Lactic Acidosis: A Case Presentation Of Acute Respiratory Failure Do To Medicine Induce Lactic Acidosis

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Abstract

CASE REPORT

The case of a 68yr old white Hispanic female who comes into the hospital for acute shortness of breath, cardiac arrhythmia, and muscle weakness which began over a week ago. It had progressively gotten worse prior to her calling the fire rescue (paramedic service). The patient was intubated upon arrival to the emergency department.

The patients past medical history is positive for hypertension HTN, non-insulin dependent diabetes mellitus NIDDM, hypothyroidism and hypercholesteremia.

The patient has had NIDDM for the past 18 years; she is presently taking Glucophage 850 mg PO BID, Glucotrol XL10 mg PO< BID and Avandia 4 mg PO QD. She takes Diovan 80 mg PO< BID for her Hypertension, Lipitor 10 mg for her hypercholesteria and Syntroid 0.150 mcg PO, QD.

Her surgical history is positive for cholecystectomy over 10 years ago and hysterectomy over 18 years ago. Her past family history is positive for HTN, NIDDM and coronary artery disease CAD.

After being intubated in the Emergency Department she is placed on a ventilator and her arterial blood gases revealed the following results:

pH: 6.97 with a PCO2 of 24, and a HCO3 level of 6. Her CBC shows a white blood count of 18,000, hemoglobin level of 12 and a hematocrit level of 36. Her electrolyte panel reveals the following results: Sodium 135, K 3.8, Cr. 118, Bun 50, Cr. 2.6

Her Chest X-ray reveals no active disease process, with a mildly enlarged heart. The renal ultrasound also is negative for any pathology.

THE PRIMARY WORKING DIAGNOSIS WAS AS FOLLOWS:

1. Acute respiratory failure
2. Acute renal failure vs. chronic renal failure
3. Metabolic acidosis
4. Lactic acidosis (medicine induced)

The patient was started on Rocephin 2 grams IV, QD and Cleocin 600 MG IV Q8 hrs. She is also given 14 ampoules of Sodium Bicarbonate, and placed on respiratory support with aerosol treatments, while blood and urine cultures where ordered.

The next day, the patients renal functions continued to deteriorate with the blood urea nitrogen levels at 89 and the creatinine levels of 5.6, her renal output also began to decline, with less than 20cc per hour.

Acute hemodialysis treatments are implemented on a schedule of Monday, Wednesday and Friday. On the second week of treatment, her renal functions begin to improve and the blood urea and creatinine levels begin to return to normal.

The patient is taken off hemodialysis treatments and continues to besupported with IV fluids and total parenteral nutrition TPN. During this time she has been taken off Glugophage and maintained with subcutaneous insulin on a sliding scale.

She is extubated on the third week and her mental status functions begin to return to normal. She is finally transferred to a rehabilitation facility on the fifth week.

FINAL DIAGNOSIS

Acute respiratory failure due to acute lactic acidosis (medicine induced), leading to Acute Tubular Acidosis. Description of lactic acidosis: a condition which occurs when serum lactic acid levels are greater than 2-4 mEq/liter. As the level increases the prognosis worsens.

- Type A lactic acidosis results from poor tissue oxygenation.
- Type B lactic acidosis is seen in conditions such as diabetes, liver or renal failure, neoplasm's and drug intoxication, to include glucophage (merformin).

SUMMARY

Glucophage induced lactic acidosis is rare and has occurred mostly in patients whose kidneys and liver functions were not at normal levels. Lactic acidosis has been reported in about 33,000 patients taking Glucophage (Metformin) over the course of a year. Although rare, lactic acidosis may have a mortality of 50 % in patients who develop it.

SOME OF THE SIGNS OF LACTIC ACIDOSIS ARE AS FOLLOWS:

1. Feeling weak and tired
2. Feeling cold
3. Stomach discomfort
4. Irregular heart rhythm
5. Muscle pain
6. Tired or lightheaded

The patient exhibited three of the above-discussed symptoms upon admission to the hospital.

CONCLUSION

Although the patient's past medical history failed to disclose any prior renal or liver abnormalities she was diagnosed with a lactic acidosis (medicine induced) which led to tubular necrosis causing renal failure.

Due to aggressive treatment with acute hemodialysis, ventilatory support and fluid management; the patients condition resolved and her renal functions return to normal. This case study concludes that aggressive treatment with hemodialysis, good fluid management, and respiratory support were the reasons for the recovery of the renal function.

References

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