

Answering Reviewers

Reviewer 1: the manuscript entitled "Primary Intestinal Lymphangiectasia presenting as limb Convulsions: a case report" represent a brief report with sufficient discussion. The title reflects the main subject but similar titles are present in literature. The introduction section is very brief and requires more specific information. the conclusion of the study is not specific and seems like results. keywords are also not specific. incomplete information has been given at several points such as :patient was healthy in past" but no information for how long. Different figures can be grouped together. figure legends needs brief heading as given in figure 2. more pictorial representation is required

Reply: Thanks for your positive comments. The clinical symptoms of PIL that we have found are mostly edema of lower limbs, and the cases manifested by numbness and convulsion of limbs are rare, so we choose "Primary Intestinal Lymphangiectasia presenting as limb Convulsions: a case report" as the topic. We added keywords and supplement the patient's previous physical condition. We also changed the statement content of the conclusion and changed figure heading in order to make it more brief.

Reviewer 2: This is a case report of a man presenting with limb convulsions and with laboratory markers of hypoproteinemia, hypoalbuminemia, hypocalcemia, hypomagnesemia, lymphopenia. Further investigation revealed increased parathyroid levels, decreased vit D, hypoinnoglobulinemia and positive fecal occult blood test in order to follow a further exploration. The presence of hypocalcemia and hypomagnesemia it seems to be compatible with the patient's presenting symptoms as it is verified by the remission after supplemented with calcium gluconate and potassium magnesium aspartate. These findings would probably generated the parathyroidism due to increased parathyroid hormone and a hypo echogenic nodule in the gland. The hypoalbuminemia, hypoproteinemia and hypocalcemia made the impression that there might be a kidney dysfunction with urine protein loss and secondary hyperparathyroidism but this kidney injury was not verified by kidney function tests and increased parathyroid hormone returned to normal after correction of hypocalcemia, pointing to a secondary cause for hyperparathyroidism. In that case, if there is also no hepatic dysfunction, one should focus on protein losing enteropathy which includes a variety of possible causes that need to be examined. For the differential diagnosis at this point one should include a gastroscopy and colonoscopy, possibly antibodies for celiac disease, $\beta 2$ microglobulin for hematopoietic tumors, assays for giardia, acid Schiff for Whipple, a cardiac triplex to check for pericardial effusion, an MRI enterography to check for possible small bowel pathology etc with prioritization depending on the results of the ensuing laboratory examination and vit D assays and the imaging of the parathyroid gland which set forward a possible hyperparathyroidism. Instead there are inaccurate reports for " blood IBD" ? or "stool IBD" ? screening (maybe calprotectin?) and for MRI examination of an unspecified anatomy. There is no proper justification for the parathyroid nodule. Furthermore there is no temporal determination of the second battery of laboratory tests in the line of events during patient's exploration. In text there is no description of the endoscopic findings but from the pictures it seems that there are scattered white spots on the mucosa. Biopsies showed that these are compatible with lymphatic dilatation but this is a conclusion that follows biopsies. Finally there

should have been a sequence where by excluding IBD, celiac, infections, lymphoma and rheumatic diseases and by showing compatible endoscopic, pathologic and further laboratory clues (like CD4 lymphopenia, lack of fat soluble vitamins, hypoglobulinemia etc) one would have been driven to the most appropriate diagnosis which is primary intestinal lymphangiectasia which is rare in adult population with no apparent symptoms of leg edema or diarrhea. In conclusion there should be a more appropriate presentation showing the differential diagnosis and the mentality that provoked the medical actions as well as the sequence of patient's handling by the responsible physician. There is repetition in the first and second paragraph of Discussion section of aforementioned text. There are abbreviations not properly explained as well as syntax and grammatical errors scattered in text. Furthermore one could point out the rarity of this clinical condition, the wide array of possible diagnosis which are relatively not straight forward and the necessity for vigilance even when the symptomatology is subtle.

Reply: Thanks for your positive comments. Our case presented with convulsions of the limbs, followed by hypocalcemia, hypokalemia, hypoproteinemia, and high PTH. PTH decreased after calcium and potassium supplementation. Pseudohypoparathyroidism, a common disease of the endocrine system, was first ruled out and parathyroid nodules were confirmed to be nonfunctional. And then we excluded kidney, liver and other diseases that might cause protein loss, protein production reduction. Finally, our diagnostic approach is targeted at protein loss intestinal disease. Endoscopic pathology confirmed the final diagnosis. We revised the first and second paragraphs of the discussion section to eliminate duplication and reorganize them in the order of differential diagnosis. Then we fixed the grammar errors in the article.