Adult E-Poster

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VENTRICULAR ARRHYTHMIA POST I-131-METAIODOBENZYLGUANIDINE (MIBG) THERAPY IN AN INOPERABLE RIGHT RETROPERITONEAL PARAGANGLIOMA

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Athirah Nur Amirulhusni, Hidayatil Alimi Keya Nordin, Zanariah Hussein
Institut Endokrin, Hospital Putrajaya, Putrajaya, Malaysia

INTRODUCTION/BACKGROUND

Paraganglioma is a rare neuroendocrine tumour arising from extra-adrenal paraganglia. First line treatment involves surgical resection; however, a proportion of patients present with unresectable tumour. (131)I-MIBG therapy has emerged as a systemic treatment option for inoperable disease. Though rare, MIBG therapy can potentially cause ventricular arrhythmia due to catecholamine surge via tumour cell lysis. This case describes a patient presenting 3 weeks after therapy with ventricular tachycardia.

CASE

A 49-year-old male with underlying hypertension, diabetes mellitus, ischaemic heart disease post-angioplasty and chronic kidney disease was diagnosed with retroperitoneal paraganglioma when he presented with abdominal mass with elevated urine normetanephrine and 3-methoxytyramine. CECT Abdomen revealed a large lesion arising from inferior vena cava (IVC) measuring 9.3 x 9.0 x 13 cm. Biopsy was consistent with moderately differentiated paraganglioma with functional scan evidence of MIBG-avid disease. Multidisciplinary team meeting deemed the tumour to be inoperable in view of huge tumour with high vascular risk, while chemotherapy with temozolomide was not suitable in view of comorbidities. Consensus opted for 131-Metaiodobenzylguanidine (MIBG) therapy. A baseline echocardiography showed good left ventricular systolic function. He received one dose of 200 mCi of I-131 MIBG and was stable throughout admission. However, the patient later presented on day 21 post-therapy with chest pain and palpitation with a heart rate of 225 beats per minute. ECG showed monomorphic ventricular tachycardia which reverted to sinus rhythm with intravenous infusion of amiodarone. A subsequent cardiac MRI reported normal left ventricular function and ejection fraction with no features of myocardial infarction or infiltration.

CONCLUSION

This case highlights a rare but significant complication of I-131 MIBG therapy in the form of ventricular arrhythmia, likely triggered by catecholamine release from tumour lysis manifesting three weeks post therapy. Close cardiac monitoring is essential especially in patients with pre-existing cardiovascular comorbidities undergoing MIBG treatment.

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PARATHYROID ADENOMA WITH PATHOLOGICAL FRACTURE IN YOUNG ADULT: A CASE REPORT

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Vinda Meydina,[1] Eva Decroli,[2] Dinda Aprilia,[2] Alexander Kam,[2] Yanne Pradwi Efendi,[2] Syafril Syahbuddin[2]
[1]Department of Internal Medicine, Faculty of Medicine, Andalas University, M Djamil General Hospital, Padang, Indonesia
[2]Division of Endocrinology, Metabolic, and Diabetes, Department of Internal Medicine, Faculty of Medicine, Andalas University, M Djamil General Hospital, Padang, Indonesia

INTRODUCTION/BACKGROUND

Primary hyperparathyroidism is a condition of hypercalcemia caused by an increase in parathyroid hormone levels. The most common cause of primary hyperparathyroidism is parathyroid adenoma (85%). Primary hyperparathyroidism often goes undiagnosed, particularly in fracture cases, which can lead to complications.

CASE

A 24-year-old female presented to the endocrinology clinic with complaints of worsening back pain and inability to walk over the past three months. The patient had a history of a fall six months prior and was treated in the orthopedic department. MRI of the lumbosacral region revealed multiple thoracolumbar vertebral fractures suspected to be due to osteoporosis. Laboratory tests indicated hypercalcemia (11.4 mg/dl) and hyperparathyroidism (PTH 791 ng/dl). Neck MRI suggested the presence of a left parathyroid adenoma. The patient was diagnosed with primary hyperparathyroidism due to a parathyroid adenoma and underwent parathyroidectomy. Two months after the procedure, her back pain was relieved and daily activity returned to normal. Laboratory test showed the improvement of serum calcium level (8.7 mg/dl).

CONCLUSION

The presence of a pathological fracture in a young adult must raise suspicion for a possible parathyroid adenoma. The diagnosis of primary hyperparathyroidism is established based on clinical findings and laboratory tests of serum calcium and parathyroid hormone levels. Early diagnosis and definitive management of parathyroidectomy are required in patients with primary hyperparathyroidism to reduce complications and improve quality of life.