Case Report
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Nephrotic syndrome and acute polyradiculoneuropathy

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Introduction

Guillain-Barré syndrome includes several acute polyradiculoneuropathies with sensitive or motor symptoms (or both), cranial nerves palsy or autonomic dysfunction. They can be cause because of axonal o demyelinating damage.

In the other hand, nephrotic syndrome is the combination of nephrotic-range proteinuria with a low serum albumin level and edema. Both diseases are suppose to have an autoinmune etiology although relation between them is in frequent.

Case report

We report a case of a 57years old male, right handed, no medical allergies and medical history of hypertension, dislypidemia and paroxismal atrial fibrilation. He was admitted to our hospital because of chest pain with ST elevation. He was performed a coronariography with a stent placed in left anterior descending coronary artery. The patient remained stable during several weeks until he delevoped symptoms of respiratory infection with normal chest Xray and normal blood tests. He was started on empiric antibiotics and discharged home with double antiplatelet therapy, warfarine, amlodipine and bisoprolol. Two weeks after being discharged, the patient went to ER because he had numbness and feeling of pins and needles in both feet, these symptoms had increasing in the last days up to both arms also. He had also developed weakness in legs and hands, feeling unable to walk without help.

When he was admitted again temperature and blood pressure were normal as well as oxygen levels and cardiac frequency. Urgent blood test were normal and CT brain scan as well.

The neurological examination showed normal cranial nerves, distal hypoesthesia in legs and arms, no vibratory sensitivity in legs, bilateral weakness in feet extension, quadriceps and extrinsic hand muscle. Deep tendon reflexes were all absent at that moment.

A lumbar punction was done after reversing warfarine effect. CSF was clear, with high protein levels (54 mg/dl), normal glucose levels and no cells.

Blood test with serologies, antinuclear autoantibodies, crioglobulines, thyroid and liver-kidney test were unremarkable although anti GM2 antibody were detected in blood test. The eletroneurogram was normal (it was done two weeks after the symptoms started).

With the diagnosis of acute polyradiculoneuropathy he was started on a treatment with human inmunoglobulines (0,4mg/kg/day during 5days). He got better after finishing the treatment, being able to walk without help, he also got lower numbness feeling in feet and hands.

One week after finishing that treatment, the patient developed edema in both legs and low diuresis. An urgent blood test showed abnormal kidney levels (urea 83mg/dl, creatinine 1,13mg/dl and glomerual filtration rate of 56ml/min). Urgent urine test also showed nephrotic-range proteinuria (more tan 500mg/dl) and hyaline-granulous casts. With these results, our patients was diagnosed of nephrotic syndrome so a corticostheroid treatment was started (1gr of metilprednisolone per day during 5days). He had a complete recovery of kidney function after that, and also edema dissapeared. Abdomen ultrasound exam was normal and kidney biopsy was not done because of the recovery of the patient. The patient was discharged without any disability.

Guillain-Barré syndrome includes several acute polyradiculoneuropathies that have an inmune ethiology (usually antibodies again as viral o bacterial infections, vaccines, surgery or even cancer).[1] Brighton criteria are used for its diagnosis and are based on symptoms, CSF and electroneurogram.[2]

Nephrotic syndrome had high proteinuria levels in urine, edema and low albumine levels. Although its ethiology is yet unclear, it seems that glomerular membrane permeability to albumine and some other proteins is broken.[3] It has been related to metabolic diseases like diabetes, autoinmune diseases, cancer, viral or bacterial infections ore some treatments.

Nephrotic syndrome and SGB have been related, mainly with membranous nephritis, focal nephrosclerosis and minimal change disease.[4] A common antigen can be the cause of both pathologies. Although, some mutations in INF2 gen (in Schwan cells and also in podocytes) have been described in people with hereditary neuropathies and glomerulonephritis.[5]

Some components of the human inmunoglobulines such as sorbitol, sucrose or sodium can increased the risk of nephritic syndrome in patients with previous kidney disease.[6]

Acknowledgments

None.

Conflicts of interest

The author declares that there is no conflicts of interest.

References

1. Sejvar JJ, Kohl KS, Gidudu J, et al. Brighton Collaboration GBS Working Group. Guillain-Barré syndrome and Fisher syndrome: case definitions and guidelines for collection, analysis, and presentation of immunization safety data. *Vaccine*. 2011;29(3):599–612.
2. Fokke C, van den Berg B, Drenthen J, et al. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. *Brain*. 2014;137(Pt 1):33–43.
3. Kodner Ch. Diagnosis and Management of Nephrotic Syndrome in Adults. *Am Fam Physician*. 2016;93(6):479–485.
4. Quek AM, Soon D, Chan YC, et al. Acute–onset chronic inflammatory demyelinating polyneuropathy with focal segmental glomerulosclerosis. *J Neurol Sci*. 2014;341(1–2):139–143.
5. Boyer O, Fabien Nevo, Emmanuelle Plaisier, et al. INF2 mutations in Charcot–Marie–Tooth disease with glomerulopathy. *N Engl J Med*. 2011;365(25):2377–2388.
6. Palabrica FR, Kwong S, Padua F. Adverse events of intravenous immunoglobulin infusions: a ten–year retrospective study. *Asia Pac Allergy*. 2013;3(4):249–256.

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