

Correspondence

Pheochromocytoma Presenting as Hypertension in Pregnancy

Sir,

We have gone through this interesting article, Pheochromocytoma presenting as hypertension in pregnancy, *JAPI* 2010;58:502-4.¹

We would like to express our views about the article.

It is not clear in the article whether the patient was operated under local, regional or general anaesthesia, what monitoring was done, was any antihypertensive agent used intravenously during surgery? This will give idea about the stormy postoperative course after MTP. Anaesthesiologist plays an important role in management of such patients.

Surgical procedure requiring 30 minutes requires adequate level of anaesthesia and close monitoring. Patient landed in severe hypertension requiring sodium nitroprusside for 24 hours in ICU.

We think patient should have undergone excision of pheochromocytoma before MTP or alongwith MTP. She had 14 weeks amenorrhoea. In early second trimester excision of tumor is the choice of treatment as also quoted by Dr. Londhey and Dr.kulkarni.^{2,3} This could have avoided stormy postoperative course after MTP. Patient had smooth postoperative course after excision of tumor.

References

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Reply from Author

Sir,

We thank Dr. Sadhana Kulkarni for showing interest in our article. Since the patient had 14 weeks amenorrhoea (early second trimester), the gynaecologist had terminated the pregnancy using ethacridine lactate. Hence, no anaesthesia was used for the MTP. Also, no antihypertensive agent was given intravenously. This method of MTP simulates labour and labour itself can increase the risk of hypertensive crisis due to catecholamine release due to uterine contractility and straining. The patient was monitored for pulse, blood pressure and respiratory rate. Therefore hypertensive crisis was detected immediately and treated successfully.

Laparoscopic excision of the tumor was done under general anaesthesia with monitoring of pulse, blood pressure, intra-arterial pressure and central venous pressure. No



antihypertensive agent was given intravenously and the procedure and recovery was uneventful.

We do agree that MTP and tumor excision should have been done simultaneously or tumor excision before MTP but the patient due to personal reasons had initially refused surgical excision of tumor. Only after developing hypertensive crisis she and her relatives realized the severity of her disease and we could counsel them to consent for surgery

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Paralysis Due to Renal Hypokalaemia: An Unusual Presentation of Leptospirosis

Sir,

A 45-year-old male was admitted to this hospital 16 hour after onset of feverishness, myalgia, progressive weakness of all limbs, respiratory muscle weakness and dysphagia. He was conscious, with a pulse of 130/min, BP 110/70 mmHg and tachypnoeic. He had conjunctival suffusion, flaccid hyporeflexic weakness and flexor plantar responses. Sensations were normal and meningeal signs absent. He had abdominal distension with absent bowel sounds.

Arterial blood showed mixed respiratory and metabolic acidosis with a pH of 7.18, pO₂ 50 mmHg, pCO₂ 48 mmHg, oxygen saturation 90% and bicarbonate 16.6 mmol/l. The serum sodium was 141 mmol/l, potassium 2.6 mmol/l, chloride 114 mmol/l and blood urea nitrogen (BUN) 11 mg/dl. He was endotracheally intubated and mechanically ventilated. Following i.v. administration of potassium chloride and correction of hypokalaemia, serum potassium improved to 3.5 mmol/l and the patient was weaned off the ventilator and extubated within 24 hour.

Investigations revealed BUN 20 mg%, serum creatinine 1.1 mg% and normal liver function Total leukocyte count (TLC) and platelet counts within normal limit. The urine pH was 5.21, urine potassium 26.9 mmol/l, urine sodium 22.2 mmol/l, urine chloride 55.8 mmol/l, urine osmolality 293 mOsm/kg and total urine volume 4300 ml. The arterial pH at this time was 7.30 and bicarbonate 17.7 mmol/l. The normal anion-gap metabolic acidosis and transtubular potassium gradient >5 suggested renal tubular dysfunction with potassium wasting.

Forty-eight hours later, the patient developed high fever and oliguria. He now had severe muscle tenderness, icterus and increased conjunctival suffusion. BUN had increased to 87 mg% and serum creatinine to 2.8 mg%. Total bilirubin was 20.6 mg% (direct: 15.3 mg%), SGOT (AST) 190 U/l, SGPT (ALT) 95 U/l, sodium 137.0 mmol/l, potassium 3.5 mmol/l and chloride 109.0 mmol/l. Platelet counts dropped to 40000 and TLC increased to 20000 with 85% polymorphonuclear cells. Abdominal ultrasonography showed normal liver and enlarged kidneys (right: 12.8x4 cm; left: 12.6x4.1 cm). A diagnosis of leptospirosis was considered. He was treated with crystalline penicillin (2MU every 6 hrly) and Ceftriaxone (1gm iv 12 hrly) He recovered

within 07 days without needing dialysis. Leptospirosis was confirmed by the presence in serum of IgM antibodies (1:50) against the heat-stable antigen of *Leptospira biflexa*.

Comment

Renal involvement in the form of interstitial nephritis and tubular necrosis is common in Leptospirosis.^{1,2} Leptospire reside in the proximal tubules,³ which are maximally affected.^{1,2} Yang et al.² showed that the proximal tubular dysfunction may be restricted to the Na⁺-H⁺ antiporter-mediated process. The thick ascending limb of the loop of Henle also could be affected in some patients, especially those with severe jaundice.⁴ The resulting abnormal sodium and chloride transport place an increased load of sodium on the distal tubule, which is relatively unaffected, resulting in impaired reabsorption of potassium.^{1,2} Hypokalaemia may therefore occur, even in patients with significant azotaemia.^{5,6} Though the negative urinary anion gap and urinary pH>5.5 in this patient suggest proximal renal tubular dysfunction, the relatively less negative urinary anion gap points to defective ammonium excretion as well.

In conclusion, Leptospirosis must be considered in patients presenting with fever and symptomatic hypokalaemia, especially in endemic areas

References

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