■ Sun-407 ■

A case of localized GI tract biclonal AL amyloidosis

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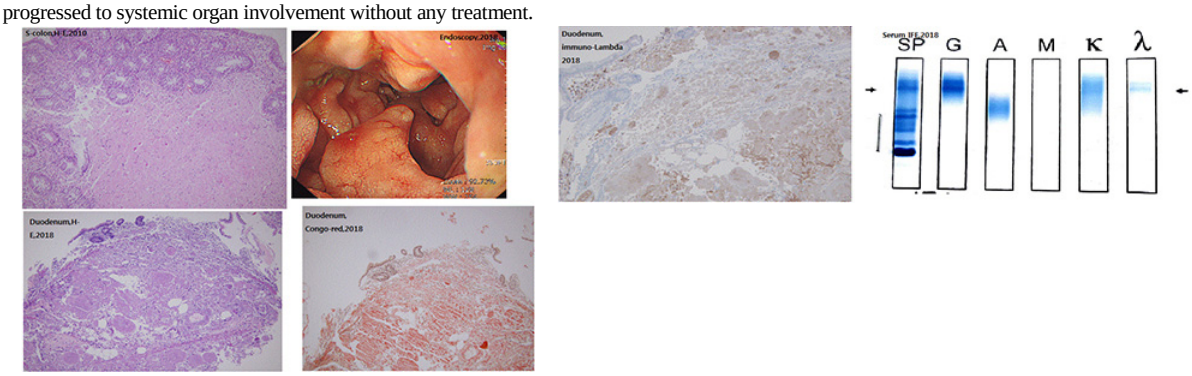
Amyloidosis is a term that the tissue deposition of insoluble fibrils composed of low molecular weight subunits of a variety of normal serum proteins on tissue and organs. The classification of amyloidosis is based on amyloid fibril proteins. AL amyloidosis in which the fibrils are composed of fragments of monoclonal light chains. AL amyloidosis is a generally systemic disorder and the diagnostic criteria contains the plasma cell dyscrasia. But here is the patient who was diagnosed with the localized AL amyloidosis on GI tract with biclonal gammopathy, even though the GI tract is not a common site in localized AL amyloidosis. The patient had diagnosed with AL amyloidosis on sigmoid polyp biopsy and denied further evaluation and treatment in 2010. On June 2018, he revisited emergent room due to epigastric pain and diagnosed with acute cholecystitis. He was treated with percutaneous cholecystostomy. On abdominal CT scan, numerous polypoid lesions were found at duodenum and small bowel. On Upper GI endoscopy, variable sized polypoid lesions and ulcerofungating mass were found at gastric antrum and duodenum. All of the biopsy specimens were revealed with lambda positive amyloid deposits. But serum immunofixation electrophoresis revealed biclonal gammopathy, two distinted band on Ig G and lambda chain. On bone marrow exam, there was no increased or abnormal plasma cell. The serum kappa/lambda ratio was in normal range. Echocardiography, EKG, liver function test, urine analysis revealed no significant abnormality. So the patient was diagnosed with localized GI AL amyloidosis. This is the case of the long lasting AL amyloidosis that have not progressed to systemic organ involvement without any treatment.



■ Sun-408 ■

Ifosfamide-Induced Encephalopathy and Nephrotoxicity in a Patient With Metastatic Osteosarcoma

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Ifosfamide is an antineoplastic, alkylating agent used to treat various malignancies, but it can cause severe toxicities including neurotoxicity and nephrotoxicity. Ifosfamide induced neuropsychiatric conditions are mostly reversible, but long-term complications like coma have been reported. Nephrotixicity presents mainly with tubular dysfunction such as Fanconi syndrome, renal tubular acidosis, and nephrogenic diabetes insipidus. We report a 49-year-old woman with metastatic osteosarcoma who was treated with high-dose ifosfamide for only 1 cycle (2000 mg/m^2 of ifosfamide on day 1 followed by continuous infusion of 2000 mg/m2/d from days 1 to 4) after treatment with adriamycin and cisplatin in 20%-reduced doses for 6 cycles (total cumulative dose of adriamycin is 288 mg/m^2 and that of cisplatin is 480 mg/m^2) when she suffered from an unexpected development of central nervous system toxicity and kidney dysfunction. During the treatment, especially on days 3 and 4, the patient tended to be tired and slept more without any psychosis. On day 5, she was confused. She laughed without reason and spoke incoherently with sluggish speech. On day 6, she was drowsy and barely responded to verbal stimulation. We scanned her brain using CT and MRI, which showed no abnormal findings. Cerebrospinal fluid laboratory tests was normal findings. However, laboratory findings showed markedly aggravated azotemia with hypokalemia and metabolic acidosis which was type 2 renal tubular acidosis. We increased bicarbonate and potassium levels to compensate for renal tubular loss. On day 10, the patient was alert and oriented suddenly and spontaneously. After that, Kidney function was somewhat improved and sustained (figure1). She refused further chemotherapy and proceeded to receive palliative care without chemotherapy. As it is impossible to prevent ifosfamide-related adverse effects, we recommend careful watching during and after chemotherapy. As with our patient, treatment with ifosfamide of patients exposed to cisplatin requires great care. If unexpected adverse effects occur, immediate discontinuation of ifosfamide treatment is strongly recommended.