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MALIGNANT STRUMA OVARII IN PREGNANCY

https://doi.org/10.15605/jafes.037.S2.38

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INTRODUCTION

Struma ovarii is monodermal teratoma predominantly composed of mature thyroid tissue. Thyroid tissue must comprise more than 50% of the overall tissue to be classified as a struma ovarii and it accounts for approximately 2.7-5% of all ovarian teratomas. Depending on the histological features, struma ovarii can be classified as benign or malignant.

CASE

We report a case of a 28-year-old Malay primigravida. She visited the antenatal clinic on her 9th gestational week. Ultrasound of the pelvic incidentally found a right ovarian cyst measuring 9 x 8 cm located above the uterus, which is multiloculated with solid-cystic component. Otherwise, she was asymptomatic at presentation. Patient underwent laparoscopic right ovarian cystectomy on her 13th gestational week. Histopathology examination revealed a mature cystic teratoma, with papillary thyroid carcinoma arising in the background of struma ovarii. She had subclinical hyperthyroidism at early pregnancy, however normalized at her 23rd gestational week. Subsequent thyroid ultrasound was normal.

She successfully delivered a healthy baby at her 38th gestational week. There was no evidence of metastasis based on the computed tomography (CT) scan of the thorax, abdomen, and pelvis. Six weeks after delivery, she underwent laparoscopic right salpingo-oophorectomy with omentectomy and right pelvic lymph node sampling which also showed no evidence of metastasis.

CONCLUSION

Struma ovarii is a rare ovarian tumour. A high index of clinical suspicion along with thorough clinical examination is crucial to diagnose such a tumour. Although benign forms are more common, malignant struma ovarii, mainly papillary thyroid carcinoma have also been reported. Long-term follow up is needed to detect recurrence.

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SEVERE HYPERCALCAEMIA OF HYPERPARATHYROIDISM WITH CARDIAC COMPROMISE; AVOIDING DIALYSIS WITH AGGRESSIVE MEDICAL THERAPY

https://doi.org/10.15605/jafes.037.S2.39

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INTRODUCTION

Severe hypercalcaemia of primary hyperparathyroidism (pHPT) is usually symptomatic and carries high mortality risk due to cardiac arrhythmia and decompensation. Treatment involves vigorous hydration alongside anti-resorptive agents such as bisphosphonate and RANK-Ligand inhibitor i.e., denosumab. Usually, serum calcium of more than 4 mmol/l necessitates dialysis. Here, we report a case of severe hypercalcaemia of hyperparathyroidism with cardiac compromise treated medically resulting to avoidance of dialysis.

CASE

The case is a 50-year-old female with hypertension and chronic kidney disease stage IIIB who was diagnosed with primary hyperparathyroidism since 2020. She was stable with mild hypercalcaemia (calcium less than 3.0 mmol/L). During endocrine follow-up, she complained of constipation, abdominal discomfort, lethargy and vomiting for 2 weeks. She has no cough, no constitutional symptoms, no bone pain, no recent fracture or immobilisation and she denied taking any supplementations. Clinical assessment done was in keeping with severe dehydration.

Blood investigations revealed severe hypercalcaemia (5.01 mmol/L) with normal phosphate and acute azotemia (urea 11, Creatinine 191). Electrocardiography showed first degree heart block, with short QT interval, and a heart rate 60-80 bpm.

Hydration with 5 litres of normal saline and intravenous denosumab was given. Nephrology team was consulted, but no dialysis was planned. On the third day of admission, hydration was increased to 6 litres/day alongside intravenous furosemide to induce forced diuresis. Calcium level reduced to 3.1 mmol/L after a week of admission. Repeated ECG showed resolution of the heart block and short QT. Right inferior parathyroidectomy was done after localisation 2 weeks after. Histopathology confirmed parathyroid adenoma.

CONCLUSION
Severe hypercalcaemia of pHPT can be successfully managed with aggressive treatment and close monitoring. Need for dialysis may be avoided but such patients should undergo parathyroidectomy as soon as possible.

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A CASE OF DENOSUMAB-INDUCE HYPOCALCEMIA:
A SEVERE AND PROLONGED CONSEQUENCES

https://doi.org/10.15605/jafes.037.S2.40

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INTRODUCTION
Denosumab is known to cause abnormalities in calcium homeostasis. The majority of such cases have been described in patients with underlying metastatic cancer, chronic kidney disease or vitamin D deficiency. History of bariatric surgery could also compound the effect of hypocalcemia necessitating intravenous treatment and prolong high dose oral supplementation.

CASE
We present a 61-year-old female with a 6-day history of progressive worsening limb numbness, tingling sensation and intermittent muscle cramps. She had gastric sleeve surgery done 20 years ago. Her regular medication includes calcium, vitamin D and iron supplement. Further history uncovered a denosumab treatment for osteoporosis 1 week ago at a private hospital.

Biochemistry revealed severe hypocalcemia with adjusted calcium of 1.33 mmol/l, mild hypophosphatemia at 0.65 mmol/l, with normal magnesium and renal function. ECG showed prolonged QT interval. PTH level was high at 34.6 pmol/l and 25-OH-vitamin D was insufficient at 33 mmol/l.

She required multiple courses of intravenous calcium gluconate bolus and infusion due to retractable severe hypocalcemia while titrating up her oral supplement in the ward. She was discharged after 8 days with serum calcium around 1.90 mmol/l. At clinic follow up 5 days later, her serum calcium decreased again to 1.64 mmol/l requiring further iv calcium infusion and oral supplement adjustment.

After 2 months, she still requires high dose replacement with 1.5 ug calcitriol twice daily, 1 g calcium carbonate thrice daily and vitamin D3 replacement to maintain normocalcemia.

CONCLUSION
This case report highlights the importance of screening for risk factors for iatrogenic hypocalcemia before initiating denosumab treatment particularly for patients with a history of bariatric surgery. Vitamin D should be adequately replaced prior to treatment and serum calcium levels should be closely monitored post treatment.

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DOSE UP-TITRATION OF EMPAGLIFLOZIN AMONG TYPE 2 DM PATIENTS UNCONTROLLED ON EXISTING ORAL ANTIDIABETIC AGENTS

https://doi.org/10.15605/jafes.037.S2.41

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INTRODUCTION
In most trials involving empagliflozin, the effect on HbA1c reduction was based on concurrent use of 2 doses of the drug without dose titration. This study aims to determine the proportion of patients who need to up-titrate empagliflozin from 10 mg to 25 mg to achieve the desired A1c reduction.

METHODOLOGY
T2DM patients uncontrolled on existing oral glucose-lowering drugs were given empagliflozin 10 mg daily for 3 months. Those who achieved a reduction in HbA1c more than 0.5% from baseline will continue the same dose for another 3 months while those those who had HbA1c reduction of 0.5% or less will be given 25 mg daily for 3 months.

RESULTS
A total of 55 (67.9%) patients had significant HbA1c reduction >0.5% after 3 months on 10 mg empagliflozin (non-titration group), while 26 (32.1%) patients required up-titration of empagliflozin to 25 mg daily for another 3 months (up-titration group). There was no further significant reduction in mean HbA1c from 7.50% (range: 7.1 to 8.15) to 7.45% (range: 6.78 to 8.13), p=0.574 after 3 months of 25 mg empagliflozin. At 3 months therapy with empagliflozin 10 mg, 55 (67.9%) patients achieved mean HbA1c reduction of >0.5% from baseline 7.8%(range: 7.5 to 8.7) to 6.95% (range: 6.53 to 7.38), p<0.001 and remains stable after the continuation for another 3 months.