Interpreting Umbilical Cord Blood Gases: Uteroplacental Insufficiency

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Uteroplacental insufficiency can be caused by many factors. These include critical maternal anemia or oxygen desaturation, maternal hypotension, excessive uterine activity with hypertonus, certain maternal medications and drugs, abnormalities of the uteroplacental vasculature, placental infarcts, placental abruption, and uterine rupture without an expulsion. The case below illustrates typical umbilical cord blood gas findings in severe uteroplacental insufficiency.

The comparative reliability of bicarbonate versus base deficit as a marker of metabolic acidosis is also addressed here.

Case 8: Uteroplacental Insufficiency

The mother was a 19-year-old, gravida 3, para 1, aborta 1, with an intrauterine pregnancy at approximately 32 weeks by poor dates with preterm labor and diffuse abdominal pain for four hours. There was a history of cocaine use, last taken on the day of admission. Uterine contractions, occurring every minute, were associated with recurrent late decelerations with almost every contraction on the FHR (fetal heart rate) monitor. With the cervix three cm dilated and 70% effaced, the mother underwent an emergency cesarean section. An abruption involving 50% of the placental surface was found at surgery. Thick meconium was noted at the delivery of a 2500 g female infant with Apgar scores of 1, 2, and 4 at one, five and 10 minutes, respectively.

Cord blood gas results were as follows (presented without base deficits):

	Umbilical Vein	Umbilical Artery
pH	6.83	6.79
Pco_2 (mmHg) ***(kPa)***	100 *13.33*	110 *14.67*
Po_2 (mmHg) ***(kPa)***	10 *1.33*	8 *1.07*
HCO_3^- (mmol/L)	16	16

"If one were to rely upon this information alone, one would conclude that the severe acidosis is essentially all respiratory in origin. This interpretation would be erroneous, as the following information demonstrates."

Interpretation

In both the umbilical venous and arterial samples, the pHs are severely acidotic, and the PCO_2s are very severely elevated. The bicarbonate ($HCO3^-$) of 16 mEq/L in the umbilical venous sample is at the lower end of normal, and the bicarbonate of 16 mEq/L in the umbilical arterial sample is marginally low. If one were to rely upon this information alone, one would conclude that the severe acidosis is essentially all respiratory in origin. This interpretation would be erroneous, as the following information demonstrates.

Bicarbonate versus Base Deficit/Base Excess

Cord blood gas results (providing both bicarbonate and base deficit) were as follows:

	Umbilical Vein	Umbilical Artery
pH	6.83	6.79
Pco_2 (mmHg) ***(kPa)***	100 *13.33*	110 *14.67*
Po_2 (mmHg) ***(kPa)***	10 *1.33*	8 *1.07*
HCO_3^-(mmol/L)	16	16
BD (mmol/L)	23	25

Had you been texting at the moment these base deficits came to your attention, without doubt, you would have texted, "OMG!" Now it becomes clear that in addition to very severe respiratory acidosis, there exists a concomitant very severe metabolic acidosis in each sample. The base deficit is defined as the amount of base required to titrate the sample to a pH of 7.40 at a PCO_2 of 40 mmHg at 37° C. Base excess is defined as the amount of acid required to titrate the sample to a pH of 7.40 at a PCO_2 of 40 mmHg at 37°C. Base deficit, unlike bicarbonate concentration, also takes into account the hemoglobin concentration. Base deficit (or base excess) is a far more satisfactory and clinically useful way to assess the non-respiratory (metabolic) portion of acid-base balance than is bicarbonate. Base deficit corrects for an elevated (or depressed) PCO_2 resulting in an increased (or decreased) bicarbonate value. Below, the venous and arterial cord gases each have been "normalized" to a PCO_2 of 38 and 49 mmHg (the mean normal venous and arterial PCO_2s), respectively, as is done artificially by the equation used to calculate the base deficit in blood gas analyzers. Then it can be seen that bicarbonate "falls," revealing the underlying metabolic acidosis quite as well as does the base deficit.

	Umbilical Vein	Umbilical Artery
pH	7.00	6.92
Pco_2 (mmHg) ***(kPa)***	38 *5.07*	49 *6.53*
Po_2 (mmHg) ***(kPa)***	10 *1.33*	8 *1.07*
HCO_3^-(mmol/L)	8.9	9.5
BD (mmol/L)	23	25

These values were obtained by using the Siggaard-Andersen Alignment Nomogram provided below. As long as two values are provided, using a straight edge, all the other data can be obtained. In this case, the PCO2 and the base deficit are placed on a straight line. For the venous umbilical cord blood gas, a straight line (line A) is extended from the PCO2 of 38 mm Hg, found on

the right edge of the nomogram and base excess (negative base deficit) of 23 mmol/L, found in the wing-shaped part of the nomogram (using hemoglobin of 15 g/100 mL found at the bottom of the wing-shaped part of the nomogram). The pH and the bicarbonate can then be read off the nomogram in the center and near left parts of the nomogram, respectively. The same approach is then used for the arterial umbilical cord blood gas. The results appear in line B in the nomogram.

"In some hospitals, in a cost-containment effort, only an umbilical artery sample is obtained. However, unless both umbilical venous and umbilical arterial samples are obtained, one cannot be certain that the only sample obtained is indeed from an umbilical artery."

As can be seen from the equation below, an increase or decrease in CO_2 will result in a reciprocal increase or decrease in $HCO3^-$ (bicarbonate).

$$CO2 + H_2O \leftrightarrow HCO_3^- + H+$$

If one were to rely upon bicarbonate as a guide to the degree of metabolic acidosis, what initially appeared to be a borderline bicarbonate value would be revealed, as efficient ventilation begins, to be a severe metabolic acidosis with low bicarbonate values. Base deficit permits metabolic acidosis to be appreciated before PCO_2 is normalized by automatically correcting for deviations of PCO_2 from "normal." Therefore, it is the more reliable parameter to follow when a PCO_2 significantly deviates from average. If the PCO_2 is at or near a normal value, it makes no difference whether one uses bicarbonate or base deficit to determine the degree of metabolic acidosis.

A significantly depressed PCO_2 is uncommon in umbilical cord blood gases, except when the mother either spontaneously hyperventilates or is hyperventilated under general anesthesia. For a mother's hyperventilation to lower fetal PCO_2, the uteroplacental circulation must be unimpeded. If the PCO_2 is artificially decreased by exposure to an air bubble, calculated bicarbonate is low, but the base deficit is unaffected. As a general rule, the base deficit should be used in the interpretation of metabolic acidosis or alkalosis. For this reason, information on the base deficit will be provided, and bicarbonate values will be omitted as additional cases are presented.

The umbilical cord blood gas values in this case show combined severe metabolic acidosis and severe respiratory acidosis. The history of diffuse abdominal pain, maternal cocaine use , and very frequent uterine contractions all suggested placental abruption, which indeed was the correct diagnosis. Placental abruption and excessive uterine activity both have the potential to critically diminish the oxygen available to the fetus, as reflected in the recurrent late decelerations. "Late decelerations are smooth, gradual, symmetrical decreases in FHR beginning at or after the peak of the contraction and returning to baseline only after the contraction has ended," and are associated with more than 50% of contractions. In any given sequence, they are both recurrent and proportional to the amplitude and duration of the underlying contraction, and the drop from baseline to nadir must be greater than 30 seconds. As umbilical arterial blood moves through the intervillous space, uteroplacental insufficiency leads to a poor exchange of carbon dioxide and oxygen, resulting in umbilical venous blood with higher than normal PCO_2, and lower than normal PO_2, and little or no correction of any underlying metabolic acidosis. As the fetus receives "under-ventilated" and under-oxygenated blood from the umbilical vein, FHR decelerations and respiratory and metabolic acidoses progress as the fetal heart rate baseline rises. Elevated PCO_2 and depressed venous PO_2, together with approximately equal derangements of both umbilical venous and arterial base deficits, are the hallmarks of uteroplacental insufficiency.

Thick meconium is very unusual in premature deliveries but is quite common in term or post-term infants. Additionally, maternal cocaine use increases the risk of intrauterine growth restriction. Either this is a large for gestational age 32-week infant, a 36-38 week average for gestational age infant, or a small for gestational age term or post-term infant. The third scenario seems most likely.

Key Points

- The base deficit reflects metabolic acidosis or alkalosis better than bicarbonate in the presence of either high or low PCO_2.
- The hallmarks of uteroplacental insufficiency are elevated PCO2 and depressed venous PO2 together with approximately equal derangements of both umbilical venous and arterial base deficits.

References:

1. *Pomerance J. Umbilical cord blood gas casebook: Interpreting umbilical cord blood gases, Part I. J Perinat 1997;17:503-4.*
2. *Collier CR, Hackney JD, Mohler JG. Use of extracellular base excess in diagnosis of acid-base disorders: A conceptual approach. Chest 1972;61(Suppl)6S-12S.*
3. *Addis A, Moretti ME, Syed F, Einarson TR, et al. Fetal effects of cocaine: An updated meta-analysis. Reprod Toxicol 2001;15:341-69.*
4. *ACOG technical bulletin. Fetal heart rate patterns: monitoring, interpretation, and management. Number 207. Int J Gynaecol Obstet 1995;51:65-74.*
5. *Macones GA, Hankins GDV, Spong CY, Hauth J, et al. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: Update on definitions, interpretation, and research guidelines. Obstet Gynecol 2008;112:661-6.*
6. *LeBlanc PE, Parekh AJ, Naso B, Glass L. Effects of intrauterine exposure to alkaloidal cocaine ("crack"). Am J Dis Child 1987;141:937-8.*

Disclosure: The author has no disclosures.

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