Antineutrophil Cytoplasmic Autoantibody-Associated Glomerulonephritis as a Complication of Home Parenteral Nutrition

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PR3-ANCA-associated glomerulonephritis · Home parenteral nutrition · Chronic bacteremia

Abstract
Patients on long-term home parenteral nutrition (HPN) occasionally develop glomerulonephritis due to chronic central venous catheter (CVC)-related infection. Most previously reported cases were membranoproliferative glomerulonephritis (MPGN). This is a case report of a 16-year-old girl receiving HPN for short bowel syndrome. After 11 years on HPN, she developed acute kidney injury with macroscopic hematuria, nephrotic-range proteinuria, and a reduced glomerular filtration rate (GFR). Initially, MPGN associated with chronic bacteremia was suspected with the assumption that the condition would be treated with antibiotics and CVC replacement. However, her kidney biopsy revealed antineutrophil cytoplasmic autoantibody (ANCA)-associated glomerulonephritis (AAG). This was consistent with the fact that the patient tested positive for proteinase 3-ANCA. Immunosuppressive therapy with methylprednisolone pulses (followed by oral prednisone) and rituximab led to remission. Her GFR and protein excretion returned to normal. Chronic bacteremia as a complication of long-term HPN may cause various types of glomerulonephritis including, rarely, AAG requiring immunosuppressive therapy.
Introduction

In recent decades, the number of patients with short bowel syndrome (SBS) has increased. Pediatric SBS is most commonly caused by congenital anomalies of the gastrointestinal tract (intestinal malrotation and volvulus, multiple intestinal atresia, total colonic Hirschprung disease, gastroschisis) and surgical resection for necrotizing enterocolitis or inflammatory bowel disease. At present, these patients may benefit from a new treatment approach, total parenteral nutrition, often in the form of home parenteral nutrition (HPN). Its administration is rather challenging and requires cooperation of patients and their caregivers. Various HPN regimens are available, often taking several hours a day. Aseptic techniques are vital and infusion pump operation must be learned. During the day, however, these individuals may participate in normal activities. With HPN, active life in one’s social environment is possible. But administration of nutrition into the central venous system poses a risk for complications with sepsis or thrombosis. Chronic bacteremia may lead to infective endocarditis or glomerulonephritis. The cases of membranoproliferative glomerulonephritis (MPGN) in HPN patients have been published. Reported here is a rare, previously unpublished case of antineutrophil cytoplasmic autoantibody (ANCA)-associated glomerulonephritis (AAG) in an HPN-treated girl.

Case Report/Case Presentation

A 16-year-old girl with SBS who had been on HPN for 11 years presented to a gastroenterology outpatient unit for a regular checkup. The examination revealed severe anemia (hemoglobin 70 g/L) and acute kidney injury of unknown etiology (serum creatinine 97 μmol/L, urea 20 mmol/L). Therefore, the girl was admitted to hospital for further investigations and treatment. When she was five, she suffered from sudden abdominal pain due to volvulus of the small intestine, causing ischemic damage and necrosis of the entire small intestine and half of the colon. Repeated revision surgeries were needed, that resulted in anastomosis of the duodenum and transverse colon. Temporarily, the patient had prerenal acute kidney injury, but her glomerular filtration rate (GFR) returned to normal. Subsequently, the girl was given daily parenteral nutrition via a central venous catheter (CVC). Her oral intake was minimal, limited to fluids. Initially, she received parenteral nutrition in the hospital. Later, her mother, a nurse, was trained to administer it intravenously via the CVC so that the girl could be provided HPN every night. She was followed in the gastroenterology outpatient unit for long-term parenteral nutrition. Over the 11 years, her condition was complicated by two attacks of acute pancreatitis and catheter-related sepsis on eight occasions (causative agents *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Staphylococcus saprophyticus*, *Acinetobacter pittii*, *Acinetobacter ursingii*), of which four required a CVC change. Two months before presentation, the girl was examined in a nephrology outpatient unit for mild proteinuria and mild microscopic hematuria. She was found to have a normal GFR and only slightly decreased hemoglobin level (116 g/L). The urinary findings were assumed to be caused by higher oxaluria and kidney damage following previous repeated sepsis events.

On admission for severe anemia and kidney damage, the overall clinical condition of the girl was unremarkable. She was afebrile, with no apparent signs of acute infection and no dysuria; her urine excretion was normal. Over the previous 2 weeks, however, the girl was breathless and tired even after walking a short distance (approx. 200 m). Apart from tachycardia (108 bpm), her lungs and heart were normal on auscultation; the patient’s blood pressure was also normal.

Laboratory test results at admission are shown in Table 1. In addition to high urea and creatinine levels, metabolic acidosis was present. Serum ion and mineral concentrations were
Table 1. Laboratory findings in the patient

|                          | Reference value | At admission May 2019 | At discharge June 2019 | Last follow up May 2021 |
|--------------------------|-----------------|-----------------------|------------------------|-------------------------|
| Blood count              |                 |                       |                        |                         |
| Hemoglobin, g/L          | 120–160         | 70                    | 114                    | 127                     |
| Erythrocytes, 10¹²/L     | 3.8–5.2         | 2.5                   | 3.89                   | 4.07                    |
| Hematocrit               | 0.35–0.47       | 0.20                  | 0.32                   | 0.36                    |
| MCV, fl                  | 82.0–98.0       | **81.6**              | 83                     | 88.2                    |
| White cells, 10⁹/L       | 4–10            | 5.57                  | 6.54                   | **3.73**                |
| Thrombocytes, 10⁹/L      | 150–400         | 169                   | 147                    | **127**                 |
| ESR                      |                 | **60/110**            | 15/24                  | 3/12                    |
| Blood chemistry          |                 |                       |                        |                         |
| CRP, g/L                 | 0–10            | 38                    | 1.1                    | <4                      |
| Procalcitonin, µg/L      | 0.0–0.5         | 0.7                   | –                      | –                       |
| Urea, mmol/L             | 3.2–8.2         | 20                    | 9.2                    | 7                       |
| Creatinine, µmol/L       | 49–90           | 97                    | 49                     | 33                      |
| Total protein, g/L       | 65–85           | 72                    | 61                     | 66                      |
| Albumin, g/L             | 32–45           | 31                    | 35                     | 41                      |
| ALT, µkat/L              | 0.10–0.78       | **1.15**              | 0.24                   | **2.49**                |
| AST, µkat/L              | 0.22–0.67       | 0.7                   | 0.27                   | **1.61**                |
| Total bilirubin, µmol/L  | 5–21            | 7                     | 5                      | 13                      |
| LDH, µkat/L              | 2–4.1           | 2.13                  | –                      | **2.72**                |
| Fe, µmol/L               | 5–33            | 6.1                   | –                      | 11.8                    |
| TIBC, µmol/L             | 24–70           | 44                    | –                      | 47.5                    |
| sTfR, mg/L               | 0.65–1.88       | **3.4**               | –                      | 0.90                    |
| Ferritin, µg/L           | 10–291          | **555**               | –                      | 158                     |
| Folate, µg/L             | 5.38–24         | 19.2                  | –                      | >24                     |
| Vitamin B12, pmol/L      | 27–170          | 112                   | –                      | 98                      |
| Quick test INR           | 0.8–1.22        | **1.26**              | **1.28**               | –                       |
| aPTT (s)                 | 22–36           | **50**                | 29                     | –                       |
| Immunology               |                 |                       |                        |                         |
| AGT (Coombs)             | Positive        | Positive              | Negative               |                         |
| Lupus anticoagulant      | Positive        | –                     | –                      |                         |
| IgM, g/L                 | 0.4–2.3         | 1.3                   | 0.94                   | 0.53                    |
| IgA, g/L                 | 0.7–4           | 3.16                  | 2.43                   | 2.67                    |
| IgG, g/L                 | 7–16            | **18.3**              | 9.7                    | 10.8                    |
| CIC, unit                | 0–50            | 112                   | –                      | 22                      |
| C3, g/L                  | 0.98–1.97       | **0.79**              | **0.89**               | **0.86**                |
| C4, g/L                  | 0.12–0.40       | 0.27                  | 0.17                   | 0.19                    |
| ANA                       | Border          | Negative              | Negative               |                         |
| Anti-dsDNA, IU/mL        | 0–20            | <12.5                 | –                      | <12.5                   |
| ANCA, U/mL               | Positive        | Negative              | Negative               |                         |
in the normal range. The erythrocyte sedimentation rate was high; C-reactive protein and procalcitonin concentrations were slightly increased. Her albumin concentration was decreased; transaminases were slightly elevated and the lactate dehydrogenase level was normal. In addition to the low hemoglobin level, she had a low red blood cell count, hematocrit, and mean corpuscular volume. The white blood cell and platelet counts were normal. While the serum iron level was just above the lower end of the normal range and the total iron-binding capacity was normal, the soluble transferrin receptor level was slightly increased. The patient had a positive direct Coombs (antiglobulin) test and tested positive for lupus anticoagulant.

Urinalysis revealed severe proteinuria and marked albuminuria. Initially, microscopic hematuria progressed to macroscopic 2 days later. The level of pyuria was slightly increased (81 WBCs/µL).

A kidney ultrasound scan showed that both kidneys were slightly enlarged, with increased parenchymal echogenicity. An X-ray of the heart and lungs revealed no pathology. Although echocardiographic findings suggested vegetations on the tip of the CVC, they were not confirmed later. The heart valves were intact. Immunological tests showed decreased complement component 3 (C3), increased circulating complexes, increased IgG and borderline antinuclear antibodies. The working diagnosis was glomerulonephritis with CVC-related bacteremia. Repeated blood cultures were performed to confirm an infectious etiology and other samples (urine, nasopharyngeal and rectal swabs) were collected for culture. The CVC was removed and empirical antibiotic therapy was initiated. Positive cultures collected from the CVC showed *Enterococcus faecalis* (also present in urine in significant amounts) and *Staphylococcus hominis*.

The initial antibiotic therapy was adjusted based on susceptibility to vancomycin combined with ceftazidime.

After 4 days of antibiotic therapy, the GFR only slightly improved but proteinuria progressed to as much as 6 g/day and microscopic hematuria became macroscopic. At that time, the patient tested positive for proteinase 3 (PR3)-ANCA. A kidney biopsy was performed (Fig. 1, 2), revealing focal necrotizing glomerulonephritis with crescent formation in up to 50% of glomeruli and mild tubulointerstitial nephritis. Immunofluorescence yielded negative results. The definite diagnosis was pauci-immune AAG. The antibiotic therapy was continued and induction immunosuppressive therapy was started according to 2019 SHARE recommendations with pulse corticosteroids and rituximab [1]. The patient also received antithrombotic and anticoagulation therapy. Due to her high risk for infectious complications, rituximab was administered instead of cyclophosphamide. Prior to methylprednisolone therapy, a bone marrow biopsy performed to assess anemia showed active granulopoiesis and slightly reduced erythropoiesis. Thus, the anemia of infection was identified. After three pulses of methylprednisolone, the girl received corticosteroids equivalent to 1 mg/kg/day prednisone, with doses being gradually decreased in accordance with the PEXIVAS protocol [2]. Her GFR gradually improved.

### Table 1 (continued)

|                      | Reference value | At admission May 2019 | At discharge June 2019 | Last follow up May 2021 |
|----------------------|----------------|-----------------------|-----------------------|-------------------------|
| **Anti-PR3, U/mL**   | 0–5            | 17.3                  | –                     | –                       |
| **Anti-MPO, U/mL**   | 0–5            | 0.7                   | –                     | –                       |
| **Urine**            |                |                       |                       |                         |
| Proteinuria, g/day   | 0–0.15         | 3.7                   | 2                     | 0.07                    |
| Albuminuria, mg/day  | 0–29           | 2440                  | 1870                  | 16                      |
| Hematuria, ery/µL    | 0–10           | 373                   | 19                    | 0                       |

Pathological values are printed in bold.
and was in the normal range after 2 weeks. Over a few days, macroscopic hematuria resolved but microscopic hematuria persisted for several weeks. Proteinuria slowly improved. Nephrotic-range proteinuria lasted for 3 months; then proteinuria decreased to 0.5 g/day. Over the next 12 months, normal amount of protein in the urine was reached and albumin concentration was just above the normal range. Rituximab and prednisone were administered as maintenance therapy. The patient was given rituximab at 6-month intervals over a period of 18 months. Rituximab therapy possibly caused herpes simplex keratitis that developed in the patient’s left eye 5 days after the first rituximab maintenance dose. Throughout the rituximab therapy, the patient only once developed catheter-related sepsis. Prednisone was tapered and stopped after 16 months of therapy. At present, 2 years from the development of AAG, the patient takes no immunosuppressants and continues her antiproteinuric therapy with an ACE inhibitor and angiotensin-1 receptor blocker. She also continues receiving long-term HPN. The long-term HPN has led to mild hepatopathy – parenteral nutrition liver disease (Table 1).

As for kidney function, the patient’s AAG has so far been successfully treated and her GFR and protein excretion are normal. However, future AAG recurrence cannot be excluded. Her overall prognosis is uncertain. There is limited experience with intestinal transplantation in the Czech Republic. Long-term HPN may be associated with a risk for further complications with sepsis and thrombosis, potentially leading to loss of vascular access for HPN.

**Discussion/Conclusion**

In the literature, only a few cases of glomerulonephritis associated with CVC-related infection in individuals on HPN have been published – see Table 2 [3–8]. Those were patients with MPGN and bacteremia (immune complex glomerulonephritis). The only exception was a female with crescentic glomerulonephritis, but immunofluorescence findings positive for IgG, IgM, and C3, and a negative serum ANCA test; therefore, she was not diagnosed with AAG.
Table 2. Glomerulonephritis associated with chronic infection in patients on long-term parenteral nutrition

| References       | Patient (sex, age) | Underlying disease | TPN, years | Blood culture                  | PR3-ANCA, U/mL | Renal biopsy                          | Treatment                              | Outcome recovery |
|------------------|--------------------|--------------------|------------|--------------------------------|----------------|---------------------------------------|----------------------------------------|------------------|
| Yared et al. [3] | M, 66              | SBS                | 5          | S. epidermidis, C. jeikeium    | N/D            | MPGN                                  | ATB, CVC removal                       | Full             |
|                  | F, 45              | SBS                | 3          | Unknown                        | N/D            | MPGN                                  | ATB, CVC removal                       | Full             |
| Ohara et al. [4] | M, 13              | SBS                | 13         | S. epidermidis                 | N/D            | MPGN                                  | ATB, CVC removal                       | Full             |
| Kusaba et al. [5]| F, 59              | Post-radiation enteritis | 2        | S. epidermidis                 | Negative       | Crescentic GN*                         | ATB, CVC removal, ESRD                 |                  |
| Sy et al. [6]    | M, 23              | SBS                | 6          | S. epidermidis                 | N/D            | MPGN IF: IgM, C3                       | ATB, CVC removal                       | Full             |
|                  |                    |                    | 2nd episode| 19.5                           | Negative       | MPGN                                  | ATB, CVC removal                       | Partial          |
|                  |                    |                    | 3rd episode| 20                             | N/D            | No                                    | ATB, CVC removal                       | Partial          |
| Hayashi et al. [7]| F, 45              | Eating disorder    | 1.5        | MRCNS                          | Negative       | MPGN                                  | ATB, CVC removal                       | Partial          |
| Okada et al. [8] | M, 12              | MMIHS              | 8          | S. epidermidis                 | Positive 33    | MPGN IF: C3, IgM, C1q                  | Pred, mizoribine, ATB, CVC removal     | Full             |
|                  | F, 24              | MMIHS              | 18         | MRSE                           | Positive 19    | MPGN IF: C3, IgM                       | ATB, CVC removal, MP, pred             | Full             |
| Current case report | F, 16              | SBS                | 11         | E. faecalis, S. hominis        | Positive 17    | Crescentic GN**                        | ATB, CVC removal, MP, pred, RTX       | Full             |

TPN, total parenteral nutrition; M, male; F, female; SBS, short bowel syndrome; S. epidermidis, Staphylococcus epidermidis; C. jeikeium, Corynebacterium jeikeium; MRCNS, methicillin-resistant coagulase-negative staphylococci; MRSE, methicillin-resistant Staphylococcus epidermidis; E. faecalis, Enterococcus faecalis; S. hominis, Staphylococcus hominis; N/D, no data; MPGN, membranoproliferative glomerulonephritis; ATB, antibiotics; CVC, central venous catheter; IF, immunofluorescence; ESRD, end stage renal disease; MMIHS, megacystis microcolon intestinal hypoperistalsis syndrome; pred, prednisolone; MP, methylprednisolone; RTX, rituximab.

*Crescentic glomerulonephritis induced by catheter-related bloodstream infection (ANCA-negative).

**Crescentic ANCA-associated glomerulonephritis.
Some of the patients were successfully treated with antibiotics and CVC replacement without requiring immunosuppressive therapy. Two patients with biopsy-confirmed MPGN and positive serum PR3-ANCA tests were treated with immunosuppressants in addition to antibiotics and CVC replacement.

To the best of our knowledge, the present case is the first reported patient on HPN with clearly confirmed PR3-AAG. The girl had crescentic glomerulonephritis with negative immunofluorescence and positive PR3-ANCA findings.

The production of PR3-ANCA may result in not only AAG but also ANCA-associated vasculitis (AAV) with possible involvement of various organs. Most commonly, AAV is characterized by pulmonary, renal, and ear, nose, and throat manifestations. Our patient only had AAG.

Even though AAG mostly develops as a primary condition, its etiology is not fully elucidated. A genetic predisposition or acquired protease/antiprotease imbalance is assumed, that is, production of PR3 antineutrophil cytoplasmic autoantibodies. The external factors may be bacterial or viral infections, as well as hypersensitivity to an unspecified antigen. In this particular patient, the only external cause to be considered as a trigger to her AAG is chronic bacteremia and a long-term contact of the vessel wall and bloodstream with the artificial material of the CVC. The girl had a history of multiple sepsis episodes and was diagnosed with microscopic hematuria and mild proteinuria 2 months before acute kidney injury. Her kidney injury and severe anemia were diagnosed during a routine gastroenterology exam without the patient suffering from acute clinical problems. Only after abnormal laboratory test results were obtained, further diagnosis and treatment followed. Her CVC-related infection was confirmed by positive blood culture results. The ESR was high at 60/110, with a not very high C-reactive protein level at 38 g/L and a procalcitonin level just above the limit. The patient was afebrile. All these findings are suggestive of chronic infection. Even though an accidental coincidence of AAG and chronic bacteremia cannot be ruled out, it is far less likely.

Glomerulonephritis concomitant with CVC-related infection is considered analogous to shunt nephritis, previously seen in patients with a ventriculointestinal shunt (As ventriculoperitoneal shunts are currently preferred, the incidence of this type of glomerulonephritis has dropped). The initial moment was catheter colonization with minimally invasive bacteria, usually coagulase-negative staphylococci. Bacterial growth led to production of immunoglobulins and immune complexes deposited in the kidneys. With kidney biopsies, the prevailing finding was MPGN with IgM, C1q, and C3 deposits on the basal membrane [9].

The mechanism of glomerulonephritis in chronic bacteremia, however, is not only that of immune complexes. In patients with abnormal reactivity to neutrophilic granulocytes (with ANCA), immunopathological processes may occur in the bloodstream, secondarily leading to endothelial damage. After ANCA bind to neutrophil structures, such as myeloperoxidase (MPO), PR3, or lysosome-associated membrane protein 2, the entire spectrum of inflammatory mediators is released which disrupt the vascular endothelium. The immunopathogenesis involves autoreactive Th1 cells (PR3-induced) and the damaging autoimmune process is maintained [10, 11]. The production of ANCA results from stimulation of B cells in chronic bacteremia, but it may also be associated with formation of originally protective neutrophil extracellular traps. As part of the defense against infectious agents, cell nucleus components and granule contents are released into the extracellular space during netosis (neutrophil death). These may be proteins (e.g., MPO or PR3) exposed to the immune system, potentially inducing autoantibody production. Subsequently, ANCA lead to neutrophil activation and disintegration, maintaining the autoimmune process.

ANCA may be targeted against PR3 or MPO. While a direct causal relationship has not been established between infection-induced production of anti-PR3 and AAG or AAV, the relationship has been confirmed for antibodies against MPO. However, those patients were not on HPN. In 2020, a systematic review was published that included 23 patients with
infection-induced AAV or AAG. Unlike the present case, those patients tested positive for antibodies against MPO. The infectious pathogens were both bacteria and viruses. While in some patients, vasculitis/glomerulonephritis resolved after they recovered from infection, others required immunosuppressants in addition to anti-infective therapy. Authors warn of a potentially catastrophic impact of immunosuppressant administration without treating infection [12].

Additionally, the presence of ANCAs was reported in a group of patients having glomerulonephritis associated with infective endocarditis. In 28% of them, serum ANCA tests were positive; their kidney biopsy yielded varied findings. Apart from antibiotics, some of the patients also received immunosuppressants and some required valvular surgery [10]. In the present case, infective endocarditis was ruled out. For patients diagnosed with crescentic glomerulonephritis due to CVC-related infection requiring immunosuppressive therapy, rituximab (along with corticosteroids) is the first choice as the risk of infectious complications is lower compared to cyclophosphamide [13].

The number of individuals on long-term HPN is currently rising. It must be borne in mind that they are at risk for not only sepsis, thrombosis, and infective endocarditis but also glomerulonephritis. Since chronic bacteremia may lead to various types of glomerulonephritis, early kidney biopsy is warranted. Even though MPGN is most commonly reported, the present patient had AAG which, to be successfully treated, requires immunosuppressive therapy in addition to antibiotic administration and CVC replacement.

**Conclusion**

This is the first report of ANCA-associated crescentic glomerulonephritis in a patient on HPN assumed to have been triggered by chronic bacteremia. We underline the importance of a timely kidney biopsy and potentially successful immunosuppressive therapy with corticosteroids and rituximab.

**Statement of Ethics**

The Ethics Committee of the University Hospital Olomouc and Faculty of Medicine and Dentistry, Palacky University Olomouc does not require ethical approval for reporting individual cases based on information obtained during patient care. This study protocol was reviewed, and the need for approval was waived by Ethics Committee of the University Hospital Olomouc and Faculty of Medicine and Dentistry, reference number 12/22. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. At the time of consenting, the patient was 18 years old, that is, of legal age as set by Czech law.

**Conflict of Interest Statement**

The authors have no conflicts of interest to declare.

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Author Contributions

Vladimir Tesar: literature search and supervision. Hana Flogelova: clinical management, literature search, and preparation of the manuscript draft. Eva Karaskova, Marie Rohanova: clinical assessment and management of the patient. Katerina Bouchalova, Vendula Latalova: literature search, contribution to the Discussion/Conclusion section. Tomas Tichy: histopathological examination. All authors read the final manuscript and approved its content with regard to their specialties.

Data Availability Statement

The patient data used in the case report are deposited in the University Hospital Olomouc, Czech Republic information system. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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