7.6%) occurring within several hours of administration, and allergic reactions such as urticaria and angioedema. There have been a few reports of anaphylaxis.

ADVERSE EFFECTS

Bone Pain

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Before prescribing, please note changes to Approved Product Information found elsewhere in this publication.

PBS dispensed price: 1000 IU in 0.5 mL (6) $147.00; 2000 IU in 0.5 mL (6) $272.00; 3000 IU in 0.3 mL (5) $331.00; 4000 IU in 0.4 mL (6) $447.00; 5000 IU in 0.5 mL (6) $526.50; 6000 IU in 0.5 mL (6) $599.50; 8000 IU in 0.6 mL (6) $659.60; 8000 IU in 0.6 mL (6) $786.00; 10,000 IU in 1.0 mL (12) $1227.00; 20,000 IU in 0.5 mL (6) $934.00; 40,000 IU in 1.0 mL (1) $690.00; 1. Joklman W. Physical Review 1993;272:449-469. 2. Fisher JV. Endorphin 1997;358:366. 3. Nowerwisk N. Med Oncol 1990;14:251-259. 4. Stowell CP, et al. Orthopedic 1995;23:1549-51. 5. Wimets C. Nephrol Dial Transplant 1995;1:33:102-3. 6. Data on file, Janssen-Cilag Australia. 7. MacDougall C. Semin Dial 1992:253 Suppl 1:9-21. EPREX® is the registered trademark of Janssen-Cilag Pty Ltd for epoetin alfa 32 IU injections. Janssen-Cilag Pty Ltd, ABN 47 000 129 575, 1-3 Merton Road, North Ryde NSW 2113. ELS 2000 NSW 060018
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of iron is recommended as soon as the pregnancy is confirmed, if the transferrin saturation is less than 30% (Grossman et al., 1993, cited in Giatris, 1998).

**Fluid Control**

We were governed by blood pressure (BP), relative blood volume (RBV), personal comfort, clinical signs and symptoms, and foetal monitoring. BP was kept within fairly tight parameters of systolic below 160 mmHg and diastolic between 70 – 95mmHg. This is to enable adequate perfusion of the placenta, and low enough to eliminate the chance of Karen suffering a cerebral vascular accident (CVA).  Given the normal blood pressure fluctuations during ultrafiltration this was a difficult challenge.

Unlike many fluid challenges we had to rehydrate Karen as opposed to her restricting her fluids. Although she was given the grace of eating and drinking virtually as she liked, she had previously disciplined herself to approximately 800mls per day. This resulted in occasionally administering up to 200mls of normal saline to boost her BP and perfuse the placenta, when her BP dropped to as low as 110/55.

BVM was used to monitor Karen’s relative blood volume (RBV). We aimed not to allow the RBV to fall below 95% in the second half of pregnancy. We found that Karen never tolerated a drop in RBV below 95%, given the strict BP guidelines we had to be vigilant according to her symptoms and our constant concern for the safety of the baby.  BVM interpretation was often challenging. Karen’s BP was only ever stable when the RBV was kept above 95% and mostly it was 97% to 101%. We noticed that the BP would rise to as high as 180/80 post dialysis after returning the blood, which we tried to return slowly (100mpm) to prevent vasospasm. The BP would settle to a more acceptable 130/70 within 10 minutes of returning the blood. Karen’s personal comfort and clinical signs and symptoms were an indicator of excessive fluid removal. Karen was reliable in telling us if she was feeling light headed, nauseous or developing cramp. The normal fluid gain of pregnancy developed as a general distribution. There was no sign of dependent fluid retention e.g. ankles, feet, hands and orbits.

The assessment of ideal weight in pregnant women on dialysis is challenging. Increase in plasma volume begins in the first trimester of pregnancy and with a 30% to 50% increase overall, allowances have to be made for plasma volume, and fetal and placental growth (Giatris, 1998). Despite the significant increase in cardiac output (30 -50%), the heart rate increases by only 15%. This is balanced by a reduced peripheral resistance. Thus in the absence of an underlying hypertension, BP changes only slightly in the first three quarters of pregnancy. BP usually rises after 30 weeks gestation. (Llewellyn-Jones, 1999).

Our overriding aim was not to allow Karen to become dehydrated. Her previous pre-eclamptic pregnancy and diabetes gave rise to real concerns about increased vascular resistance and we understood that if we removed excessive fluid we would reduce the size of the vascular bed, thus increasing Karen’s BP while reducing the available fluid for the baby. Given the normal fluid gains in pregnancy (Giatris, 1998) we felt confident that over hydration was preferable to drying her out. After the first trimester we were comfortable to base Karen's fluid increases on the weight gain which, considered as a linear increase, is approximately 450gms -500gms every ten days (Giatris, 1998).

**Blood Pressure**

From the eighth week of pregnancy the mother’s mean arterial pressure and total peripheral resistance decrease. They reach their lowest point in the middle of the pregnancy and return to normal or above the pre pregnant readings, at term. In normal pregnancy BP should remain as low if not lower than the basal systolic and diastolic pressures, due to the smooth muscle effect of progesterone which causes decreased vascular resistance (Mageness & Rosenfeld, 1989, cited in McNabb, 2004). Additionally, natriuretic peptides have a relaxant effect upon vascular smooth muscle, stimulated by angiotensin II or noradrenaline (Cunningham, Gant, Leveno, Gilstrap, Hauth & Wenstrom, 2001). In the pre-eclamptic patient, BP is elevated and together with the discovery of proteinuria (>1gm/24hours) is an ominous sign of pre-eclampsia developing. It differs from chronic hypertension in that the BP and proteinuria occur after 20 weeks gestation. Chronic hypertension is an elevated BP that predates the pregnancy and is present 12 weeks postpartum.

Pre-eclampsia is a pregnancy-specific disorder which complicates about 5 –7% of all pregnancies (Wagner, 2004). To describe the full severity of the disorder is far beyond the scope of this paper. Risks associated with pre-eclampsia include acute renal failure, placental abruption, cerebrovascular accident, cardiovascular complications, disseminated intravascular coagulation and maternal death. Karen was commenced on low dose aspirin as a preventative measure given her past history of pre-eclampsia. Although the evidence to support this is not conclusive, the studies which have been done do show a reduction in the frequency of preclampsia (Coomarasamy et. al., 2001, cited in Wagner, 2004; Jungers, 1997, cited in Holley & Reddy, 2003; Taberianet. al., 2001). The Collaborative Low dose Aspirin Study in Pregnancy found that women at risk of early onset pre-eclampsia, serious enough to require very early pre-term delivery might well benefit from the use of low dose aspirin (CLASP Trial, 1994; 1996).

**Diabetes Management**

An insulin pump was introduced at 8 weeks gestation. Inserted subcutaneously into Karen’s abdomen, the insulin pump is the best available method for optimal glycaemic control (McElduff et al, 2005). The
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pump delivered a basal rate of insulin pre-programmed to coincide with the varying demands over every 24 hour period. Karen could also give herself boluses according to her blood glucose levels (BGL) as required. BGLs were maintained between 3-6mmol/L throughout her pregnancy. The desired preprandial target is 4.5-5.5mmol/L and postprandial < 8mmol/L at 1 hour and 7mmol/L at 2 hours (McElduff et al, 2005).

The Baby During Dialysis

The baby’s well being was monitored by the dialysis nurses (several of whom were current registered midwives) during dialysis using a cardiotocograph (CTG). This commenced at 23.5 weeks gestation when the baby is considered to be viable, compatible with life outside of the mother, at that stage (Giatras, 1998). We positioned Karen semi-reclined with left lateral tilt from twenty weeks and left lateral on a bed with ample pillows for support from 26 weeks to ensure decompression on the vena cava, maintaining adequate venous return to the heart for perfusion of the placenta (Kinsella & Lohmann, 1968 cited in Cunningham et al, 2001). The foetal heart was checked as soon as we positioned Karen in this way to eliminate the possibility of cord compression.

Karen’s reporting of her baby’s fetal movements, hourly fetal heart and CTG in last half hour every dialysis was recorded from 24 weeks. Hypotensive episodes are common in the last half hour of any dialysis and our main responsibility was to prevent any significant alteration to the uteroplacental and foetal perfusion (Giatras, 1998).

Karen was admitted to the obstetric ward at 23 weeks gestation for BP monitoring, rest and daily monitoring of the baby. This was a difficult time for Karen as she was now facing the time when in her previous pregnancy, things went so terribly wrong. As I accompanied Karen to her room on the day of admission, she noted that she was being accommodated in the same (single) room as in the previous pregnancy, but very bravely asked me not to organize a different room. She seemed positive that things could turn out differently this time and I was moved to observe Karen ‘face her demons’ with such courage.

Karen was discharged home after four weeks of constant vigilance, patience and perhaps the good fortune that this second pregnancy, with the same father, was not going to develop into pre-eclampsia. A different father is a significant risk factor in the event of pre-eclampsia onset (Wagner, 2004). Her BP did fluctuate dramatically during the admission from 110/55mmHg to 160/80mmHg. Post discharge, her obstetrician had Karen attend her obstetrician every afternoon, at 5pm, when Karen’s BP would normally be at its highest. Throughout the pregnancy an alpha antagonist (methyldopa) was used successfully and titrated (250mg-500mg daily) by her obstetrician according to BP. Methyldopa has been used in pregnancy for over forty years and remains the first drug of choice for essential hypertension (Cockburn, et. al., 1982, cited in Hou, 1999).

Close monitoring of the baby was essential, so weekly serial scanning of the pregnancy, checking fetal well being and amniotic volumes was performed (Holley & Reddy, 2003). The results of all scanning, including the 19 week obstetric and anatomy scan had all been very reassuring, raising our hopes that Karen’s baby would be born healthy with a good chance of survival.

At 28 weeks there was a subtle change in the mood of the more senior staff to whom Karen’s care was allocated. We knew now that there was increased hope that Karen would give birth to a live baby and our efforts toward this outcome were beginning to feel challenged by the obstetrician’s decisions. There was clearly an atmosphere emerging, built on not only familiarity with our pregnant patient, but increasing responsibility, concern and determination that we would get her through this. When her obstetrician decided to discharge Karen home we were somewhat perplexed. We thought that she would be safer in hospital, but as it turned out Karen’s BP improved in the evenings after leaving hospital so our fears were unfounded. Karen agreed that it was better to be home, that she felt less stressed, less scrutinized, more rested and adequately supported by her husband and mother.

Multidisciplinary Team

The success of the pregnancy in a dialysis patient is closely related to the success of the multidisciplinary management (Giatras, 1998; Nakabayashi, 1999). Multidisciplinary meetings were held at 16 and 32 weeks gestation. In Karen’s case we enlisted the contributions of her nephrologists, obstetrician, endocrinologist, nurses, midwives, diabetes nurse educators, dietitian, social worker and the baby’s neonatologist.

Karen, her husband and mother were present at these meetings which provided a useful means for communication of all that was to be done for Karen and opportunities for everybody to ask questions. The multidisciplinary approach, the gathering of information, guidelines, advice and sharing of our clinical findings and outcomes of Karen’s daily care, brought together the plan, enabled its implementation and rewarded all concerned with Karen’s positive outcome.

The meetings gave Karen, her husband and mother the opportunity to ask questions, listen to explanations from each specialist source and above all, know that every aspect of her care was being covered. All being present meant that everyone heard what was being said and done, and that Karen’s queries could be clarified by any member of the multidisciplinary team.

Pregnancy Complications

At 30 weeks, Karen developed an itch over her abdomen which had been diagnosed as pruritic urticarial papules and plaques of pregnancy (PUPPPS). Believed to be due to an inflammatory response
brought about by the stretching of the superficial layer of the skin, PUPPPs did not pose any additional dangers to the pregnancy (Cohen et al., 1989; Beckett & Goldberg, 1991). The itch became more general, particularly on the soles and hands which was beginning to give the picture of cholestasis, a liver condition linked to high levels of circulating oestrogen which inhibit the intraductal transport of bile acids (Baker et al, 2006). Liver function tests were proving to be within the normal range for pregnancy and it was the defining high bile salts result of 85_mol (reference range 1–26), performed in Melbourne and for which we had to wait several days, that confirmed Karen had cholestasis. Cholestasis usually presents between 32–36 weeks gestation and affects 0.5–1% of pregnancies. Its mechanism is not clear, but cholestasis is associated with sudden fetal death in utero, meconium liquor and premature labour (Baker et al, 2006).

Birth Preparation
At 34 weeks pregnant her obstetrician decided that it was time to prepare for delivery and organized that Karen be admitted for the administration of corticosteroids to mature the baby’s lungs and deliver the baby (Llewellyn-Jones, 1999). After the second dose of steroids was given, Karen began to experience painful contractions, the CTG showed a non-reassuring trace of ‘late’ decelerations. Karen was immediately prepared for an emergency caesarian section which resulted in the birth of her baby girl.

Post Partum
The baby was well at birth with apgars of eight at one minute and nine at five minutes, breathing well on normal room air. She was transferred to the neonatal intensive care unit (NICU) where she was monitored and cared for by NICU nurses until she was discharged at four weeks. Immediately postpartum, Karen was transferred to the High Dependency Unit. Her blood loss from the birth was estimated at 1200mls and she had not been dialysed for 2 days. Her BP was as low as 80/40 (post spinal anaesthesia) and it was difficult to dialyse her on the first day. We knew that there would be a major fluid shift sometime in the first few days after the birth and daily dialysis continued for the first week post partum.

Karen had gained a total of 16 kgs during her pregnancy. On the first day we weighed her, 3 days postpartum, she was 5.3 kgs below her pre birth, pre dialysis weight. The 5.3kgs represented the weight of the baby (2.4kgs) the placenta (not weighed), amniotic fluid (copious, according to the obstetrician) plus the estimated blood loss (1200mks). We continued to remove fluid over the week and arrived at a dry weight 10 kilos below that of her pre birth dry weight. Prevention of pulmonary oedema was our main aim and we were successful in preventing this complication.

Karen left hospital eight days after her caesarian and continued to visit her baby daily and attend second daily dialysis. The baby was formula fed after Karen had persevered with 8 days of breastfeeding after which time her milk simply didn’t ‘come in’. Her BP, blood results and BGLs have remained stable. She was 6 kgs above her normal pre pregnancy dry weight, which she can attribute to a healthy body weight increase from a pregnancy which gave her a healthy baby daughter. She was the first dialysis dependant mother to give birth at the Royal Hobart Hospital.

Conclusion
The management of a pregnancy on dialysis is rare and for those of us who have participated in the care would have found it a daunting prospect with no past experience from which to draw. Ever mindful of risk factors, gathering information, constant vigilance, communication and enlisting the cooperation of the multidisciplinary team, contributes vastly to a young woman on dialysis with diabetes, becoming a mother against the odds. It is intended that our experience shared by distribution of this paper be a significant contribution to the available information regarding the management of a woman with diabetes on dialysis.

*Consent was given by the patient to tell her story. A fictitious name has been used to protect her identity.

| Table 1. Dialysis Prescription: Alterations to accommodate pregnancy |
|---------------------------------|-----------------|-----------------|
| Prescription | Pregnancy | Pre-pregnancy |
| Type * | Haemodialysis | Haemodiafiltration |
| Time * | 4.5 hrs | 5 hrs |
| Frequency * | 6 days/week | 3 days/week |
| Dialyser * | Fresenius FX60 | Fresenius HF 80S |
| Potassium | 2 mmol/l | 2 mmol/l |
| Sodium | 135 mmol/l | 140 mmol/l |
| Glucose | 5 mmol/l | 5 mmol/l |
| Bicarbonate * | 25 mmol/l | 32 mmol/l |
| Calcium | 1.3 mmol/l | 1.3 mmol/l |
| Heparin bolus | 2000 IU | 2000 IU |
| Heparin infusion * | 1250 IU/hr | 1500 IU/hr |
| Fluid Control * | BVM & Volumetric Fluid Control | Volumetric Fluid Control |

* Parameters requiring change
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Table 2. Blood Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Frequency</th>
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</thead>
<tbody>
<tr>
<td>Full Blood count</td>
<td>weekly</td>
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<tr>
<td>Ferritin/ Iron Studies</td>
<td>monthly</td>
</tr>
<tr>
<td>PTH</td>
<td>monthly</td>
</tr>
<tr>
<td>Creatinine</td>
<td>weekly</td>
</tr>
<tr>
<td>Urea &amp; Electrolytes</td>
<td>weekly</td>
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<tr>
<td>Bicarbonate</td>
<td>weekly</td>
</tr>
<tr>
<td>Calcium</td>
<td>weekly</td>
</tr>
<tr>
<td>Phosphate</td>
<td>weekly</td>
</tr>
<tr>
<td>Liver Function Tests</td>
<td>weekly</td>
</tr>
<tr>
<td>Fructosamine</td>
<td>monthly</td>
</tr>
<tr>
<td>Random Glucose</td>
<td>weekly</td>
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<tr>
<td>Urates</td>
<td>weekly</td>
</tr>
<tr>
<td>Hba1c</td>
<td>3 monthly</td>
</tr>
<tr>
<td>B12 folate</td>
<td>3 monthly</td>
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<tr>
<td>Bile Salts</td>
<td>as ordered</td>
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Table 3. Education Topics: Provided by midwifery trained nephrology nurses

<table>
<thead>
<tr>
<th>Patient</th>
<th>Nursing Staff</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antenatal</td>
<td>Normal pregnancy</td>
</tr>
<tr>
<td>Diabetes</td>
<td>BP parameters</td>
</tr>
<tr>
<td>Diet &amp; fluids</td>
<td>Signs &amp; symptoms of Preeclampsia</td>
</tr>
<tr>
<td>Parenting</td>
<td></td>
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<tr>
<td>Breastfeeding</td>
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References

