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Unraveling the association between chronic inflammatory demyelinating polyradiculoneuropathy and peritoneal Dialysis

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Abstract

Background Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a rare disease seen in the general population and has been reported as showing an increased incidence in the peritoneal dialysis (PD) population, as documented in case reports.

Methods We conducted a case-control study using data from the Taichung Veterans General Hospital electric medical record database from the years 2010 to 2023. We defined cases as CIDP with End-stage kidney disease (ESKD) and controls as without CIDP. A logistic regression analysis was used to investigate the association between CIDP and dialysis modality, age, gender, dialysis duration, plasma potassium > 5.5 mEq/L and < 2.5 mEq/L, and intact parathyroid hormone (i-PTH) > 613 pg/mL.

Results Our findings suggest that PD may be a risk factor in the ESKD population (Odds ratio: 5.125, C.I.: 1.078 ~ 24.372, $p = 0.040$) according to logistic regression analysis. Dialysis duration, gender, diabetes mellitus, HbA1c > 7%, hypokalemia, hyperkalemia, and hyperparathyroidism did not show an association with CIDP.

Conclusion There seems to be an association between PD and CIDP in this case-control study. Possible mechanisms may involve systemic inflammation induced by peritoneal dialysate exchange or the content of the dialysate. Further studies are still needed.

Introduction

Peripheral neuropathy, including uremic neuropathy and carpal tunnel syndrome, has been well-described in end-stage kidney disease (ESKD) patients [1]. On the contrary, demyelinating neuropathy specifically related to peritoneal dialysis (PD) has been seldom reported [2, 3]. In 1993, the first case reports described 3 cases of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) in PD patients [4]. In the most recent case series taken from 2022 [5], Bhuta K et al. reported three cases while also summarizing 6 cases from previous case reports [6–8]. According to these case reports,

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this population seemed to possess certain characteristics. These included significantly more male than female patients, a young age at disease onset, a duration from starting dialysis to CIDP of less than or around 1 year, and the switching of PD to hemodialysis (HD) not halting the clinical progression of CIDP. In these case reports, the effective treatment when involving immunosuppression included steroids, intravenous immunoglobulin (IVIG) and plasmapheresis. Kidney transplants involving immunosuppression medication showed an excellent response. Though the mechanism of CIDP remains unknown, the inflammation caused by PD is one of the hypotheses. We aim to characterize the association of CIDP in PD patients through this case-control study.

Materials and methods

Patients and data collection

We retrospectively identified cases of CIDP in an ESKD population. To avoid coding error, we included CIDP, acute inflammatory demyelinating polyradiculoneuropathies (AIDP) and polyneuropathy in ESKD patients from Taichung Veterans General Hospital Electric Medical Records system from the years 2010 to 2023. The diagnoses of the above conditions were based upon ICD-9/ ICD-10 codes (CIDP 357.8/ G61.81, AIDP 357.0/ G61.0, Polyneuropathy 356.9/ G62.9). We scrutinized the clinical history and laboratory data of each patient, including electrophysiology and cerebrospinal fluid samples. The confirmation of CIDP diagnosis was determined by a neurologist in our hospital (TYC) based upon the 2010 EFNS/PNS CIDP criteria [9]. We identified 42 patients with polyneuropathy, with none of them having a clinical diagnosis of CIDP upon review of their medical records. Among the 22 patients initially coded as having CIDP, only 10 who were ultimately diagnosed with definite CIDP went on to receive immunosuppressive medication.

The control group was defined as ESRD patients without CIDP, and retrospectively selected from the Taichung Veterans General Hospital Electric Medical Records system from the years 2010 to 2023. We initially identified 1,498 PD and 4,771 HD patients, finally selecting patients from those who were still under follow-up in our dialysis unit on October 31, 2023, in order to confirm that CIDP had not occurred. The control group ultimately included 291 HD patients and 228 PD patients. This study was approved by the Institutional Review Board (IRB) of Taichung Veterans General Hospital (IRB No. CE22229B). According to Taiwan's Human Subjects Research Act, a waiver for informed consent was granted by the Institutional Review Board of Taichung Veterans General Hospital because this retrospective study involved the use of pre-existing data that were analyzed anonymously. The information obtained from the database was fully anonymized and de-identified to ensure the privacy and confidentiality of the participants. In order to provide clinical characteristics of PD patients with CIDP, we obtained informed consent for participation and publication from patients listed in Tables 1 and 2.

We selected diabetes mellitus (DM), hypertension (HTN), peripheral arterial occlusive disease (PAOD), anti-neutrophil cytoplasmic antibodies (ANCA) vasculitis, systemic lupus erythematosus (SLE) and human immunodeficiency virus (HIV) infection as comorbidities, due to the above conditions being associated with peripheral neuropathy. The ICD coding is listed in Table S1.

We also selected age, gender, dialysis duration, plasma potassium > 5.5 mEq/L and < 2.5 mEq/L, and intact parathyroid hormone (i-PTH) > 613 pg/mL as possible risk factors of polyneuropathy in ESKD patients according to previous case reports and literature review [10–13].

Statistical analysis

Descriptive data analysis was conducted using IBM SPSS Statistics (v26) to calculate the median for continuous variables. Results obtained from the analysis of categorical variables were summarized as proportions/ percentages. Inferential statistical analyses, including the Mann-Whitney U test, chi-square test, and univariate logistic regression were performed using IBM SPSS Statistics (v26). P-values less than 0.05 were considered as the cut-off for statistical significance.

Results

The flow chart for case and control selection is illustrated in Fig. 1, while demographic data is shown in Table 3. Two of the risk factors, dialysis modality, and PAOD, showed statistically significant differences between the two groups at $p=0.027$ and 0.048 respectively. No statistically significant difference was detected when considering gender, age, dialysis duration, DM, HTN, ANCA, SLE, HTN, HIV, PTH > 613 pg/ml, K < 2.5 mEq/L, K > 5.5 mEq/L and HbA1c > 7%. After univariate logistic regression, peritoneal dialysis showed statistical significance with an increased odds ratio (Odds ratio: 5.125, C.I.: 1.078 ~ 24.372, $p=0.040$) (Table 4). The clinical characteristics of the 8 CIDP cases with PD are listed in Table 1. Each of the cases wad diagnosed by the neurologist according to electrophysiology data and clinical presentation. The dose and type of PD fluids are listed in Table 2.

Discussion

Our data shows that peritoneal dialysis may be a risk factor for CIDP in the ESKD population. To compare with previous case reports, the summary of previous literature is listed in Table S2. Although previous case reports reported a total of 15 male patients and 1 female PD patient associated with CIDP, our data did not find

Table 1 Clinical characteristics of the 8 confirmed CIDP cases with peritoneal dialysis

Case	Disease progression speed	Age	Sex	Cause of ESKD	Medical disease	PD duration	Motor	Sensory	Immunosuppression	Response	Shift to HD
1	3 months	18	Male	IgA nephropathy	HTN	3 months	LEs : weakness in 3 months	LEs : numbness, soreness for 3 months	TPE Steroid Imuran	Improved	Yes
2	5 months	62	Male	DKD	SCC HTN DM	4 months	LEs : weakness in 5 months	LEs : numbness and soreness for 9 months	IVIG Steroid	Partial	Yes
3	6 months	65	Male	FSGS	HTN	6 years	No weakness	LEs : numbness for 1 year, left calf pain for 6 months	Steroid	Partial	No
4	3 months	52	Female	ADPKD	HTN	4 years	LEs : weakness in 3 months	LEs : numbness for 1 year, left calf pain for 5 months	IVIG Steroid	Improved	No
5	1 month	22	Male	IgA nephropathy	HTN	1 year	LEs : weakness in 1 month	LEs : numbness for 1 month	TPE	No	Yes
6	1 month	46	Female	Lupus nephritis	DM, HTN	6 years	LEs : weakness in 5 months	LEs : numbness for 1 year	IVIG	Improved	Yes (nephrectomy)
7	1 month	63	Female	Lupus nephritis	TCC	22 months	LEs : weakness for 4 months	LEs : numbness in 4 months	IVIG Steroid	Improved	No
8	3 months	48	Male	DKD	HTN, DM, Cirrhosis	2 years	LEs : weakness in 3 months	LEs : numbness in 3 months	IVIG Steroid	Resolved	No

Note: LE=Lower extremity; PD=Peritoneal dialysis; HD=Hemodialysis; CCR=Creatinine Clearance; hs-CRP=High sensitivity C-reactive protein; PET=Peritoneal Equilibrium Test;

DM=Diabetes Mellitus; HTN=Hypertension; ADPKD=Autosomal Dominant Polycystic Kidney Disease; TCC=Transitional Cell Carcinoma; SCC=Squamous Cell Carcinoma; DKD=Diabetic Kidney Disease; FSGS=Focal Segmental Glomerulosclerosis; TPE=Therapeutic Plasma Exchange; IVIG: Intravenous Immunoglobulin

Table 2 PD fluid type and dose in confirmed CIDP cases

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
PD Duration	3 months	4 months	6 years	4 years	1 year	6 years	2 years
PD fluid exposure (L/day)	9	no data	10	8.9	13.6	12.4	7
1.5% (L/day)	9		4	5	5		7.5
2.5% (L/day)			4	2.5	7	7	
4.25% (L/day)			As needed			5	
Icodextrin (L/day)			2	1.5	2	2	
Nutrineal (L/day)							
Biocompatible fluid (L/day)							
Residual Kidney Function (L/day)	1.77		0	0	0	0	1.7 L
Weekly Peritoneal CrCl (L/week/1.73m2)	35.44		52.76	36.34	56.43	47.9	15.16
Weekly Kidney CrCl ((L/week/1.73m2))	42.8		0	0	0	0	27.46
KT/V	1.73		1.82	2.65	1.95	1.81	2.14
PET function	Low Average		Low Average	Low Average	Low Average	Low Average	High Average
Serum hs-CRP(mg/dl)	0.141		0.08	0.023	0.131	1.44	1.7

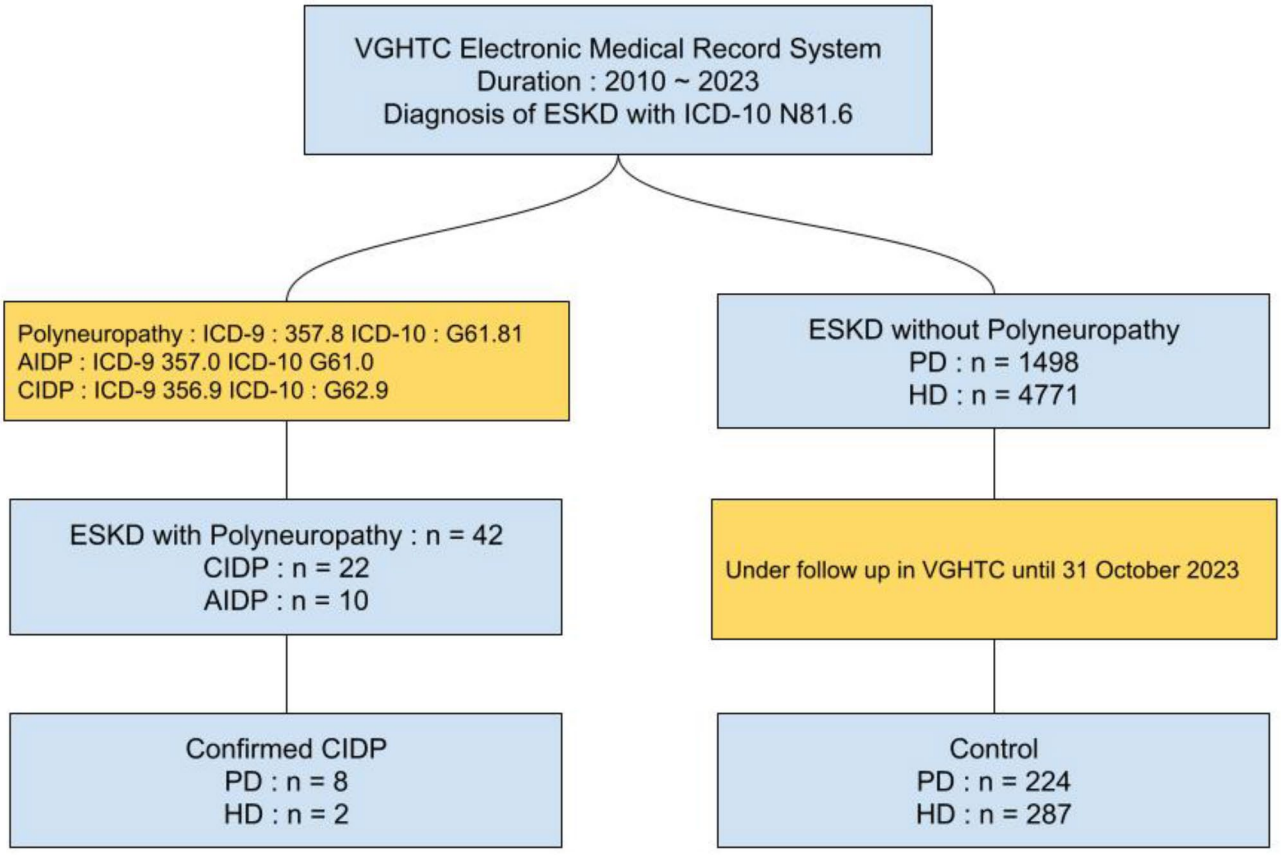

Fig. 1 The flow chart for the case and control selection

a significant association. The prevalence of males was higher than that of females in ESKD patients both in Taiwan as well as other regions of the world over the past decade [14, 15], which may explain the higher reported rate of CIDP in male ESKD cases.

Peripheral neuropathy is a common condition in ESKD patients, particularly among those who are on long-term dialysis. The most prevalent form of peripheral polyneuropathy is uremic neuropathy (UN) [1, 16]. Other systemic diseases causing polyneuropathy include diabetes, amyloidosis, multiple myeloma, SLE, vasculitis, vitamin B12 deficiency and pyridoxine deficiency. UN has been reported to have a prevalence of 60–90% [1, 10], and typically presents itself with symmetric, length-dependent and sensory-predominant dysfunction in the lower limbs more than that seen in the upper limbs.

Table 3 Demographics of the case and control groups

	Case (*n* = 10)		Control (*n* = 511)		*p* value
Gender					0.526
Male	7	(70.0%)	289	(56.6%)	
Female	3	(30.0%)	222	(43.4%)	
Age	50.0	(36.3–63.5)	60.0	(48.0–70.0)	0.145
Dialysis modality					0.027*
PD	8.0	(80.0%)	224.0	(43.8%)	
HD	2.0	(20.0%)	287.0	(56.2%)	
Dialysis duration (months)	36.0 (10 ~ 72)		47.0 (18 ~ 88)		0.308
Comorbidity					
ANCA	0	(0.0%)	4	(0.8%)	1.000
SLE	2	(20.0%)	25	(4.9%)	0.090
HTN	3	(30.0%)	233	(43.6%)	0.526
DM	5	(50.0%)	182	(35.6%)	0.342
PAOD	1	(10.0%)	226	(44.2%)	0.048*
HIV	0	(0.0%)	2	(0.4%)	1.000
Laboratory data					
PTH > 613 pg/ml	2	(20.0%)	438	(86.0%)	0.731
K < 2.5 mEq/L	0	(0.0%)	1	(0.2%)	1.000
K > 5.5 mEq/L	0	(0.0%)	55	(10.7%)	0.610
HbA1c > 7%	4	(40.0%)	57	(11.1%)	0.751

Chi-square test or Mann-Whitney U test, Median (IQR). *$p < 0.05$

Table 4 Univariate logistic regression

	p value	Odds ratio	95% C.I.	
			Lower	Upper
Peritoneal dialysis	**0.040***	5.125	1.078	24.372
PAOD	0.063	0.140	0.018	1.114

*$p < 0.05$

The disease often progresses over the course of weeks to months. Nerve conduction studies (NCS) have revealed axonal injury with decreased sensory amplitude and, to a lesser extent, motor amplitude, while preserving conduction velocity [13, 17]. If the disease pattern presents itself with prominent motor involvement or rapid clinical progression over the course of days to weeks, CIDP should be listed in the differential diagnosis. CIDP is a rare disease within the general population, with a prevalence ranging from 0.7 to 10.3 cases per 100,000 people [18]. According to the 2021 EFNS/PNS guidelines, the diagnosis of CIDP relies on a combination of clinical, electrodiagnostic and CSF laboratory results with the elimination of CIDP mimics [19]. Electrodiagnostic criteria for peripheral nerve demyelination are paramount. The typical CIDP diagnosis is defined as progressive or relapsing, symmetric, proximal, and distal muscle weakness in both the upper and lower limbs, with sensory involvement in at least two limbs. The disease course usually develops in a period of 8 weeks. Tendon reflex is usually reduced or absent in all limbs. In our medical center, we diagnosed eight cases of CIDP with progressive weakness and paresthesia, with nerve conduction studies (NCS) confirming a diagnosis of demyelination. We thought it to be unusual to detect predominantly PD-related cases in our center over the past 10 years, which had previously handled approximately 240–260 PD cases annually.

In previous case reports, CIDP mostly occurred within one year after starting dialysis. In our study, the diagnosis of CIDP occurred as late as 5 years. This may be due to a relatively low awareness of alternative diagnoses aside from uremic neuropathy amongst nephrologists. The weekly KT/V values of our cases during the three months prior to CIDP diagnosis exceeded 1.7, the recommended target listed in the International Society of Peritoneal Dialysis (ISPD) guidelines [20], thus making UN less likely. Though diabetes mellitus, HIV, ANCA vasculitis and SLE could each cause polyneuropathy, these afflictions did not show a significant difference between the two groups in our results.

There have been several studies which found that hyperkalemia in ESKD could influence the excitability of peripheral nerves via NCS examination [11–13, 17]. Hypokalemia could cause reversible neuropathy [21–23]. In a meta-analysis study, duration of diabetes, age, and HbA1c were all found to be associated with significantly increased risks of diabetic polyneuropathy [24]. The middle molecule parathyroid hormone (PTH) was also suspected to be causing UN as well [25, 26]. We further examined the effects of HbA1c > 7, chronic hypokalemia (K < 2.5mEq/L) or hyperkalemia (K > 5.5mEq/L), and hyperparathyroidism > 613 pg/ml (plasma intact-PTH more than 9 times the upper limit), and discovered that there was no difference detected between the two groups.

The possible mechanisms surrounding CIDP remain unclear in the general population [27]. Koike et al. showed that a sural nerve biopsy in CIDP revealed the destruction of myelin by macrophages [28]. Autoantibodies and complement systems may help target specific structures on the myelin [29]. Although humoral and cellular immunity have been postulated, the pathophysiology of CIDP is still unknown.

It has been recognized that PD could cause local peritoneal inflammation [30] as well as systemic inflammation [31]. The causes of systemic inflammation have been postulated to be due to the decreased clearance of pro-inflammatory cytokines resulting from worsening residual kidney function, repeated endotoxin exposure, non-physiological dialysis solution exposure, repeated peritonitis and fluid overload [31, 32]. Interleukin (IL)-6 and C-reactive protein (CRP) are potential biomarkers for systemic inflammation. IL-6 was reported to be elevated in peritoneal fluid and associated with increased peritoneal solute transport rate [33, 34]. Elevated plasma

IL-6 and CRP were also reported to be elevated in PD patients, but could not demonstrate having association with peritoneal inflammation [35]. The prognostic value of elevated CRP in PD is still under investigation, however, studies have found a positive association with PD drop-out rate and mortality [36].

Due to the sural nerve biopsy in CIDP showing infiltration demonstrated by Koike [28, 29] et al., monocytes/macrophages appeared to be the important link between CIDP and PD. In 1984, Goldstein CS et al. analyzed macrophages from plasma and peritoneal fluid and found that the chemotaxis score of plasma monocytes was higher than that seen in healthy subjects and hemodialysis patients [37]. They proposed that peritoneal fluid exchange would cause the removal of 30 ~ 40 million macrophages daily and stimulate the bone marrow to replace resident macrophages with immature monocytes. Jacobson SH et al. found that the peripheral blood monocytes of PD patients displayed a higher expression of CD 62 L and lower CD11b/CD18 than healthy and kidney function insufficiency patients [38]. These monocytes had a higher potential to express CD11b upon facing chemotactic factors. Sutherland TE et al. found that repeated peritoneal dialysate replacement in a mouse model would change the composition of macrophages in peritoneal fluid from resident macrophages to a pro-inflammatory monocyte-derived phenotype [39]. Interestingly, they also found that adding glucose degradation products (GDPs) would further cause more severe inflammation. Louwe PA et al. demonstrated that monocytes instilled in the peritoneum after mild sterile inflammation would compete with resident macrophages for the biological niche and thus remain in a long-lasting pro-inflammatory status [40].

We hypothesize that PD may place patients in a pro-inflammatory status due to activated macrophages and monocytes. We were unable to analyze the serum C-reactive protein levels due to the retrospective study design along with there being missing data in the control group. The highest level seen within several months prior to CIDP diagnosis was lower than 2 mg/dL in our study(Table 2). We postulate that the systemic inflammatory reaction may stem from the content of the dialysate fluid, such as glucose degradation products (GDPs), or the macrophage dynamics caused by the repeated exchange of dialysate effluent fluid. Hereto, more research is still required.

Limitations

The retrospective case-control nature of this study inherently unmeasured confounding factors. Due to the low prevalence of CIDP and insufficient awareness by nephrologists, we could only detect 10 cases over a period of 14 years. Therefore, there may be an underdiagnosis of CIDP in HD patients. The result of a single-center study may not be able to be generalized to all the ESKD population. Additionally, the ICD coding error may have caused an incorrect prevalence of comorbidities in the control group.

Conclusion

Peritoneal dialysis may be an important risk factor for CIDP in the ESKD population. The diagnosis of CIDP is paramount as timely treatment may improve a patient's quality of life. Additional studies are still necessary in order to confirm the association between PD and CIDP, while monocytes may also play an important role in immunopathogenesis.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-024-03830-5.

Supplementary Material 1

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Author contributions

CHC provided clinical insights and expertise throughout the development of the study. YJC took the lead in drafting the study, formulating research questions, and designing the methodology. TYC contributed specialized knowledge in neurology, confirming diagnoses and providing invaluable suggestions that enhanced the neurological aspects of the study. All authors commented on the draft manuscript and approved the final manuscript for submission.

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Data availability

The data that support the findings of this study are available from Taichung Veterans General Hospital(TVGH) but restrictions apply to the availability of these data, which were used under license for the current study and are not publicly available. However, data may be made available upon reasonable request and with permission from TVGH. Researchers interested in accessing the data should contact Cheng-Hsu Chen to request access.Please note that the data availability is subject to the policies and procedures of Taichung Veterans General Hospital.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board (IRB) of Taichung Veterans General Hospital (IRB No. CE22229B). According to Taiwan's Human Subjects Research Act, a waiver for informed consent was granted by the Institutional Review Board of Taichung Veterans General Hospital because this retrospective study involved the use of pre-existing data that were analyzed anonymously. The information obtained from the database was fully anonymized and de-identified to ensure the privacy and confidentiality

of the participants. Informed consent was obtained from the patients with identifying information in Table 1 and Table 2.

Consent for publication
Consent for publications was obtained from the patients with identifying information in Table 1 and Table 2.

Competing interests
The authors declare no competing interests.

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