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Cent. Eur. J. Med. • 3(4) • 2008 • 514-516
DOI: 10.2478/s11536-008-0046-6

Central European Journal of **Medicine**

Hypercalcemia: a rare cause of cerebral infarction

Case Report

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Received 19 March 2008; Accepted 1 June 2008

Abstract: Hypercalcemia is a definite but rare cause of acute pancreatitis. It is often overlooked in the presence of more common etiological factors such as gallstones, alcohol and trauma. Cerebral infarction secondary to hypercalcemia has also been described. We have done a literature review to explore the possible pathological processes causing cerebral infarction in hypercalcemic patients.

Keywords: *Hypercalcemia • Pancreatitis • Stroke*

1. Introduction

We describe a case in which the diagnosis of the cause of acute pancreatitis secondary to hypercalcemia was delayed due to the incidental presence of gallstones. Our patient later developed cerebral infarction, which is a rare complication of hypercalcemia.

2. Case Report

Written informed consent was obtained from the patient for publication of this case report and accompanying images

A 46-year-old female was referred for medical assessment with acute onset of confusion. She was admitted 3 weeks ago in the surgical unit with acute pancreatitis and cholelithiasis. She had been complaining of vomiting and abdominal pain since admission. She had a history of hypertension for which she was on losartan 50mg once daily. She was a smoker and there was no history of alcohol or drug use.

On examination the patient was agitated, confused and dehydrated. She was apyrexial with a normal blood pressure and pulse rate. Neurological examination revealed neck stiffness and the Glasgow Coma Scale (GCS) was E4M6V4. The rest of the clinical examination was unremarkable. Investigations revealed WCC 13.4 (Neutrophils 10.8), CRP of 20 mg/L and a normal capillary blood glucose level. CT brain was normal. She was treated empirically for bacterial meningitis and viral encephalitis. Over the next 12 hours, her condition deteriorated (GCS = E2M4V1) and therefore she was transferred to ITU and intubated. Subsequent lumbar puncture revealed normal CSF. Despite 24 hours of treatment, the patient's condition did not improve.

This was her second admission over the last 2 months with acute pancreatitis. Previous scans had revealed gallstones and that were thought to be the source of her problems. She was awaiting cholecystectomy.

During further investigations in ITU, the patient was found to have calcium of 3.44 mmol/L (reference range 2.10-2.60 mmol/L), phosphate 0.58 mmol/L (reference range 0.80-1.45 mmol/L), and parathyroid hormone level of 122.5 pmol/L (reference range 1.1-6.9 pmol/L). She was diagnosed with severe hypercalcemia secondary to primary hyperparathyroidism and was treated with intravenous fluids and pamidronate (2 doses of 60mg given 6 hours apart). Over the next 5 days the patient's conscious level came back to normal (GCS = E4M6V5). Clinical examination during this period revealed right lower limb hyperreflexia with right-sided extensor planter response. She also had a single episode of generalized seizure during the recovery phase. Repeat CT brain was normal; however subsequent MRI brain revealed an area of diffuse white matter signal change around the occipital horn of the left lateral ventricle consistent with infarction (Figure 1).

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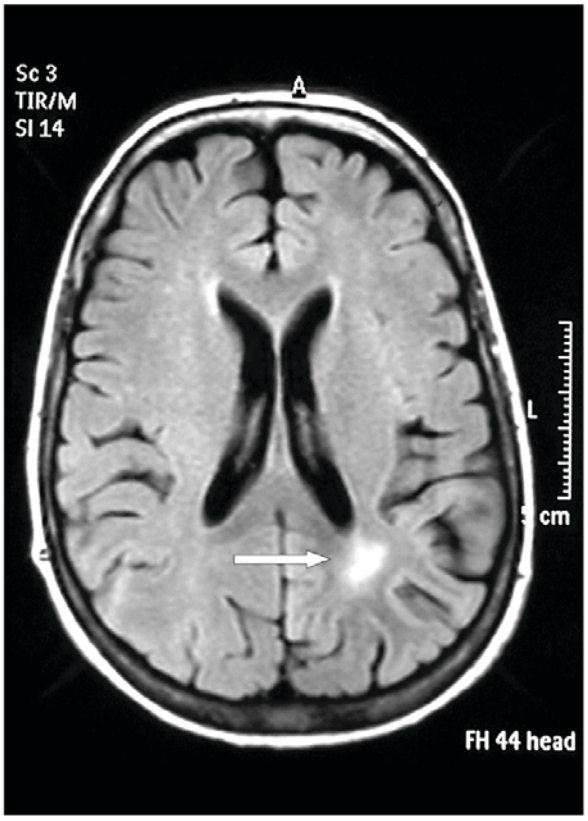
Figure 1. MRI Brain.



The patient was followed up in the outpatient clinic. Her calcium and conscious level have been normal since discharge from the hospital. However she does continue to have some residual right-sided weakness.

3. Discussion

Acute pancreatitis secondary to hypercalcemia is a well-known entity. Despite this it tends to be overlooked. Calcium levels should be part of the confusion screen and should be checked in all patients presenting with acute pancreatitis or abdominal pain.

Neurological manifestations of hypercalcemia include worsening fatigue, depression and confusion and may progress in some cases to stupor and coma. Fasiculations, myoclonus and asterexis are often seen; seizures are infrequent [1]. On examination patients are often hyperreflexic. Electroencephalogram characteristically shows slowing of occipital rhythms and periodic frontal slow waves. All of the above mentioned pathological findings resolve with the correction of serum calcium levels.

Cerebral infarction due to hypercalcaemia has also been described as a rare complication [2]. The exact mechanism is not known. Bartter suggested precipitation of calcium phosphate and other salts in the brain [3], where as Karpati and Frame suggested hypomagnesaemia as an etiological factor [4]. Arterial spasm has also been suggested as a possible cause in cerebral infarction associated with hypercalcemia [5]. Allen *et al.* showed varying degrees of arterial spasm related to differing concentrations of the calcium environment [6]. Edvinsson et al showed that nifedipine (a drug that selectively inhibits calcium influx) reduced the *in vitro* spasm of human pial arteries [7]. Calcium activates several of the factors in the clotting system and the presence of dehydration leads to a hypercoaguable state. These in part may account for occasional reports of cerebral infarction and widespread thrombosis in hypercalcemic patients.

In conclusion, the exact mechanism through which hypercalcemia causes cerebral infarction is still elusive but our review of the literature point to a number of processes, probably working in combination. Hypercalcemia is a potentially treatable cause of confusion and serum calcium levels should be checked routinely as part of the confusion screen. Calcium levels should also be checked in all patients presenting with acute pancreatitis. Whether it should be checked in all patients presenting with abdominal pain, is a matter open for debate. However, in such patients, we should keep a low threshold in requesting this relatively inexpensive test.

References

[1] Fitz T.E., Hallman B.L., Mental changes associated with hyperparathyroidism, Arch. Intern. Med., 1952, 89, 547-51
[2] Bostrom H., Alveryd A., Stroke in Hyperparathyroidism, Acta Med. Scand., 1972, 192, 299-308
[3] Bartter F.C., The parathyroid gland and its relationship to diseases of the nervous system, Res. Nerv. Ment. Dis., 1953, 32, 1-20
[4] Karpati G., Frame B., Neuropsychiatric disorders in primary hyperparathyroidism. Clinical analysis with review of the literature. Arch. Neurol., 1964, 10, 387-97
[5] Walker G.L., Williamson P.M., Ravich R.B., Roche J., Hypercalcemia associated with cerebral vasospasm

causing infarction, J. Neurol. Neurosurg. Psychiatry, 1980, 43, 464-7
[6] Allen G.S., Gross C.J., Henderson L.M., Cerebral artery spasm, J. Neurosurg., 1976, 44, 585-93
[7] Edvinsson L., Brandt L., Andersson K.E., Bengtsson B., Effect of calcium antagonist on experimental constriction of human brain vessels, Surg. Neurol., 1979, 11, 327-30