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BRIEF COMMUNICATION

Movement Disorders Resulting From Bilateral Basal Ganglia Lesions in End-Stage Kidney Disease: A Systematic Review

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ABSTRACT

Objective The basal ganglia (BG) are susceptible to fluctuations in blood urea levels, sometimes resulting in movement disorders. We described patients with end-stage kidney disease (ESKD) presenting with movement disorders associated with bilateral BG lesions on imaging.

Methods We report four patients and systematically reviewed all published cases of ESKD presenting with movement disorders and bilateral BG lesions (EBSCOhost and Ovid).

Results Of the 72 patients identified, 55 (76.4%) were on regular dialysis. Parkinsonism was the most common movement disorder (n = 39; 54.2%), followed by chorea (n = 24; 33.3%). Diabetes mellitus (n = 51; 70.8%) and hypertension (n = 16; 22.2%) were the most common risk factors. Forty-three (59.7%) were of Asian ethnicity. Complete clinical resolution was reported in 17 (30.9%) patients, while 38 (69.1%) had incomplete clinical resolution with relapse. Complete radiological resolution occurred in 14 (34.1%) patients.

Conclusion Movement disorders associated with BG lesions should be recognized as a rare and potentially reversible metabolic movement disorder in patients with ESKD.

Keywords Basal ganglia; Chorea; End-stage kidney disease; Magnetic resonance imaging; Parkinsonism.

Movement disorders associated with bilateral basal ganglia (BG) lesions in patients with end-stage kidney disease (ESKD) were first reported in three Korean patients in 1998. Since then, similar cases presenting with either hyperkinetic movement disorder or parkinsonism have been described, although it remains relatively rare.[1] A longitudinal study over two years involving seventy Middle Eastern ESKD patients identified this syndrome in only three (4.3%) patients.[2]

Here, we describe four patients with ESKD on dialysis presenting with movement disorder and bilateral BG lesions and conducted a systematic review of this syndrome focusing on its phenomenology and prognosis.

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MATERIALS & METHODS

We described four cases and performed a literature search in EBSCOhost and Ovid using Medical Subject Headings (MeSH) terms with no language restrictions (September 9, 2021): [("renal disease" OR "uraemia") AND ("basal ganglia" OR "striatal" OR "putaminal" OR "caudate" OR "hyperintensity" OR "hypodensity") AND ("movement disorder" OR "chorea" OR "Parkinsonism")]. This study was approved by the Research Ethics Committee of UKM (JEP-2021-689).

RESULTS

Of the 1,033 articles screened, 38 articles fulfilled our search term criteria, yielding a total of 72 patients (Supplementary Figure 1 in the online-only Data Supplement), and details were outlined in Supplementary Table 1 (in the online-only Data Supplement). Approximately 43 (59.7%) patients were Asians. The most common premorbid/comorbid conditions were type 2 diabetes mellitus (T2DM) (n = 51; 70.8%), followed by hypertension (n = 16; 22.2%). Parkinsonism was the most common movement disorder (n = 39; 54.2%), followed by chorea (n = 24; 33.3%) (Table 1). Most patients were on regular dialysis before the onset of the movement disorder (n = 55; 76.4%). Therapy was mainly supportive involving more intensive dialysis, correction of hyperglycemia with insulin therapy, adequate hydration, and symptomatic therapy for the movement disorder.

Neuroimaging findings included bilateral BG hypodensities on CT (n = 22), bilateral BG hypointensities on magnetic resonance imaging (MRI) T1WI with corresponding T2WI hyperintensities, and hyperintensities in the corresponding regions on diffusion-weighted imaging (DWI) and on apparent diffusion coefficient (ADC) sequences. Seven patients had positron emission tomography (PET)/single photon emission computed tomography (SPECT) imaging, which showed hypoperfusion in the BG (n = 4). Two patients' magnetic resonance sepectroscopy (MRS) findings showed a low N-acetyl aspartate ratio (NAA) in the BG; one also showed an elevated lactate peak.

Clinical outcome data were available for 55 patients; 17 (30.9%) had complete resolution of the movement disorder (parkinsonism: n = 7; chorea: n = 10), while 38 (69.1%) had incomplete resolution (parkinsonism: n = 25; chorea: n = 13). Complete resolution was more frequently observed with chorea than parkinsonism (43.5% vs. 21.9%; p = 0.09). Radiological follow-up was available for 41 patients, with complete resolution in 14 patients (34.1%), while 27 patients showed incomplete resolution (65.9%) (Table 1).

Table 1. Summary of study characteristics of 72 patients with ESKD and acute movement disorder

Characteristic	Value
Age (yr)	57 (35–82)
Ethnicity	
Asian	43
Caucasian	6
Hispanic	3
Not specified	20
Premorbid/comorbid	
DM	30 (41.7)
DM, hypertension	10 (13.9)
DM, hypertension, coronary heart disease	3 (4.2)
Hypertension	2 (2.8)
DM, hypertension, ischemic heart disease, lacunar infarct	1 (1.4)
DM, hypertension, stroke	1 (1.4)
DM, hypertension, seizures	1 (1.4)
DM, hypertension, hypercholesterolemia	1 (1.4)
DM, hypertension, anemia	1 (1.4)
DM, hypertension, schizoaffective disorder, polysubstance abuse in remission	1 (1.4)
DM, hypertension, peripheral vascular disease, hypothyroidism, sarcoidosis	1 (1.4)
DM, hypertension, hypercholesterolemia, depression, hypercalcemia, submandibular and perihilar lymphadenopathy, sarcoidosis, hyperparathyroidism	1 (1.4)
DM, coronary disease	1 (1.4)
Hypertension, congenital renal agenesis	1 (1.4)
N/R	17 (23.6)
Duration of dialysis (yr, range)	0–20
Acute movement disorder	
Parkinsonism	29 (40.3)
Chorea	18 (25.0)
Parkinsonism, dysarthria, dysphagia	4 (5.6)
Chorea, dysarthria	4 (5.6)
Parkinsonism, dysarthria	3 (4.2)
Parkinsonian gait, dysarthria	2 (2.8)
Dystonia	2 (2.8)
Dyskinesia	1 (1.4)
Chorea, parkinsonism	1 (1.4)
Chorea, restless leg syndrome	1 (1.4)
Chorea, dyskinesia	1 (1.4)
Orolingual and facial dyskinesias, parkinsonian gaits, dysarthria, dysphagia	1 (1.4)
Parkinsonian gait	1 (1.4)
Generalized dyskinesia, ataxia, dysarthria	1 (1.4)
N/R	3 (4.2)
Radiological findings	
PET/SPECT	
Hypoperfusion in bilateral basal ganglia	1 (1.4)
Hyperperfusion in bilateral basal ganglia	1 (1.4)

Table 1. Summary of study characteristics of 72 patients with ESKD and acute movement disorder (continued)

Characteristic	Value
Hypoperfusion in bilateral striatum	1 (1.4)
Hypoperfusion in bilateral putamen, right caudate, bilateral occipital cortex, right frontal cortex	1 (1.4)
Hypoperfusion in putamen, left caudate, bilateral lateral frontal cortex	1 (1.4)
Normal	2 (2.8)
N/A	65 (90.3)
CT	
Bilateral basal ganglia hypodensity	14 (19.4)
Bilateral lenticular nuclei hypodensity	3 (4.2)
Bilateral basal ganglia hypodensity with surrounding oedema	1 (1.4)
Bilateral basal ganglia and thalami hypodensity	1 (1.4)
Bilateral lenticular nuclei hypodensity with mass effect on right caudate nucleus	1 (1.4)
Bilateral basal ganglia, thalamus, midbrain hypodensity	1 (1.4)
Bilateral putamen, globus pallidus hypodensity	1 (1.4)
Right lenticular nucleus and right caudate head	1 (1.4)
Right putamen	1 (1.4)
Striatum	1 (1.4)
Normal	5 (6.9)
N/R	9 (12.5)
N/A	33 (45.8)
Magnetic resonance imaging	
Bilateral basal ganglia atrophy	36 (50.0)
Bilateral putamen and global pallidus atrophy	7 (9.7)
Bilateral lenticular nuclei atrophy	2 (2.8)
Bilateral caudate and lenticular nuclei atrophy	4 (5.6)
Bilateral basal ganglia and thalami	2 (2.8)
Bilateral striatum atrophy	1 (1.4)
Right basal ganglia atrophy	1 (1.4)
Right lentiform nucelus, left putamen and right occipital lobe	1 (1.4)
Right lentiform nucleus	1 (1.4)
Right putamen	1 (1.4)
Striatum	1 (1.4)
Bilateral caudate, lentiform nucleus, frontal and temporal regions	1 (1.4)
Age-related atrophy	1 (1.4)
Normal	1 (1.4)
N/R	7 (9.7)
N/A	5 (6.9)
Magnetic resonance sepectroscopy	
Low N-acetyl-aspartate, presence of lactate in left basal ganglia	1 (1.4)
Low N-acetyl aspartate in bilateral basal ganglia	1 (1.4)
N/A	70 (97.2)

Table 1. Summary of study characteristics of 72 patients with ESKD and acute movement disorder (continued)

Characteristic	Value
Clinical course	
Partial recovery	28 (38.9)
Complete recovery	20 (27.8)
No improvement	15 (20.8)
Near complete recovery	5 (6.9)
N/R	4 (5.6)
Radiological course	
Partial resolution	18 (25)
Complete resolution	14 (19.4)
No resolution	6 (8.3)
Near complete resolution	3 (4.2)
N/R	14 (19.4)
N/A	17 (23.6)

Data are reperesented as median (range) or *n* (%). ESKD, end-stage kidney disease; DM, diabetes mellitus; N/R, not reported; N/A, not applicable; PET, positron emission tomography; SPECT, single photon emission computed tomography.

Case series

Case 1

A 49-year-old Malay man with T2DM and hypertension for 20 years and ESKD on regular hemodialysis for ten years presented with a three-month history of generalized 'involuntary movements' that was acute in onset. The movements worsened when he was stressed or performed specific tasks and affected his daily routine and sleep. There was no family history of similar illnesses or neurological disorders or history of hyponatremia (or rapid correction thereof). Examination revealed generalized chorea predominantly affecting his upper limbs and face, which worsened upon action and speaking. The rest of the neurological examination was normal. The Montreal Cognitive Assessment score was 23/30 (primary school education). The urea level was 11.5 mmol/L, serum creatinine level was 634 μmol/L, and HbA1C was 6.0%. Antinuclear antibody, anti-dsDNA, lupus anticoagulant and anti-cardiolipin antibodies, serum ceruloplasmin, manganese, and tumor markers (carcinoembryonic antigen, carbohydrate antigen 19-9, prostate-specific antigen, and alpha fetoprotein) were negative or within the normal range. Brain MRI showed symmetrical hyperintense lesions in the bilateral lentiform nuclei on T2WI and fluid-attenuated inversion recovery (FLAIR) (Figure 1A-C). MRA of the cerebral arteries showed right middle cerebral artery atherosclerosis (not shown). The dialysis regimen was optimized by his nephrologist, with daily dialysis and high-flux dialyzers to improve dialysis efficiency. Trials of medications including haloperidol (10 mg, bid), clonazepam (2 mg, bid), tetrabenazine (25 mg, bid), and amantadine

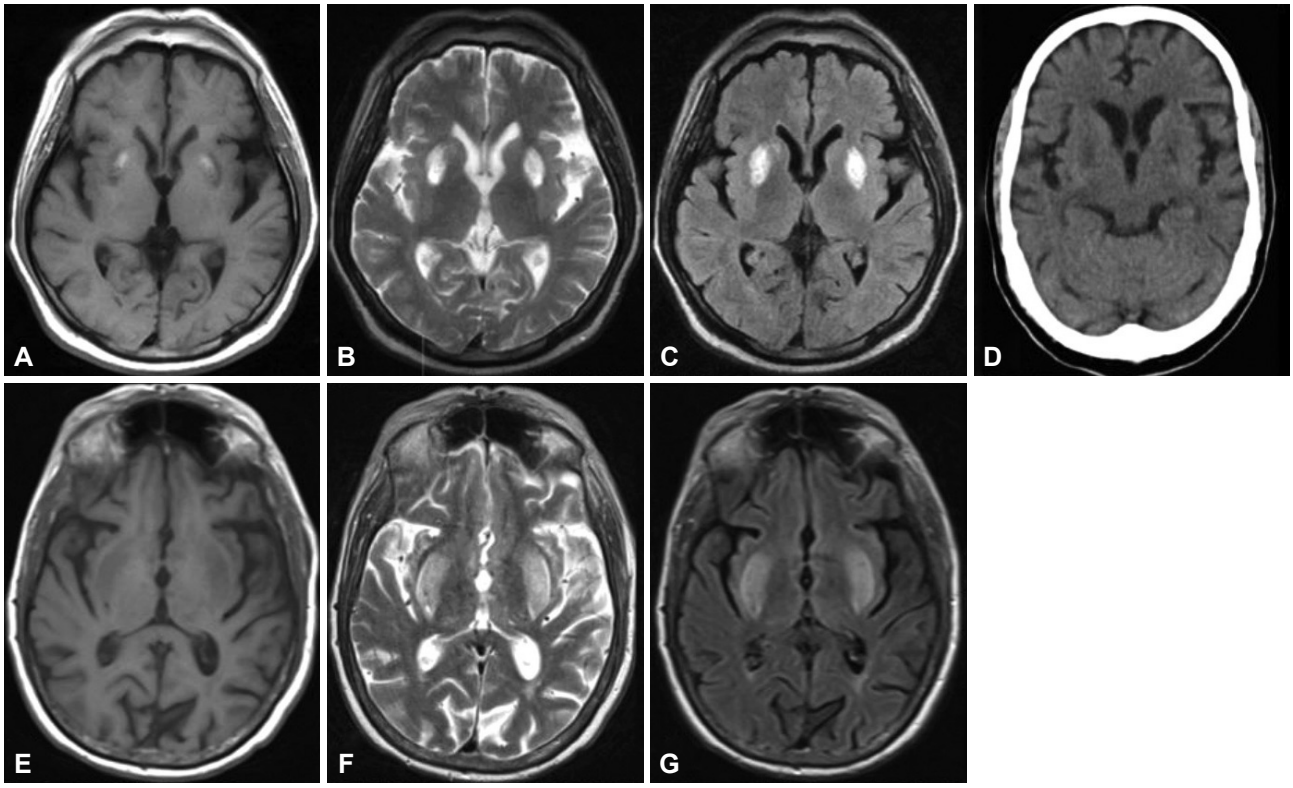

Figure 1. A 49-year-old man with end-stage kidney disease (ESKD) on hemodialysis for 10 years presented with generalized involuntary movements (case 1). MRI of the brain in axial T1-weighted (A), T2-weighted (B), and fluid-attenuated inversion recovery (FLAIR) (C) images. Symmetrical signal abnormalities in the bilateral lentiform nuclei, returning low signal intensity on the T1-weighted image and high signal intensities on the T2-weighted and FLAIR images. The symmetrical T1 hyperintense signals in the globus pallidi most likely represents calcification. A 43-year-old man with ESKD on regular hemodialysis presented with generalized body weakness and progressive slowness in movement and hypophonia (case 2). The unenhanced CT of the brain (D) reveals a symmetrical hypodensity in the lentiform nuclei. A 71-year-old man with ESKD on regular hemodialysis presented with bilateral choreiform movements (case 4). Magnetic resonance imaging of the brain in axial T1-weighted (E), T2-weighted (F), and FLAIR (G) images demonstrate abnormalities in the bilateral lentiform nuclei returning low signal intensity on the T1-weighted image and high signal intensities on the T2-weighted and FLAIR images.

(50 mg, bid) resulted in only minimal improvement despite multiple adjustments.

Case 2

A 43-year-old Malay man with long-standing T2DM, hypertension, and ESKD on regular hemodialysis for three months presented with a three-month history of generalized body weakness and progressive slowness in movement and hypophonia. He required a walking frame to ambulate at home. There was no history of recurrent falls, memory disturbances, or urinary incontinence. Physical examination revealed bilateral cogwheel rigidity, bradykinesia, and intermittent resting tremor of the left hand. His medications included amlodipine, perindopril, clopidogrel, sitagliptin, frusemide, and calcium carbonate. Unenhanced CT of the brain showed symmetrical hypodensities in the bilateral BG (Figure 1D). Blood urea and serum creatinine levels were not available as they were assessed in another center and were not available during the initial consultation. Following the initial consultation, the patient failed to return for subsequent follow-up. His family members provided a recent update that the patient had passed away two years after his initial visit due to an unrelated cause.

Case 3

A 68-year-old Indian man was diagnosed with T2DM, hypertension, and ESKD. He had been on regular hemodialysis since 2015 and presented in June 2018 for 'lack of strength' in his legs. He also complained of deterioration in the two months preceding his clinic visit, which led to the use of a wheelchair. There was no history of urinary symptoms, memory problems, or falls. On examination, there was hypomimia, jaw tremor, bradykinesia on finger taps and generalized rigidity consistent with parkinsonism. His renal profile showed elevated blood urea (18.8 mmol/L) and serum creatinine (914.8 μmol/L) levels. He was initiated on levodopa with modest improvement in his bradykinesia. Brain CT showed symmetrical hypodensities within the bilateral BG regions.

Case 4

A 71-year-old Indian man with hypertension and ESKD on regular hemodialysis at another center for four years presented with a three-day history of bilateral generalized choreiform movements involving the tongue, which caused difficulty ambulating and eating. He had received the Comirnaty vaccine (Pfizer-BioNTech) two weeks prior to his presentation. He also complained of fatigue, vomiting, and loss of appetite three weeks prior to presentation. On examination, there was moderate to severe generalized severe chorea, which also involved his tongue. His blood urea level was 20 mmol/L, and serum creatinine level was 1,000 μmol/L (one day after hemodialysis). Thyroid function test, serum ammonia, liver function test, diabetic screen, erythrocyte sedimentation rate, C-reactive protein, lupus screen, and anemia screen were normal or negative. Brain MRI showed bilateral lentiform hyperintensity on T2-weighted MRI and FLAIR (Figure 1E-G). He was treated with oral sulpiride and underwent another hemodialysis the same day. After dialysis, the chorea was less severe. EEG was normal. He had no further choreiform movements by the 3rd day.

DISCUSSION

We reported four ESKD patients with acute/subacute movement disorders associated with bilateral BG lesions and systematically reviewed 72 such cases from its initial description in 1998. Parkinsonism was the most common movement disorder, followed by chorea. The radiological findings were relatively homogenous with symmetrical bilateral BG lesions on CT and MRI,[3] with DWI and ADC hyperintensities indicating a possible vasogenic origin. Complete clinical resolution occurred in 30.9% of the patients, while radiological resolution occurred in only 34.1% of the patients, suggesting that this syndrome may be fully reversible, although we are unable to determine the predictors for full recovery based on this review. Recovery was associated with more intensive dialysis and appropriate management of the movement disorder. The patients presenting with chorea were more likely to fully recover than those with parkinsonism.

The underlying pathogenesis of this syndrome remains unclear. T2DM, hypertension, and ESKD may collectively lead to metabolic acidosis, oxidative stress, and microvascular changes, increasing the risk of ischemic damage to the BG. In support of this notion, MRS findings showed reduced metabolism,[4,5] and PET/SPECT scans showed hypoperfusion[6-9] within the BG. The presence of a lactate peak on MRS implied the possibility of selective mitochondrial dysfunction within the BG.[10] The abrupt onset and spontaneous recovery of the movement disorder in some patients suggested that acute ischemia may be the final insult.[11] Conversely, chronic and continual metabolic derangement in severe ESKD may lead to permanent damage to the BG structures,[12] which could explain the lack of clinical improvement with dialysis in some patients. A recent review suggested manganese toxicity as a cause of BG lesions in ESKD patients with parkinsonism.[13] However, only the patient in Case 1 had manganese levels tested, which were within the normal range.

Slightly more than half of the patients identified by this review were Asians. It is unclear whether this syndrome is more prevalent among Asians, similar to nonketotic hyperglycemia-induced chorea (NKHC), which also affects BG structures.[14] While this observation could be entirely due to higher reporting by researchers from Asian countries, it also raises the question of whether Asians are generally more predisposed toward metabolic BG insults.

Limitations and conclusion

There are several limitations in this review. First, clinical and radiological outcome measures were not consistently reported. Second, we were unable to determine the long-term outcome, as no prospective follow-up findings were reported in most of the reports. Nevertheless, we believe this review adds to the expanding literature on this syndrome and points toward the possibility of endogenic susceptibility among specific individuals. Future studies should investigate the long-term outcomes of this condition and determine the underlying predisposing causes.

Supplementary Materials

The online-only Data Supplement is available with this article at https://doi.org/10.14802/jmd.21185.

Conflicts of Interest

The authors have no financial conflicts of interest.

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None

Author Contributions

Conceptualization: Nurul Husna Baharudin, Norlinah Mohamed Ibrahim. Data curation: Kah Hui Yap, Nurul Husna Baharudin. Formal analysis: Kah Hui Yap. Investigation: Kah Hui Yap, Nurul Husna Baharudin. Methodology: Kah Hui Yap, Nurul Husna Baharudin. Project administration: Kah Hui Yap, Nurul Husna Baharudin. Resources: Kah Hui Yap. Supervision: Norlinah Mohamed Ibrahim. Validation: Norlinah Mohamed Ibrahim. Visualisation: all authors. Writing—original draft: Kah Hui Yap, Nurul Husna Baharudin. Writing—review & editing: Abdul Halim Abdul Gafor, Rabani Remli, Shen-Yang Lim, Wan Asyraf Wan Zaidi, Shahrul Azmin, Shahizon Azura Mohamed Mukari, Raihanah Abdul Khalid, Norlinah Mohamed Ibrahim.

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Supplementary Table 1. Characteristics of end-stage kidney disease patients with acute movement disorders

Study/country	Main clinical characteristics	PET/SPECT	CT	MRI	Clinical course	Radiological course
Bhagwan et al.[1] (2018)/ South Africa	Age: case 1–7, 44–68 years Ethnicity: Indian Premorbid/comorbid: DM (n = 6), hypertension (n = 1) Duration of (ES)RD: case 1–7, 8–75 days Duration of dialysis prior to AMD: 1–2 years Type of AMD: Parkinsonism (n = 5), chorea (n = 1), dystonia (n = 1)	N/A	Bilateral basal ganglia hypodensities (n = 2)	Hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2-weighted MRI (n = 6) Vasogenic and cytotoxic oedema on DWI and ADC	Levodopa for parkinsonism, haloperidol for chorea, and combination of risperidone and clonazepam for dystonia Two patients showed complete recovery Five patients showed partial recovery One patient had no improvement	Complete resolution (n = 2)
Chen et al.[2] (2015)/Taiwan	Age: 56 years Ethnicity: not specified Premorbid/comorbid: DM Duration of (ES)RD: 10 years Duration of dialysis prior to AMD: 1 years Type of AMD: dyskinesia	N/A	Bilateral basal ganglia hypodensities	Hypointensity in bilateral caudate and lenticular nuclei in T1; hyperintensity in same regions and external capsules, thalami, and periventricular WM in T2-weighted MRI	Regular hemodialysis and haloperidol resulted in complete resolution	N/A
Choi et al.[3] (2015)/Korea	Age: 60 years Ethnicity: Korean Premorbid/comorbid: DM Duration of (ES)RD: 6 years Duration of dialysis prior to AMD: 6 years Type of AMD: Parkinsonism	SPECT: hypoperfusion in bilateral basal ganglia	N/A	Hyperintensity in bilateral lenticular nuclei (basal ganglia) in T2-weighted MR and ADC	Partial improvement with dialysis (stabilization of urea nitrogen [BUN] and creatinine levels)	N/R
Cupidi et al.[4] (2006)/Italy	Age: 68 years Ethnicity: Caucasian Premorbid/comorbid: DM, hypertension, coronary heart disease Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/A Type of AMD: Parkinsonism	N/A	Bilateral basal ganglia hypodensity	Hypointensity in bilateral caudate and lenticular nuclei in T1; hyperintensity in same regions and external capsules, thalami, and periventricular WM in T2-weighted MRI	Complete recovery with insulin therapy (decreased blood creatinine level and blood glucose was well controlled)	N/R
Dicuonzo et al.[5] (2010)/Italy	Age: 51 years Ethnicity: Caucasian Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: chorea	N/A	Bilateral lenticular nuclei hypodensity	Hypointensity in bilateral basal ganglia (sparing caudate) in T1; hyperintensity in same regions in T2-weighted MR and FLAIR; hyperintensity with high ADC on same regions in DWI; decreased N-acetyl-aspartate and presence of lactate in left basal ganglia lesion on Proton MRS	Partial improvement with olanzapine with relapse-remitting course	N/R
Fabiani et al.[6] (2013)/Brazil	Age: 60 years Ethnicity: not specified Premorbid/comorbid: N/R Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: Parkinsonism	N/A	N/A	Hyperintensity in bilateral basal ganglia (caudate, putamen, thalamus)/LFS in FLAIR	Complete recovery with dialysis and levodopa/benserazide	Complete resolution
Fernandes et al.[7] (2015)/Brazil	Age: 63 years Ethnicity: not specified Premorbid/comorbid: N/R Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: Parkinsonism	N/A	Bilateral basal ganglia hypodensities	Vasogenic edema with T2/fluid-attenuated inversion recovery hyperintensity in the basal ganglia compatible with the LFS	N/R	N/A
Hamed et al.[8] (2020)/Egypt	Age: Case 1: 58 years Case 2: 62 years Case 3: 52 years Case 4: 60 years Case 5: 58 years Case 6: 60 years Ethnicity: not specified Premorbid/comorbid: Case 1: DM Case 2: DM Case 3: hypertension and congenital renal agenesis Case 4: DM Case 5: DM, hypertension Case 5: hypertension Duration of (ES)RD: Case 1: 3 years Case 2: 5 years Case 3: 2 years Case 4: 8 years Case 5: 3 years Case 6: 6 years Duration of dialysis prior to AMD: Case 1: 1 years Case 2: N/A Case 3: N/A Case 4: 2 years Case 5: N/A Case 6: 3 Type of AMD: Case 1: Parkinsonism Case 2: chorea Case 3: Parkinsonism Case 4: Parkinsonism Case 5: Parkinsonism Case 6: dystonia	N/A	Case 3: unremarkable Case 4: normal Case 6: hypodensity in the right putamen	Case 1: hypointensity in the right lentiform nucleus, left putamen, and the right occipital lobe in T1; hyperintensity in the same regions in T2 and DWI Case 2: hypointensity in the putamen and globus pallidus externa in T1; hyperintensity in the same regions in T2 and DWI Case 3: hypointensity in the bilateral basal ganglia and thalami in T1; hyperintensity in the same regions in T2 and DWI Case 4: hypointensity in the right putamen and globus pallidus externa in T1; hyperintensity in the same regions in T2 and DWI Case 5: hypointensity in the right lentiform nucleus in T1; hyperintensity in the same regions in T2 and DWI Case 6: hypointensity in the right putamen in T1; hyperintensity in the same regions in T2 and DWI	Case 1: no recovery Case 2: no recovery Case 3: no recovery Case 4: partial improvement Case 5: no recovery Case 6: no recovery	Case 1: complete resolution Case 2: complete resolution Case 3: complete resolution Case 4: N/A Case 5: near complete resolution Case 6: N/A
Ishii et al.[9] (2016)/Japan	Age: 47 years Ethnicity: Japanese Premorbid/comorbid: DM Duration of (ES)RD: 4 years Duration of dialysis prior to AMD: 4 years Type of AMD: Parkinsonism	PET: hypoperfusion in bilateral striatum	N/A	Hypointensity/edematous in bilateral striatum (especially putamen) Hyperintensity in same regions in T2-weighted MRI; cystic lesion in bilateral putamen (vacuolated changes following focal necrosis	No clinical improvement with levodopa/ benserazide	N/R
Kim et al.[10] (2006)/Korea	Age: 55 years Ethnicity: Asian Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD : 4 years Type of AMD: Parkinsonism	N/A	N/A	Hypointensity/edema in bilateral basal ganglia Hyperintensity in same regions in T2-weighted MRI; cystic lesion in bilateral putamen (vacuolated changes following focal necrosis	Partial recovery	Partial resolution
Kim et al.[11] (2015)/Korea	Age: Case 1: 45 years Case 2: 56 years Case 3: 56 years Case 4: 35 years Case 5: 48 years Ethnicity: Korean Premorbid/comorbid: Case 1–5: DM Duration of (ES)RD: 9.5–20 years Duration of dialysis prior to AMD (years): N/R Type of AMD: chorea	N/A	Case 1: hypointensity in bilateral putam en and globus pallidus Case 2: N/A Case 3: not contributable Case 4: N/A Case 5: N/A	Case 1: DWI hyperintensity in the bilateral putamen and globus pallidus Case 2: hypointensity in T1; hyperintensity in T2 in the same regions with swelling Case 3: not contributable Case 4: hyperintensity in the center and hypointensity in the periphery in T1, hyperintensity in the same regions in T2 with swelling Case 5: hypointensity in T1; hyperintensity in T2 in the same regions	Case 1: partial recovery Case 2: complete recovery Case 3: partial recovery Case 4: partial recovery Case 5: complete recovery	Case 1: N/A Case 2: no resolution Case 3: N/A Case 4: N/A Case 5: no resolution
Kiryluk et al.[12] (2008)/ United States	Age: 71 years Ethnicity: Hispanic Premorbid/Comorbid: DM, hypertension, hypercholesterolemia, depression, hypercalcemia, submandibular and perihilar lymphadenopathy, sarcoidosis, hyperparathyroidism Duration of (ES)RD: 3 years Duration of dialysis prior to AMD: 3 years Type of AMD: chorea	N/A	N/A	Hyperintensity in bilateral putamen and global pallidus on T2-weighted MR and FLAIR	Complete recovery with conservative management and rehabilitation	Complete resolution
Kumar and Goyal[13] (2010)/ United States	Age: 48 years Ethnicity: not specified Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: N/R	N/A	Bilateral basal ganglia, thalamus, and midbrain hypodensity	Hyperintensity in bilateral basal ganglia (caudate, putamen, thalamus)/LFS, midbrain, and mesial temporal lobes on T2/FLAIR	N/R	Complete resolution
Kuppachi et al.[14] (2013)/ United States	Age: 58 years Ethnicity: Hispanic Premorbid/comorbid: DM, hypertension, peripheral vascular disease, hypothyroidism, sarcoidosis Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD: 4 years Type of AMD: chorea	N/A	Bilateral basal ganglia hypodensity	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2	Partial improvement with conservative management	N/A
Lee et al.[15] (2007)/Korea	Age: Case 1–4, 44–68 years Ethnicity: Korean Premorbid/comorbid: DM Duration of (ES)RD: 0.5–2 years Duration of dialysis prior to AMD (years): N/R Type of AMD: chorea, dysarthria	N/A	Bilateral basal ganglia hypodensity	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 Increased ADC in DWI	Partial improvement with dialysis and supportive management	Partial resolution
Lee et al.[16] (2006)/Korea	Age: 48 years Ethnicity: Korean Premorbid/comorbid: DM Duration of (ES)RD: 15 years Duration of dialysis prior to AMD: 4 years Type of AMD: Parkinsonism	SPECT: hyperperfusion in bilateral basal ganglia	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 Increased ADC in DWI	Partial improvement with dialysis	Complete resolution
Li et al.[17] (2008)/Australia	Age: 77 years Ethnicity: Caucasian Premorbid/comorbid: DM, hypertension Duration of (ES)RD: 6 years Duration of dialysis prior to AMD: 6 years Type of AMD: Parkinsonism	N/A	Bilateral basal ganglia (lentiform and caudate nuclei) hypodensity	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 and FLAIR Hyperintensity in lentiform nuclei and caudate nuclei head in DWI	No clinical improvement with dialysis	No resolution
Lin[18] (2011)/ Taiwan	Age: Case 1: 57 years Case 2: 82 years Ethnicity: Chinese Premorbid/comorbid: Case 1: DM, hypertension Case 2: DM, hypertension, ischemic heart disease, lacunar infarct Duration of (ES)RD: Case 1: 5 years Case 2: 2 years Duration of dialysis prior to AMD : Case 1: 2 years Case 2: N/R Type of AMD: Case 1: chorea Case 2: chorea	N/A	N/A	Case 1: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2, FLAIR, and DWI (low ADC) Case 2: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2, FLAIR, and DWI (low ADC)	Case 1: complete recovery with dialysis and supportive management Case 2: complete recovery with haloperidol	Case 1: complete resolution Case 2: complete resolution
Lizarraga et al.[19] (2017)/ United States	Age: 47 years Ethnicity: Asian Premorbid/comorbid: DM Duration of (ES)RD: 2 years Duration of dialysis prior to AMD (years): N/R Type of AMD: chorea and parkinsonism	N/A	Striatal vasogenic edema surrounding areas of central cytotoxicity, blood extravasation and hyperdense lenticulostriatal arteries in the striatopallidal junctions	Striatal vasogenic edema surrounding areas of central cytotoxicity, blood extravasation and hyperdense lenticulostriatal arteries in the striatopallidal junctions	Near complete recovery	Partial resolution
Mahajan et al.[20] (2014)/India	Age : 52 years Ethnicity: Indian Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD: 4 years Type of AMD: chorea	N/A	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2, FLAIR, and ADC	Complete recovery	N/A
Mehta et al.[21] (2017)/India	Age: 57 years Ethnicity: not specified Premorbid/comorbid: DM Duration of (ES)RD: 4 years Duration of dialysis prior to AMD (years): N/A Type of AMD: Parkinsonism	N/A	N/A	Diffuse oedematous symmetrical T2 and fluid-attenuated inversion recovery hyperintensities in bilateral caudate and lentiform nuclei with mild diffusion restriction	Complete resolution with daily haemodialysis	Near complete resolution
Nzwalo et al.[22] (2012)/Portugal	Age: 63 years Ethnicity: Caucasian Premorbid/comorbid: DM, hypertension, schizoaffective disorder, and polysubstance abuse in remission Duration of (ES)RD: 3 years Duration of dialysis prior to AMD (years): 3 years Type of AMD: Parkinsonism	N/A	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 and FLAIR	Complete recovery	N/R
Ozben et al.[23] (2011)/Turkey	Age: 54 3 years Ethnicity: not specified Premorbid/comorbid: DM Duration of (ES)RD: 5 years Duration of dialysis prior to AMD: 3 years Type of AMD: Ballism	N/A	N/A	Hypointensity in bilateral l entiform nucleus in T1 Hyperintensity in same regions in T2 and FLAIR	Complete recovery	N/A
Park et al.[24] (2007)/Korea	Age: 68 years Ethnicity: not specified Premorbid/comorbid: DM, hypertension Duration of (ES)RD: 4 years Duration of dialysis prior to AMD: 1 years Type of AMD: chorea	N/A	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2, FLAIR, and ADC	Near complete recovery	Partial resolution
Park et al.[25] (2015)/Korea	Age: 52 years Ethnicity: not specified Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: Parkinsonism	PET: no abnormality	N/A	Hyperintensity in the bilateral basal ganglia in T2 FLAIR, DWI and ADC (LFS)	Near complete recovery without anti-Parkinson medication	Complete resolution
Rao et al.[26] (2019)/India	Age: 78 years Ethnicity: Indian Premorbid/comorbid: DM, hypertension Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/A Type of AMD: chorea	N/A	N/A	Age-related atrophy	Complete recovery with intravenous fluids and broad-spectrum intravenous antibiotics at doses that were modified according to renal function	N/R
Rathi and Mudrabettu[27] (2012)/India	Age: 35 years Ethnicity: Indian Premorbid/comorbid: N/R Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: N/R	N/A	Hypodensity in bilateral basal ganglia with surrounding oedema	Hyperintensity in bilateral basal ganglia in T2/FLAIR with LFS	No recovery with intubation, hyperventilation, and by injecting dexamethasone to reduce the intracranial pressure with intensive dialysis support	No resolution
Rozenberg and Telman[28] (2012)/ Israel	Age: 64 years Ethnicity: not specified Premorbid/comorbid: DM, hypertension, dyslipidaemia Duration of (ES)RD: N/R Duration of dialysis prior to AMD: N/R Type of AMD: chorea	N/A	Hypodensities in the bilateral basal ganglia	N/A	Complete recovery with tetrabenazine	Complete resolution
Shin et al.[29] (2008)/Korea	Age: 44 years Ethnicity: not specified Premorbid/comorbid: DM, depression Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/A Type of AMD: chorea & restless leg syndrome	N/A	N/A	Hyperintensity in bilateral basal ganglia and right thalamus in T2/FLAIR	Partial recovery	Partial resolution
Sperling and Bhowansingh[30] (2018)/ United States	Age: 62 years Ethnicity: Caucasian Premorbid/comorbid: DM, hypertension, hypercholesterolemia Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/A Type of AMD: chorea	N/A	Hyperdensity in the right lenticular nucleus and right caudate head	Hyperintensity in the caudate nucleus portion of the right basal ganglia in T1 and T2 Postcontrast MRI of the brain showed no abnormal enhancement excluding the possibility of a mass lesion No abnormal restricted diffusion in the right basal ganglia in DWI which excluded ischemia	Complete recovery with home insulin regimen with the addition of inpatient corrective coverage. This included Lantus 20 units every morning and 10 units of NovoLog 3 times a day with meals	N/R
Tajima et al.[31] (2012)/Japan	Age: 64 years Ethnicity: Caucasian Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD: 0.25 years Type of AMD: Parkinsonism	N/A	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 and ADC in DWI Low N-acetyl aspartate in same areas in Proton MRS	Partial improvement with dialysis and supportive management	Partial resolution
Wali et al.[32] (2011)/India	Age: 67 years Ethnicity: Indian Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD: 4 years Type of AMD: dyskinesia and chorea	N/A	N/A	Hypointensity in bilateral basal ganglia in T1 Hyperintensity in same regions in T2 and FLAIR Hyperintensity in ADC in DWI	Complete recovery with increasing frequency of dialysis	Near complete resolution
Wang et al.[33] (1998)/Taiwan	Age: Case 1: 50 years Case 2: 49 years Case 3: 49 years Ethnicity: Chinese Premorbid/comorbid: Case 1: DM, hypertension Case 2: DM Case 3: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD: Case 1: N/R Case 2: 0.67 years Case 3: N/R Type of AMD: Parkinsonism	N/A	Case 1: bilateral lenticular nuclei hypodensity Case 2: no a bnormality Case 3: basal ganglia and part of bilateral thalami hypodensity	Case 1: hypointensity in bilateral lenticular nuclei in T1; hyperintensity in same regions in T2 Case 2: hypointensity in bilateral lenticular nuclei in T1; hyperintensity in same regions in T2 Case 3: hypointensity in bilateral basal ganglia and part of bilateral thalami in T1; hyperintensity in same regions in T2	Case 1: partial improvement with oral biperiden and diazepam Case 2: partial improvement without specific treatment Case 3: partial improvement with dialysis	Case 1: NR Case 2: NR Case 3: partial resolution
Wang and Cheng[34] (2003)/Taiwan	Age: Case 1: 61 years Case 2: 45 years Case 3: 60 years Case 4: 58 years Case 5: 63 years Case 6: 52 years Case 7: 59 years Case 8: 67 years Case 9: 67 years Case 10: 50 years Case 11: 49 years Case 12: 49 years Ethnicity: Chinese Premorbid/comorbid: Case 1: DM, hypertension, coronary disease Case 2: DM, hypertension, anemia Case 3: DM, hypertension Case 4: DM, hypertension Case 5: DM, hypertension, stroke Case 6: DM, hypertension, seizures Case 7: DM Case 8: DM, hypertension Case 9: DM, hypertension Case 10: DM, hypertension, coronary disease Case 11: DM, coronary disease Case 12: DM Duration of (ES)RD: Case 1: 2.83 years Case 2: 4 years Case 3: 3 years Case 4: 3 years Case 5: 2 years Case 6: 2 years Case 7: 1 years Case 8: 5 years Case 9: 1 years Case 10: 1.58 years Case 11: 0.67 years Case 12: 0.25 years Duration of dialysis prior to AMD: Case 1: 2.33 years Case 2: 3 years Case 3: 3 years Case 4: 3 years Case 5: 2 years Case 6: 2 years Case 7: 1 years Case 8: 1 years Case 9: 1 years Case 10: 1.58 years Case 11: 0.67 years Case 12: None Type of AMD: Case 1: Parkinsonism Case 2: Parkinsonism, dysarthria, dysphagia Case 3: Parkinsonism, dysarthria, dysphagia Case 4: orolingual and facial dyskinesias, parkinsonian gaits, dysarthria, dysphagia Case 5: Parkinsonism, dysarthria, dysphagia Case 6: Parkinsonian gait Case 7: Parkinsonian gait, dysarthria Case 8: Parkinsonism, dysarthria Case 9: Parkinsonism, dysarthria Case 10: Generalised dyskinesia, ataxia, dysarthria Case 11: Parkinsonian gait, dysarthria Case 12: Parkinsonism, dysarthria, dysphagia	N/A	Case 1: bilateral basal ganglia hypodensity Case 2: N/A Case 3: bilateral basal ganglia hypodensity Case 4–12: /R	Case 1: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2 Case 2: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2 Case 3: N/R Case 4: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2 Case 5–6: N/A Case 7–12: N/R	Case 1: no clinical improvement with dialysis Case 2: partial improvement with dialysis and supportive management Case 3: no clinical improvement with dialysis and supportive management Case 4: partial improvement with clonazepam and biperiden Case 5: no clinical improvement Case 6: complete recovery Case 7: partial improvement Case 8: no clinical improvement Case 9: no clinical improvement Case 10: partial improvement Case 11: partial improvement Case 12: complete recovery	Case 1–2: N/R Case 3: partial resolution Case 4: partial resolution Case 5: no resolution Case 6: N/A Case 7: partial resolution Case 8: partial resolution Case 9: partial resolution Case 10: partial resolution Case 11: N/R Case 12: partial resolution
Wang et al.[35] (2004)/Taiwan	Age: Case 1: 67 years Case 2: 67 years Ethnicity: Chinese Premorbid/comorbid: Case 1, 2: DM Duration of (ES)RD: Case 1: N/R Case 2: 1 year Duration of dialysis prior to AMD: Case 1: 5 years Case 2: 1 year Type of AMD: Case 1: Parkinsonism, dysarthria Case 2: Parkinsonism	PET: Case 1: h ypoperfusion in bilateral putamen, right caudate, bilateral occipital cortex, right frontal cortex Case 2: hypoperfusion in putamen, left caudate, bilateral lateral frontal cortex	Case 1: bilateral lenticular nuclei hypodensity Case 2: bilateral basal ganglia hypodensity	Case 1: hypointensity in bilateral lenticular nuclei and caudate nuclei in T1; hyperintensity in same regions in T2 Case 2: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2	Case 1: partial improvement with dialysis and supportive management Case 2: N/R	Case 1: complete resolution Case 2: partial resolution
Yaltho et al.[36] (2010)/ United States	Age: 43 years Ethnicity: Hispanic Premorbid/comorbid: DM Duration of (ES)RD (years): N/R Duration of dialysis prior to AMD (years): N/R Type of AMD: chorea	N/A	Bilateral lenticular nuclei hypodensity with mass effect on right caudate nucleus	Hyperintensity in bilateral basal ganglia in T2 scan	Near complete improvement with dialysis and haloperidol (discontinued and change to pimozide after 1 week due to significant improvement)	N/R
Yerdelen et al.[37] (2008)/Turkey	Age: 52 years Ethnicity: not specified Premorbid/comorbid: DM, hypertension Duration of (ES)RD: 5 years Duration of dialysis prior to AMD: 5 years Type of AMD: Parkinsonism	N/A	Bilateral basal ganglia hypodensities	Hyperintensity in bilateral caudate and lentiform nucleus, frontal, and temporal regions in T2 and DWI and ADC	No improvement with supportive management and hemodialysis.	N/A
Yoon et al.[38] (2016)/Korea	Age: Case 1: 43 years Case 2: 60 years Ethnicity: Korean Premorbid/comorbid: Case 1: DM Case 2: DM, hypertension Duration of (ES)RD: Case 1: 15 years Case 2: 6 years Duration of dialysis prior to AMD: Case 1: 2 years Case 2: 6 years Type of AMD: case 1, 2, Parkinsonism	Case 1: N/A Case 2: normal on PET	Case 1: N/A Case 2: no abnormality	Case 1: hypointensity in bilateral basal ganglia in T1; hyperintensity in same regions in T2 and FLAIR Case 2: hyperintensity in bilateral basal ganglia in T2 and FLAIR; hyperintensity in left globus pallidus interna, anterior portion of the posterior limb of the internal capsule in DWI (low ADC)	Case 1: complete recovery with dialysis Case 2: near complete recovery	Case 1: no resolution Case 2: N/R

PET, positron emission tomography; SPECT, single photon emission computed tomography; DM, diabetes mellitus; ESRD, end-stage renal disease; AMD, acute movement disorder; DWI, diffusion weight imaging; ADC, apparent diffusion coefficient; N/A, not applicable; N/R, not reported; WM, white matter; FLAIR, fluid-attenuated inversion recovery; LFS, lentiform fork sign; MRS, magnetic resonance sepectroscopy.

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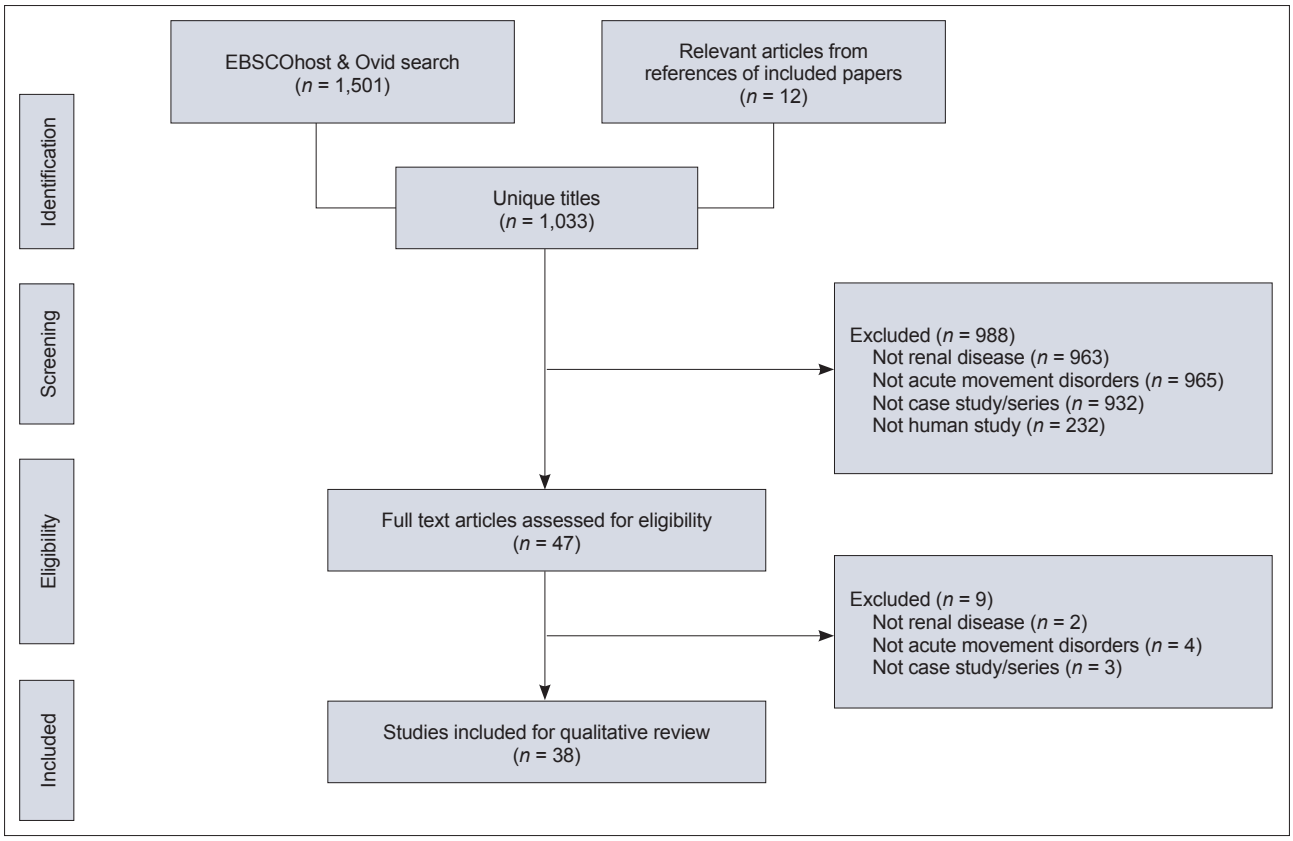

Supplementary Figure 1. Study search per the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guideline.