CASE REPORT

Safe delivery of two parturient women in severe metabolic acidosis

Ina Ismiarti Shariffuddin,¹ Vineya Rai,¹ Y K Chan,¹ Rajesh Kumar Muniandy²

¹Department of Anaesthesiology, University of Malaya, Kuala Lumpur, Wilayah Persekutuan, Malaysia
²Medical Based Department, Universiti Malaysia Sabah, Kota Kinabalu, Sabah, Malaysia

Correspondence to

Dr Rajesh Kumar Muniandy,
 rajeshkumar.ums@gmail.com

Accepted 3 May 2014

SUMMARY

Care of an acutely ill parturient is particularly difficult when we have to balance the needs of both mother and the fetus to survive. The literature suggests there should be emphasis on stabilising the mother's condition. In dealing with metabolic acidosis, however, we believe delivering the baby early might not only relieve the threat of the acidosis on the mother, it may be the only way to deliver a live baby. We report two parturient women with severe metabolic acidosis which was considerably reduced very soon after the delivery and how our timely delivery resulted in the birth of two neurologically intact babies.

BACKGROUND

Stillbirths affect 2.6 million fetuses every year.¹ Most are due to poor care by providers who have failed to focus on the precarious nature of fetal existence in utero.^{1–2} Fetal well-being is very dependent on maternal well-being. When parturients are critically ill, there is often debate on how far we should stabilise the mother before subjecting ourselves to the challenge of delivering the fetus.^{3–4} We accept this consideration but our experience with two of these parturients show that delivering the fetus early may not only avert fetal death but also improve the parturient considerably.

Case 1

A 34-year-old woman, at 37 weeks of gestation, was admitted to our obstetric unit with a 24 h history of poor oral intake, vomiting and shortness of breath. This was also associated with a 3-day history of polydipsia and polyuria. She was diagnosed with type 2 diabetes mellitus 7 years ago and was on insulin treatment. However, her blood glucose was poorly controlled with glycated haemoglobin of 9.6.

She appeared lethargic and dehydrated with acidotic breathing. Investigation revealed a random blood sugar level of 20.6 mmol/L. Venous blood gas showed a pH of 7.2 with base excess (BE) of -19 and bicarbonate level of 10 mmol/L. Urine was positive for ketones. A diagnosis of diabetic ketoacidosis was performed and the patient was admitted to our intensive care unit (ICU) for close monitoring and treatment. A sliding scale of insulin was started and she was hydrated accordingly. The fetus was monitored daily via cardiotocography (CTG).

Her obstetric history revealed that she had had a fresh stillbirth 7 years previously when she presented to the obstetric unit at 32 weeks of gestation with diabetic ketoacidosis. Her management in this current admission was rapidly escalated to a team effort consisting of care from obstetric, endocrine, intensive care and anaesthesia providers whose initial plans

were focused on stabilising the mother. Realising that the fetus was a huge consideration and that we could not allow the mother to have another stillbirth, we decided to proceed with caesarean section as soon as the BE was less than 10 mmol/L. Serial arterial blood gas (ABG) of the patient was taken and the results are shown in [table 1](#).

An intrathecal injection of 1.8 mL of 0.5% heavy bupivacaine and 20 µg fentanyl was administered. Intraoperative blood pressure was maintained between systolic of 100–130 mm Hg and diastolic of 50–70 mm Hg. A 3.3 kg fetus was delivered uneventfully. The Apgar score of the baby was 9 at 1 min and 10 at 5 min. The baby's ABG showed a pH of 7.18, partial pressure of carbon dioxide (pCO₂) 48.6 mm Hg, partial pressure of oxygen (pO₂) 12.2 mm Hg, bicarbonate 15 and BE -9. The baby was admitted to the special care nursery for observation. The caesarean section was otherwise uneventful. The patient was monitored in ICU again postoperatively and her ABG were as shown in [table 2](#). She was discharged to the ward 3 days later. The patient and her baby were discharged 20 days after admission.

Case 2

Our second patient, a 29-year-old primigravida, at 34 weeks of gestation, was admitted to our surgical ward with a 1-day history of sudden onset of epigastric pain. This was associated with frequent vomiting. Blood investigation revealed a very high level of serum amylase and slightly raised liver enzymes. Ultrasound of the hepatobiliary system showed acute cholecystitis secondary to cholelithiasis and a diagnosis of acute pancreatitis was performed. She deteriorated clinically with respiratory failure and metabolic acidosis requiring intubation and was further managed in ICU. Despite adequate resuscitation, she remained acidotic with her ABG as shown in [table 3](#). The fetus was monitored daily with bedside ultrasound and CTG. Intramuscular dexamethasone was given to the patient to mature the fetal lungs.

The management of this patient was a team effort consisting of care from obstetric, surgical, neonatology, intensive care and anaesthesia providers. The team decided to proceed with emergency caesarean section under general anaesthesia at day 2 of the patient's admission to ICU. The baby was delivered with an Apgar score of 7 at 1 min and 10 at 5 min. His umbilical blood gas was pH 7.21, pCO₂ 41.8 mm Hg, pO₂ 52.8 mm Hg, BE -10.1 and bicarbonate of 15.8. The patient's ABG was monitored postoperatively and was as shown in [table 4](#).

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To cite: Shariffuddin II, Rai V, Chan YK, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2014-205135

Table 1 Serial arterial blood gases from admission until the operative day

Date and time (h)	pH	pO ₂ (mm Hg)	pCO ₂ (mm Hg)	Glucose (mmol/L)	Lactate	BE	HCO ₃
Day 1 12:56	7.384	144	13.9	–	–	–16.4	13.2
Day 1 16:09	7.358	148	22.2	10.9	2.4	–11.8	15.8
Day 1 20:12	7.348	150	24.0	10.4	1.4	–11.7	15.8
Day 1 23:57	7.356	176	23.4	9.7	2.4	–11.2	16.0
Day 2 10:13	7.380	151	21.7	11.1	2.4	–11.6	15.9
Day 2 12:20	7.351	156	23.4	11.2	2.8	–10.6	16.7
Day 2 17:52	7.396	156	21.3	7.9	2.2	–11.2	16.3
Day 3 05:50	7.36	149	22.1	7.3	2.1	–12.4	15.6
Day 3 17:06	7.441	132	21.2	6.1	2.1	–9.2	18.1

BE, base excess; pCO₂, partial pressure of carbon dioxide; pO₂, partial pressure of oxygen.

Table 2 Serial arterial/venous blood gas postdelivery

Date and time (h)	pH	pO ₂ (mm Hg)	pCO ₂ (mm Hg)	Glucose (mmol/L)	Lactate	BE	HCO ₃
Day 1 12:08	7.373	75.3	26.6	7.3	1.4	–9.1	17.7
Day 1 23:09	7.400	102	26.6	5.7	1.3	–7.8	18.7
Day 2 05:47	7.406	84.3	25.2	7.2	1.4	–8.3	18.4
Day 2 12:13	7.426	98.7	25.3	7.0	1.6	–7.2	–9.2
Day 2 23:57	7.447	113	27.7	5.0	1.2	–4.5	21.3
Day 3 05:40	7.446	115	30.6	5.1	1.2	–2.7	22.7
Day 3 12:15	7.456	111	27.4	7.9	1.9	–2.6	23.2
Day 4 06:12 (VBG)	7.410	39.3	33.2	–	–	–3.2	21.6
Day 08:35 (VBG)	7.425	37.9	38.5	–	–	0.8	24.5

BE, base excess; pCO₂, partial pressure of carbon dioxide; pO₂, partial pressure of oxygen.

Table 3 Serial arterial blood gases from admission until the operative day

Date and time (h)	pH	pO ₂ (mm Hg)	pCO ₂ (mm Hg)	Glucose (mmol/L)	Lactate	BE	HCO ₃	FiO ₂	PEEP
Day 1 22:55	7.284	140	19.6	5	0.7	–16.6	12.4	HFM	–
Day 2 03:00	7.206	327	25.7	5.3	0.6	–16.6	12.0	1.0	10
Day 2 08:00	7.254	181	23.8	5.4	0.6	–15.6	12.7	0.4	8
Day 2 11:40	7.257	148	24.2	5.1	0.7	–15.6	12.7	0.4	8

BE, base excess; pCO₂, partial pressure of carbon dioxide; pO₂, partial pressure of oxygen.

Table 4 Serial arterial blood gas postdelivery

Date and time (h)	pH	pO ₂ (mm Hg)	pCO ₂ (mm Hg)	Glucose (mmol/L)	Lactate	BE	HCO ₃	FiO ₂	PEEP
Day 1 17:11	7.246	138	28.4	7.7	1.2	–14	13.9	0.35	14
Day 1 23:34	7.311	179	23.5	6.3	0.6	–12.2	15	0.35	12
Day 2 08:46	7.338	160	30.9	6.6	0.7	–8.5	17.7	0.35	4
Day 2 12:19 (postextubation)	7.364	149	29.7	6.5	1.1	–7.8	18.4	FM	–
Day 3 12:21	7.434	135	33.4	4.9	0.7	–1.7	23.3	FM (40%)	–

BE, base excess; pCO₂, partial pressure of carbon dioxide; pO₂, partial pressure of oxygen.

DISCUSSION

Our two cases highlight situations which are life-threatening for the mother as well as for the unborn child. Both the parturients had care escalated upwards by being admitted to the ICU and both benefitted from comprehensive team care which saw the providers balancing the difficult job of stabilising the mothers' condition and determining the optimal timing of the delivery in order to have the unborn children delivered safely.

The first parturient already had a fetal death at an unusually early part of the third trimester in her previous pregnancy due to severe diabetic ketoacidosis. We had to push for the delivery of the baby against the conventional wisdom of stabilising the mother all the way.^{3 4} The fetus highlighted in the case report by Carroll *et al* died while the parturient was being stabilised for diabetic ketoacidosis. How far should we persist in trying to correct an uncorrectable acidosis? In the first case, the acidosis

was difficult to manage despite adequate insulin cover and hydration. Only after the delivery of the fetus was there any semblance of a return to normal of the acidosis which was seen in both parturients, within a matter of about 48 h. In the second patient, her vast improvement even made it possible to be extubated the day after the caesarean section.

Both the babies were acidotic after the delivery. Although the level of acidosis did not reach the level described by Low *et al*⁵ to be associated with severe newborn neuropathy, it reached a level high enough to be described as moderately severe in his series. From the physiology point of view, acidosis in the parturient causes a leftward shift in the maternal oxygen dissociation curve.³ This, together with decreasing uterine blood flow in the parturients who compensate for the acidosis by hyperventilating, can remarkably decrease oxygen delivery to the fetus and threaten its survival.

In giving care to these two parturients we were driven by the need not only to prevent maternal and fetal deaths but also to make sure the fetuses were delivered neurologically intact,

unharmful by the metabolic and tissue calamities in the mother.^{6–8} This was especially so with the first parturient who had already had the misfortune of having a prior fetal death in the setting of a diabetic ketoacidosis. We believe that delivering them when we did not only averted mishaps in the fetuses but indirectly and very rapidly improved the metabolic acidosis in the parturients. Delivering the babies probably reduced the metabolic load on the parturients and also assisted remarkably in their recovery. We are advocating timely delivery in metabolic acidosis especially when walking the tightrope of balancing both the maternal and fetal's needs to survive.

Contributors All the authors contributed equally to the production of this manuscript.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- ▶ Severe metabolic acidosis is a life-threatening event in a parturient, both to the mother and fetus.
- ▶ Early delivery is vital for a good outcome to the mother and fetus.
- ▶ In spite of resuscitation and treatment, metabolic acidosis is very resistant to stabilisation in an undelivered parturient.
- ▶ Metabolic acidosis tends to be more easily resolved with early delivery.

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