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Treatment of Pasireotide LAR-Associated Hyperglycemia In A Patient With Acromegaly

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Abstract #937

TREATMENT OF PASIREOTIDE LAR-ASSOCIATED HYPERGLYCEMIA IN A PATIENT WITH ACROMEGALY

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Objective: Hyperglycemia was noted in pasireotide long-acting release (PAS LAR)–treated patients with acromegaly during clinical trials. We describe a clinical approach to hyperglycemia in a patient with acromegaly and diabetes mellitus type 2 (DM2) during PAS LAR initiation and treatment.

Methods: Clinical response to PAS LAR, including self-monitored blood glucose (SMBG) levels, HbA1c, IGF-1, and adverse effects, was reported.

Case Presentation: A 44-year-old male was referred for consultation in 2007 owing to uncontrolled DM2 despite maximum metformin (MET) dose. Testing led to a biochemical diagnosis of acromegaly. An 8-mm pituitary adenoma was seen on MRI. Transsphenoidal pituitary adenoma resection was performed, but residual tumor remained. Postoperative IGF-1 levels were 394 ng/mL (age-normalized range, 75-216 ng/mL). Octreotide (OCT) therapy was initiated, switched to OCT LAR 20 mg monthly, increased to 40 mg monthly, and twice-weekly cabergoline (CAB) 0.5 mg was added; none of these treatments normalized IGF-1 or resolved symptoms. Change to either lanreotide or pegvisomant (PEG), combined with CAB, was also unsuccessful; IGF-1 levels were 364 ng/mL (age-normalized range, 61-200 ng/mL), FPG 174 mg/dL, and HbA1c 7.3%. Metformin was initially decreased postoperatively then re-optimized. Before switching from PEG to PAS LAR, SMBG frequency increased. Within 24 hours of PAS LAR initiation (and continuation of CAB), glucose levels increased to 200 to 300 mg/dL. Liraglutide (LIRA) was added, although severe nausea slowed titration, and glimepiride (GLIM) was added. After 6 weeks of PAS LAR, LIRA, GLIM, and MET, IGF-1 levels were reduced to 239 ng/mL, whereas glucose and HbA1c increased to 265 mg/dL and 7.9%, respectively. Frequency of SMBG remained consistent. Severe nausea improved upon increase to GLIM 8 mg daily and switch from LIRA to dulaglutide. Glucose levels slowly dropped to 150 to 160 mg/dL. Three months after PAS LAR initiation, IGF-1, glucose, and HbA1c were 274 ng/mL, 172 mg/dL, and 7.9%, respectively.

Discussion: PAS LAR resulted in IGF-1 reduction. Significant hyperglycemia occurred immediately after initiation and required rapid changes to DM2 medication. Following recommendations to increase SMBG, frequent medication adjustment, and addition of GLP-1 analogue in response to hyperglycemia returned SMBG levels to baseline, although HbA1c was predictably unchanged at 3 months.

Conclusion: Hyperglycemia is an expected adverse effect of PAS LAR. Vigilant SMBG and rapid response to hyperglycemia using GLP-1 analogues can be an effective option for treating patients with acromegaly and DM2 who are receiving PAS LAR, although more study is warranted.