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Chronic diarrhea: More than just bugs and drugs

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Amyloidois is the term for a group of protein folding disorders characterized by the extracellular deposition of insoluble polymeric protein fibrils in tissues and organs. Amyloidosis is commonly systemic, occasionally organ-limited, and rarely a solitary localized mass. This latter presentation is commonly referred to as tumoral amyloidosis. Although reports exists of these often called "amyloidomas" showing up in almost every tissue/organ, the GI tract has a prevalence that is not well document making it an outstanding diagnostic challenge. A 66 year-old male with a history of IV drug abuse, comes to our hospital to be evaluated due to diarrhea that started 2 months ago; 3-5 depositions a day, watery in consistency no blood or mucus, associated with epigastric abdominal pain described as "burning" in nature, 7/10 intensity without radiation and a 50 pound weight loss. Denied fever, chills, shortness of breath, nausea or vomiting; symptoms were non consistently worsened with food ingestion and did not improve with OTCmedication for diarrhea. PMH was significant for HCV diagnosed 1 month ago. On physical exam the abdomen showed a prominent liver edge 5cm below the costal margin non tender, non-distended without ascites. Laboratory work-up upon admission showed metabolic acidosis, acute kidney injury, no electrolyte disturbances and an elevated TSH. Esophagogastroduodenoscopy (EGD) done the day after admission showed an esophagus that was normal and a friable mass in the antrum of the stomach that bled on contact, cold forceps biopsy was taken. A colonoscopy was also performed, with unremarkable findings; random biopsies taken. Pathology reports a tissue that on red Congo stain has apple-green birefringence indicative of amyloid fibrils in both colonic and gastric samples. The deposition of amyloid fibrils in other organs were sought out with negative results; thus giving the impression of single system involvement. Gastrointestinal amyloidosis causes severe malabsorption due to the deposition of the protein fibrils, explaining the patient's chronic diarrhea and significant weight loss. Since patient's malabsorption caused wasting and malnourishment, total parenteral nutrition was indicated while the patient received chemotherapy for the treatment of amyloidosis. This case illustrates that there is an important risk of misunderstanding and diagnosis delay of patients that present with malabsorption. Even if the clinical symptoms are not obvious upon initial presentation, the hypothesis of gastrointestinal amyloidosis should be considered among the possible diagnosis of patients with chronic diarrhea and weight loss. In doing so, quality of life as well as morbidity improvement should be evident

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PDZK1 targets PTEN to inhibit AKT signaling and malignant phenotypes in gastric cancer

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PI3K/AKT pathway, which is frequently altered in gastric carcinoma, can be negatively regulated via dephosphorylation of PIP3 to PIP2 by (Phosphatase and Tensin Homolog) PTEN. In the present study, PDZK1 was identified as a novel binding protein of PTEN, in which the interaction was mediated by the PDZ2 and PDZ3 domain of PDZK1 with the last four amino acids (ITKV) in the carboxyl terminus of PTEN (PTEN-CT). Our data from PDZK1 over-expression and siRNA-mediated knock-down experiments further demonstrated that by associating with PTEN, PDZK1 inhibits the phosphorylation of PTEN. In addition, over-expression of PDZK1 down-regulated AKT and ERK signals. Consistent with these results, PDZK1 suppressed gastric cancer cell proliferation, impeded the formation of anchorage independent colonies in soft agar and retarded the growth of xenografts in nude mice. Furthermore, PDZK1 was significantly downregulated in gastric cancer tissues in comparison to that in normal gastric tissues. Collectively, this study shows that down-regulation of PDZK1 expression enhances the PTEN inactivation, which may contribute to the carcinogenesis of gastric cancer.

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