SAT-LB110

Background: Sulfonylurea poisoning can cause sustained hypoglycemia refractory to intravenous dextrose. Traditional treatment for sulfonylurea induced hypoglycemia includes intravenous dextrose and glucagon as well as diazoxide in refractory cases. Octreotide is recommended for sulfonylurea poisoning in adult and pediatric patients with laboratory evidence of hypoglycemia.

Clinical Case: An 89 year-old female with chronic kidney disease stage III, hypothyroidism, and diabetes mellitus type II, hypertension who presented with intractable nausea and diarrhea. Patient had been taking cefdinir for an UTI the prior week. On CT scan of the abdomen, colitis was demonstrated. Clostridium Difficile Assay was positive. She was incidentally found to have profound hypoglycemia with a blood glucose level of 30 mg/dL. Patient had hypoglycemia unawareness. Despite receiving 4 ampules of dextrose 50%, glucose level did not significantly improve. In the ED, patient was afebrile and hemodynamically stable. Her labs were significant for a hyponatremia of 125 mmol/L with an acute kidney injury [AKI] (Cr 1.94 mg/dL from 1.5 mg/dL). Patient was placed initially on a dextrose 5% normal saline infusion, but glucose levels continued to decline after brief response. Due to poor IV access, internal jugular central line was placed and patient was placed on D10NS infusion with good glycemic response. Patient had taken sulfonylurea despite not eating appropriately for 2 days. After 24 hours on D10 normal saline infusion, patient was able to maintain normal to slightly hyperglycemic levels with consistent carbohydrate diet. Her nausea and diarrhea had considerably improved after starting vancomycin 125 mg every 6 hours. Sulfonyurea was indefinitely discontinued.

Conclusion: Patients presenting with sulfonylurea induced hypoglycemia complicated by poor PO intake, AKI, and infection can be safely treated with supportive measures like proper hydration, and dextrose infusion medication is appropriately metabolized by body without the need for octreotide infusion.

References:Glatstein M, Scolnik D, Bentur Y. Octreotide forthe treatment of sulfonylurea poisoning.Clin Toxicol (Phila). 2012 Nov;50(9):795-804. doi:10.3109/15563650.2012.734626. Epub 2012 Oct 10.

Thyroid

THYROID NEOPLASIA AND CANCER

Aberrant Glycan Expression Changes With the Phenotype of Follicular Thyroid Tumours.

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MON-LB85

ENDO 2020 ABSTRACT**Aberrant glycan expression changes with the phenotype of follicular thyroid tumours.**Lee Kai Yin, MBBS[1], Dinesh Kumar Srinivasan MBBS, PhD[2], Feng Pan MMed[2], Samantha Peiling Yang MBBS, MRCP[3], Rajeev Parameswaran MBBS, FRCS[1,4,].1. Department of Endocrine Surgery, National University Health System, Singapore2. Department of Anatomy, Yong Loo Lin School of Medicine, Singapore3. Department of Endocrinology, National University Health System, Singapore4. Department of Surgery, Yong Loo Lin School of Medicine, Singapore**Abstract:**

Glycosylation is the most common post-translational modification of proteins and plays an important role in cell communication, interaction and adhesion. Aberration of glycosylation is a hallmark of cancer cells and plays an important role in oncogenesis and cancer progression including metastasis[1]. One of the markers of aberrant glycosylation (*O*-linked) is the binding of the lectin *Helix pomatia* agglutinin (HPA), and this has been shown in a wide range of human cancers[2], especially in tumours with a more aggressive phenotype. To study the alteration in cellular glycosylation, detected by lectin *Helix pomatia* agglutinin (HPA) binding in various phenotypes of follicular thyroid tumours, ranging from adenoma to carcinoma and metastatic follicular thyroid cancers. Lectin histochemistry was performed on archival paraffin wax-embedded specimens of 6 follicular adenomas, 10 minimally invasive follicular carcinomas, 13 widely invasive follicular cancers and 4 metastatic follicular thyroid cancers. For positive controls, sections of rat kidney were used, which shows strong and characteristic HPA labelling were included in each labelling experiment. For negative controls, the lectin was omitted and the specificity of binding was confirmed by incubating the sections with HPA in the presence of 0·1-mol/l GalNAc. Sections were scored as positive when 5% or more of the cancer cells labelled positive and scored as negative when less than 5% labelled positive for HPA binding. Assessments of HPA binding were performed by two observers who were blinded to the identity of the sample, and their results were compared. Positive labelling was seen in 20% of adenomas, 60% of minimally invasive carcinomas, 77% of widely invasive carcinomas and 100% of metastatic carcinomas ($p<0.05$). In the minimally invasive carcinoma group, all tumours that showed vascular invasion showed positive HPA labelling, whereas only 20% of patients with capsular invasion showed positivity. We speculate that as the phenotype of the thyroid tumours changes from benign to the malignant phenotype, the glycan expressions change. However, the underlying molecular mechanisms leading to this change needs to be further investigated.

References:1. Magalhães, A., Duarte, H.O. & Reis, C.A. 2017, "Aberrant Glycosylation in Cancer: A Novel Molecular Mechanism Controlling Metastasis", *Cancer Cell,* vol. 31, no. 6, pp. 733-735.2. Parameswaran, R., Sadler, G. & Brooks, S. 2011, "Helix pomatia Agglutinin Binding Glycoproteins in Thyroid Tumors", *World Journal of Surgery,* vol. 35, no. 10, pp. 2219-2227.3. Parameswaran, R., Tan, W.B., Nga, M.E., Soon, G.S.T., Ngiam, K.Y., Brooks, S.A., Sadler, G.P. & Mihai, R. 2018, "Binding of aberrant glycoproteins recognizable by Helix pomatia agglutinin in adrenal cancers", *BJS open,* vol. 2, no. 5, pp. 353-359.