FP005 **RENAL PRESENTATION OF PRIMARY SJöGREN'S SYNDROME: HYPOKALEMIC PARALYSIS WITH TYPE 1 DISTAL RENAL TUBULAR ACIDOSIS**

Arina Yespotayeva[3], Kairat Kabulbayev[5], Assiya Kanatbayeva[1], Olga Mashkunova[2], Elvira Turarova[2], Salim Berkinbayev[4], Aigerim Botabekova[6]

[1]Nephrology, Asfendiyarov"TM"s Kazakh National Medical University, Almaty, Kazakhstan, [2]Rheumatology, Asfendiyarov"TM"s Kazakh National Medical University, Almaty, Kazakhstan, [3]Nephrology, Scientific Research Institute of Cardiology and Internal Diseases, Almaty, Kazakhstan, [4]Cardiology, Scientific-Research Institute of Cardiology and Internal Diseases, Almaty, Kazakhstan, [5]Nephrology, Scientific-Research Institute of Cardiology and Internal Diseases, Almaty, Kazakhstan and [6]Rheumatology, Scientific-Research Institute of Cardiology and Internal Diseases, Almaty, Kazakhstan

INTRODUCTION AND AIMS: Sjögren's syndrome is an autoimmune disease with glandular and extraglandular manifestations. Tubulointerstitial nephritis (TIN) is the main renal involvement associated with primary Sjögren syndrome (pSS). TIN can manifest as distal renal tubular acidosis (RTA), nephrogenic diabetes insipidus, proximal tubular dysfunction, and others [1], of which RTA is the main clinical presentation [2]. RTA has been reported in 4.3 to 9% of pSS patients; it is more common in middle-aged women, and two-thirds of them will develop symptoms [2, 3].

METHODS: In the present report, we aimed to present a 42-year-old female patient admitted with paralysis to hypokalemia, who was later diagnosed with distal type 1 renal tubular acidosis (RTA) secondary to Sjogren's syndrome (SS).

RESULTS: Case reportWe report a 42-year-old woman who presented with chronic paralysis in the upper and lower extremities, dryness and redness in the eyes, dry mouth, severe hypokalemia. She has severe intermittent fatigue and muscle weakness for the past 1 year. Detailed history revealed that she has been complaining of dry eye and mouth for the last 6 months, 11 months ago was diagnosed Guillain-Barre syndrome and hypokaliemia (1.2 mmol/l) The patient was sent to our clinic for diagnosis and further treatment. On physical examination, BP was 110/70 mm Hg, pulse was 76/min and rhythmic. Laboratory tests revealed severe hypokalaemia (2.1mmol/l), hyperchloremic metabolic acidosis with compensatory hyperventilation (arterial pH 7.2; Cl-118 mEq/L, PCO2 49.9 mmHg), normocytic normochromic anaemia (Hb 10.5 g/dL, MCV 84 fL), and BUN 4.58 mmol/l, creatinine levels 85 mmol/l, (GFR 72.8 ml/min/1.73 m2 CKD-Epi). The tubular defect was accompanied by mild hypocalcemia (total calcium of 1.10 mmol/l, ionized calcium of 1.22 mmoL/L; normal 1.15-1.32 mM/L). On subsequent days, sustained hypokalemia (< 3.5 mEq/L), with metabolic acidosis and alkaline blood pH (> 7.5) suggested RTA1. The pSS diagnostic findings included positive antibodies to the ribonucleoprotein antigen La (SS-B) and Ro (SS-A), antinuclear antibody (ANA) 1:342 with granulated pattern, and negative results for rheumatoid arthritis (RA) test, anti-dsDNA, anti-Sm, anti-SCL70 and anti-centromerase antibodies. With all lab results, a distal RTA (dRTA) diagnosis due to pSS was made. Hypokalemia and metabolic acidosis were treated with intravenous sodium bicarbonate, asparcam 2 tablets 3 times a day - under the control of electrolytes in the blood.The patient was discharged and we will follow her up in our clinic every six months.

CONCLUSIONS: Sodium bicarbonate may be equally effective for the therapy of RTA1, however, it is associated with side effects such as gas formation of CO2 in the stomach. Nonetheless, treatment should be aimed at the immunological disease and work to decrease the lymphocytic infiltration of the exocrine glands and extra-glandular tissues, at least, partially to correct the acidification defect, improve bone metabolism and improve overall length and quality of life of primary SS patients.