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Bowel Obstruction in Patients with Neuroendocrine Tumor (NET) Treated With ^{177}Lu-DOTATATE: A Comparative Cohort and Time-to-Event Analysis

Alvaro A. Ordonez[1], Abigail Pepin[1], Neil K. Taunk[1], Jennifer R. Eads[1], Philipose G. Mulugeta[1]

[1]Hospital of the University of Pennsylvania

BACKGROUND

Previous studies have suggested that peptide receptor radionuclide therapy (PRRT) with ^{177}Lu-DOTATATE may increase the risk of bowel obstruction (BO). This may particularly happen in patients with mesenteric or peritoneal disease, possibly due to radiation-induced inflammation and exacerbation of desmoplastic fibrosis. However, most studies lacked non-PRRT control groups, limiting the ability to distinguish PRRT effects from the natural history of advanced disease or known risks of BO from prior bowel surgery. We compared BO rates between patients with NET who received PRRT and those who did not, and identified independent risk factors using multivariate and time-to-event analyses.

METHODS

We conducted an Institutional Review Board-approved retrospective cohort study of 492 patients with NET, including 211 treated with PRRT and 281 without PRRT. BO occurrence, timing relative to PRRT, and clinical covariates (including pre-treatment bowel resection, peritoneal carcinomatosis, liver metastases, and primary tumor site) were analyzed. BO events were cross-referenced with PRRT dosing dates to identify episodes occurring within 3 months of treatment. Multivariate logistic regression evaluated factors associated with BO. Because the PRRT cohort had longer follow-up (median 9.1 vs 6.7 years, $p<0.001$), sensitivity analyses included incidence rate comparisons, Kaplan–Meier estimation, and Cox proportional hazards modeling.

RESULTS

Crude BO rates were higher in the PRRT group (30.3% [64/211] vs 22.1% [62/281]; OR 1.54, 95% CI 1.02–2.31, p=0.047). However, PRRT patients had significantly more advanced disease, including higher rates of peritoneal carcinomatosis (31.8% vs 13.9%), liver metastases (100% vs 61.2%), and stage IV disease (92.9% vs 72.2%). Among PRRT patients with BO, 89.1% (57/64) had events before PRRT initiation. Only 5 patients (2.4%) developed BO within 3 months of treatment, while the remaining post-PRRT events occurred a median of 15 months later. On multivariate logistic regression, PRRT was not an independent predictor of BO (OR 1.02, 95% CI 0.49–2.11, p=0.97), whereas prior bowel

resection (OR 7.14, p<0.001) and peritoneal carcinomatosis (OR 4.17, p<0.001) were significant. Similarly, Cox regression confirmed PRRT was not associated with increased BO risk (HR 1.28, 95% CI 0.80–2.04, p=0.30), while bowel resection (HR 5.27, p<0.001) and peritoneal carcinomatosis (HR 2.51, p<0.001) remained independent predictors.

CONCLUSIONS

After multivariable adjustment, PRRT was not an independent risk factor for BO. Prior bowel resection and peritoneal carcinomatosis are the dominant predictors for BO in NET. Previously reported PRRT–BO associations may reflect confounding by indication rather than a direct treatment effect.

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