CONCLUSION

More than half of the participants had good knowledge on hypoglycemia. Continuing refresher education is important for HCP from all departments regardless of seniority.

PP-05

PEPTIDE RECEPTOR RADIONUCLIDE THERAPY INDUCED CARCINOID CRISIS: A CASE REPORT AND REVIEW OF LITERATURE

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INTRODUCTION

Peptide receptor radionuclide therapy (PRRT) is a therapeutic option in inoperable or metastatic neuroendocrine tumors (NET). PRRT is promising in prolonging survival and delaying disease progression in patients with advanced bronchopulmonary carcinoid. However, it may lead to worsening of carcinoid symptoms or even precipitate carcinoid crises.

RESULTS

A 62-year-old man with underlying advanced lung carcinoid tumor developed carcinoid crisis after receiving PRRT. The carcinoid crisis was successfully treated with intravenous octreotide infusion. Several prophylactic measures were taken to prevent PRRT- induced carcinoid crisis. Pre-medications included corticosteroid, a selective 5-HT3 receptor antagonist, parenteral ranitidine and chlorpheniramine for H1 and H2 antagonism, respectively, to prevent the release of the mediators from tumor tissue and/or blocking their effects on target organs. Octreotide infusion was given at 50 µg/hour. Despite measures, he developed carcinoid crisis manifesting as hypotension, tachycardia, multiple episodes of intense diarrhea and flushing at 10 hours post-PRRT. He was immediately resuscitated with crystalloid. Octreotide infusion was increased up to 125 µg/hour. Bridging therapy with long acting somatostatin analogue, lanreotide, was also started. The carcinoid crisis resolved with treatment. Octreotide infusion was tapered by 25 µg hourly and then stopped 24 hours after PRRT.

CONCLUSION

Carcinoid crisis usually occurs during the first PRRT cycle, either during the infusion or 12 to 48 hours after. Acute tumor lysis mediated by radiation cellular damage, resulting in sudden release of supraphysiologic amounts of hormonally active substances, leads to profound carcinoid symptoms. Emotional stress is also contributory. Lastly, administration of amino acids such as lysine and/or arginine as a renal protective measure may play a role in the pathophysiology of PRRT-induced carcinoid crisis, as these may be used as substrates for the synthesis of vasoactive hormones by the carcinoid cells.

PP-06

A CASE OF INSULIN-INDUCED PERIPHERAL NEUROPATHY

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INTRODUCTION

Insulin-induced peripheral neuropathy, known as treatment-induced diabetic neuropathy (TIDN), is an uncommon treatment-induced neuropathic pain and/or autonomic dysfunction that occurs in patients after a rapid improvement in glycaemic control.

RESULTS

We report a patient with underlying type 1 DM who developed TIDN after rapid improvement in glycaemic control following admission for diabetic ketoacidosis. He developed severe neuropathic pain and autonomic dysfunction manifesting as severe postural hypotension resulting in postural giddiness and unsteady gait. He was initially managed as diabetic neuropathic pain. Despite the high dosage of analgesics, pain did not improve, and postural giddiness also persisted. His HbA1C decreased from 17.5% to 7.4% in two months. The diagnosis of TIDN was made after considering the rapid reduction in HbA1c and his clinical presentation of pain and autonomic dysfunction that were not alleviated with the treatment plan for diabetic neuropathy. The patient's insulin dosage was reduced and glycaemic targets were relaxed. Two weeks after the adjustment of medications, his condition improved tremendously.