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ORIGINAL RESEARCH

Polydipsia-polyuria syndrome associated with traumatic spinal cord injury

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Abstract

Introduction: Polydipsia and polyuria associated with traumatic spinal cord injury (SCI) are rare consequences. The hypothetical pathophysiological mechanisms involve mild traumatic brain injury (TBI) and/ or vagus dysfunction associated with spinal cord injury.
Methods: In a retrospective study of 11 patients, we investigated associations between polydipsia-polyuria syndrome and various clinical and therapeutic factors: medullary section syndrome, neurological level, medication, neurosurgical intervention, kinesitherapy program, associated comorbidities, functional level at discharge and patient quality of life (QoL).
Results: The beginning of the kinetic program (Spearman correlation coefficient =0.631) and desmopressin treatment (Spearman correlation coefficient =0.708) had statistically significant effects on resolution of polydipsia-polyuria syndrome (PPS). Patient QoL was statistically significant improved after resolution of PPS (t-test, p =0.001).
Conclusions: Mobilization programs appear to promote resolution of PPS. Desmopressin treatment is beneficial and, together, medication and kinesitherapy elevate patients' QoL. The association between SCI and transient PPS requires additional investigation in additional patients.

Keywords

Polyuria; Polydipsia; Spinal cord injury

1. Introduction

Polydipsia-polyuria syndrome (PPS) associated with traumatic spinal cord injury (SCI) is rare. This syndrome occurs in patients with high neurological levels of SCI, injuries to the cervical or upper thoracic levels, with complete or incomplete medullary syndromes. Reported cases of PPS in SCI patients were diagnosed as full or partial central Diabetes Insipidus (DI) with or without neurosurgical intervention [1–3]. For example, Farel diagnosed a single male case of tetraplegia after SCI associated with partial DI [1]. Prasad described transient DI in three male SCI cases, two with injury at the cervical neurological level and one at the thoracic level [2]. Kusely presented a female case with paraplegia after SCI and central DI [3]. As we described above, the occurrence of DI associated with SCI is cited in just a few cases, but this challenging combination worsens patient quality of life (QoL) [4], requiring considerable physical, social, environmental, and psychological intervention to stabilize the patient and increase function [5]. However, the recognition, understanding, and proper management of this complex syndrome leads to prompt and appropriate neurorehabilitation [6].

The magnocellular nuclei of the hypothalamus is responsible for antidiuretic hormone (ADH) synthesis [7], which can be affected in several conditions, such as: hypophysis tumors (adenoma, pinealoma, craniopharyngioma), meningoencephalitis, stroke, aneurysms, autoimmune diseases, metastasis (breast or bronchi) [8, 9], nephrogenic pathologies, and, last but not least, traumatic brain injury (TBI), which is one of the main causes of central DI [10, 11].

Recognition of multiple causes of altered ADH secretion like Salt-wasting syndrome (SWS), syndrome of inappropriate antidiuretic hormone (SIADH), and DI is important when evaluating a patient who has suffered a complex polytrauma such as SCI asociating polyuria and polydipsia.

In the present study of SCI patients, we aimed to investigate associations between PPS and various factors: neurological (medullary section syndrome, neurological level) therapeutical (medication, neurosurgical intervention, kinesitherapy program), clinical (comorbid pathologies) and functional outcomes.

2. Method

Of the 4570 patients diagnosed with traumatic SCI and admitted between 2005–2020 in the Neuro-Muscular Rehabilitation

TABLE 1. Demographic characteristics of cases: age, spinal cord injury (SCI), neurological level, American spinal Injury Association Impairment Scale (AIS), score.

No.	Age (years)	SCI	Neurological level	AIS score
1	36	luxation C5-C6 and C7-T1	C7	A
2	32	fracture C5 and luxation C5-C6	C4	A
3	25	fractures and luxation C6-C7	C6	A
4	20	fracture T1	T1	A
5	43	fractures C5-C6	C5	A
6	20	fractures C6-C7	C5	A
7	27	fractures C5-C6	C5	A
8	63	fractures and luxation C6-C7	C5	A
9	63	luxation C6-C7, myelopathy C3-T1	C4	A
10	69	myelopathy with compression C3-C8	C3	C
11	45	myelopathy and compression C3-C6	C2	C

Department, only 11 fulfilled the following inclusion and exclusion criteria.

Inclusion criteria included: patients diagnosed with traumatic SCI, over 18 years old, with polyuria (over 3 liters urine by day) and polydipsia (over 3 liters liquids ingested by day). Exclusion criteria included: any of the following SCI-associated pathologies, noted in the past or in present: moderate or severe TBI; hypophysis tumors (adenoma, pinealoma, craniopharyngioma): meningoencephalitis: stroke; aneurysms; autoimmune diseases; metastasis (breast or bronchi); acute or chronic renal failure; hypokalemia (<3.5 mEq/L); hypercalcemia (>5.2 mEq/L); diabetes mellitus; amyloidosis; myeloma; and, uropathies. TBI severity was classified using the Glasgow Coma Scale (GCS), as follows: severe TBI GCS 3–8 points; moderate TBI GCS 9–12 points; and mild TBI GCS 13–15 points [10].

Evaluated parameters: demographic data were included for all 11 patients. Various clinical and paraclinical assessments were also included. Polyuria was confirmed by a 24-hour urine collection; polydipsia was confirmed by monitoring fluid ingestion. Blood glucose, urea and serum creatinine concentrations were used to exclude diabetes mellitus and kidney failure. Serum electrolyte concentrations at patient admission and discharge were used to exclude hypokalemia, hypercalcemia (may cause nephrogenic DI) and hyponatremia (may cause inappropriate antidiuretic hormone secretion). Computed tomography (CT) scans of the brain at patient admission were included.

Additional tests, including plasma and urinary osmolarity, plasma level of vasopressin (reference values: 2–8 ng/L) and the ability to concentrate after administration of exogenous vasopressin to evaluate for dehydration, were performed in only one patient for monetary reasons.

In addition, at admission and discharge, patients were evaluated using the American Spinal Injury Association Impairment Scale, (AIS); patients were assessed for medullary section syndrome, and their neurological level determined [12]. Quality of life was evaluated using Flanagan's Quality of Life (QoL) Scale at occurrence of polydipsia-polyuria and after its resolution [13]. The Functional Independence Measure score (FIM) was obtained at patient admission and discharge [14]. Comorbidities and specific complications were included. The time intervals between occurrence and resolution of various clinical, pathological, or functional events (SCI, the necessity of surgical intervention, polyuria polydipsia, the kinetic program, or desmopressin treatment) were calculated.

Patients' therapeutical management: all patients received necessary and appropriate treatment for their conditions and comorbidities, in accordance with current best practices and their therapeutic needs.

All patients benefited by kinesitherapy procedures after they received approval for mobilization from a neurosurgeon (mobilization data). The kinesitherapy was daily provided by a kinesiotherapist and consisted of specific exercises for 60 minutes per day. The objectives were use of a wheelchair, standing or walking, according to the neurological deficit of every patient.

All 11 patients were treated with desmopressin, a synthetic analog of vasopressin antidiuretic hormone, which acts as a selective agonist of V2 receptors expressed in the renal collecting duct to increase water re-absorption and reduce urine production [15]. Sublingual desmopressin, as desmopressin lyophilizate, was administered to patients as a sublingual melt tablet [15].

Statistical analysis: demographic characteristics were examined using descriptive statistical methods. To analyze QoL we used a paired t-test, with a p-value < 0.01 considered statistically significant. Data were examined for normality of distribution, and correlations between different time intervals were examined using the Spearman correlation test.

To enable statistical analysis, time intervals (in days) were calculated as follows: the total number of days between the date when the traumatic SCI occurred and the date of neurosurgical intervention; the total number of days between the date when the traumatic SCI occurred and the date the patient started mobilization; the total number of days between the date when the traumatic SCI occurred and the date of polyuria and polydipsia onset; the total number of days between the day when the traumatic SCI occurred and the date when polyuria and polydipsia were resolved; the total number of days the

patient received desmopressin treatment; and, the total number of days when the patient experienced polyuria and polydipsia (i.e., the difference between the date of occurrence and resolution).

3. Results

3.1 Demographic data

All patients were males, with an age range between 20 and 69 years, with a median age 36 years (Table 1, Fig. 1).

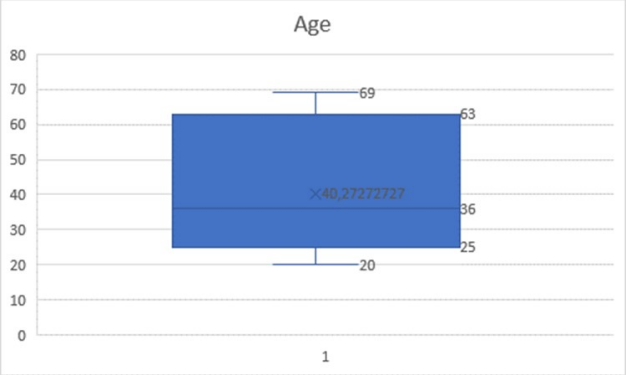

FIGURE 1. SCI patients age data, box and whisker plot.

3.2 Medullary section syndrome and AIS degree score

Nine patients had complete medullary section syndrome, having A degree on the AIS scale; and, 2 patients had incomplete medullary section syndrome, having C degree on the AIS scale (Table 1).

3.3 Neurological level

Of the 11 patients analyzed, 10 had cervical neurological level SCI (C SCI) and one had thoracic neurological level SCI (TSCI) (Table 1). For the 10 patients with C SCI neurological levels: 2 had the highest cervical level (C2-C3) and 8 had injuries at the C5-C6 level. The patient with the T SCI had an injury at T1.

3.4 Mobilization program and functional level at discharge

There was a strong correlation between resolution of polydipsia-polyuria and when the mobilization program started. The temporal reference point for the two parameters was the moment of SCI occurrence (Table 2, Spearman correlation coefficient = 0.631; Fig. 1). The mobilization program was active, i.e., the patient was raised from bed and taken to the gym rooms for kinesitherapy. At discharge, the functional level of all patients with complete medullary section syndrome was wheelchair-bound. For patients with incomplete medullary section the functional level at discharge was ambulatory for the younger patient (45 years old) or wheelchair for the oldest, (69 years old).

3.5 Association of polydipsia and polyuria with neurosurgical interventions for SCI

All patients underwent neurosurgical intervention on the spine. Transient polydipsia-polyuria symptom occurrence and neurosurgical intervention were weakly correlated (temporal reference point, the moment of SCI occurrence, (Table 2, Spearman correlation coefficient = 0.461; Fig. 2).

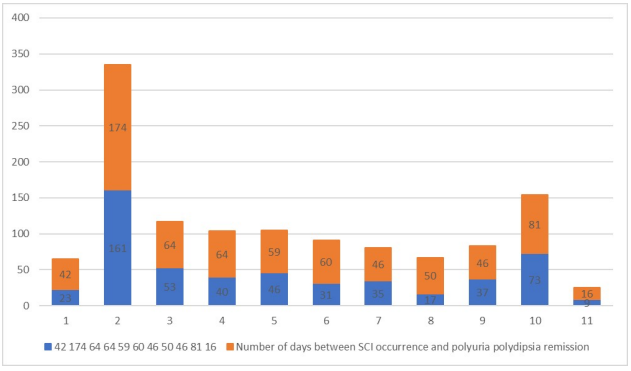

FIGURE 2. Number of days between SCI occurrence and occurrence of polydipsia-polyuria (blue) and Number of days between SCI occurrence and resolution of PPS (red).

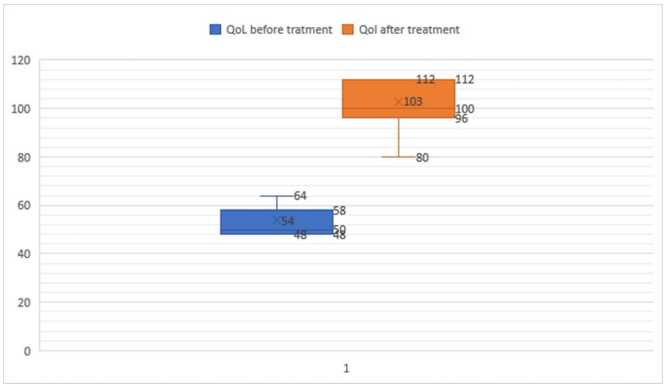

FIGURE 3. Quality of life scores measured before (blue) and after (red) desmopressin treatment.

3.6 Treatment of PPS

All patients who experienced clinical symptoms of polydipsia-polyuria received desmopressin. The number of days of desmopressin treatment and the number of days of polydipsia-polyuria were strongly correlated (Table 2, Spearman correlation coefficient = 0.708).

Five patients were treated with 60 mcg of desmopressin daily, and six patients received 120 mcg daily for 6 patients; for both groups, half of the dose was given in the morning and the other half in the evening. Importantly, plasma Natrium (Na) concentrations and other clinical parameters were monitored in all treated patients. Just one patient had a low level of plasma Na from the beginning of desmopressin treatment (Table 3). All patients needed monitoring the level of plasma Na and its constant correction during the treatment with desmopressin, in order to have normal range of plasma Na level when treatment with desmopressin finished (Table 3).

TABLE 2. Spearman correlations.

No.	Number of days between SCI occurrence and neurosurgical intervention	Number of days between SCI occurrence and mobilization	Number of days between SCI occurrence and polydipsia-polyuria occurrence	Number of days between SCI occurrence and resolution of polydipsia-polyuria	Number of days of desmopressin treatment	Number of days of polydipsia-polyuria
1	8	25	23	42	45	19
2	40	159	161	174	23	13
3	13	29	53	64	11	11
4	5	53	40	64	10	24
5	31	26	46	59	17	13
6	5	30	31	60	32	29
7	3	25	35	46	31	11
8	9	31	17	50	30	33
9	9	37	37	46	8	9
10	7	58	73	81	7	8
11	6	30	9	16	6	7

Spearman correlation coefficient between Number of days between SCI occurrence and mobilization and the Number of days between SCI occurrence and resolution of polydipsia-polyuria = 0.631 (strongly correlated).

Spearman correlation coefficient between Number of days between SCI occurrence and neurosurgical intervention and the Number of days between SCI occurrence and polydipsia-polyuria occurrence = 0.461 (weakly correlated).

Spearman correlation coefficient between Number of days of desmopressin treatment and the Number of days of polydipsia-polyuria = 0.708 (strongly correlated).

TABLE 3. Creatinine clearance, serum urea concentration, serum creatinine concentrations and natremia before and after desmopressin treatment in SCI patients.

No	Creatinine clearance before treatment	Creatinine clearance after treatment	Urea before treatment mg/dL	Urea after treatment mg/dL	Creatinine before treatment mg/dL	Creatinine after treatment mg/dL	Na before treatment	Na after treatment
1	104	108	40	25	0.9	0.8	127	142
2	100	124	14	13	0.5	0.4	144.6	138
3	141	112	25	30	1.1	1.0	138	140.5
4	114	122	25	13	0.6	0.8	135.5	135
5	100	116	5	10	0.4	0.5	139	143
6	120	127	31	29	0.9	0.6	135.4	137
7	100	119	38	20	0.8	0.7	135	137
8	100	111	27	24	0.85	0.7	135	128
9	110	100	21	26	0.7	0.6	130	128
10	100	120	19	10	0.7	0.8	135	131
11	100	110	41	20	0.7	0.7	135	132

TABLE 4. Functional independence measure evaluated at patients' discharge (FIM at discharge), patients' Flanagan's Quality of Life measured before (QoL before treatment) and after (QoL after treatment) desmopressin treatment.

No.	FIM at discharge	QoL before treatment	QoL after treatment
1	70	48	100
2	57	50	112
3	53	58	96
4	75	60	100
5	42	64	112
6	56	58	96
7	52	48	112
8	50	50	100
9	50	48	80
10	50	50	112
11	95	56	112

t-test, $p = 0.001$.

3.7 Quality of life score

After mobilization program and desmopressin treatment, all the patients' QoL was improved (mean QoL score of 54 at admission and 103 at discharge, $p = 0.001$, *t*-test, Fig. 3, Table 4), clinical symptoms of polyuria and polydipsia were resolved and the increase in QoL score was at least 36 points.

3.8 Associated comorbidities

All patients had mild TBI [16], with normal CT scans at admission. Six of the 11 patients, (including patient number 8), were diagnosed with depression and received appropriate treatment (Table 5). The remaining patients were not diagnosed with clinical depression, but they all had depression mood, requiring psychological support. Any other comorbidities have no known impact on the polyuria and polydipsia that occur in patients with SCI (Table 5).

3.9 Paraclinical diagnostic features

For patient number 8, plasma and urinary osmolarity and plasma vasopressin were normal before and after vasopressin treatment.

4. Discussion

4.1 PPS diagnosis

In the present study of SCI patients, transient polyuria and polydipsia lowered quality of life and slowed rehabilitation [6]. Urinary and plasma vasopressin and osmolality should be measured together to compensate for the insensitivity of the commercially available vasopressin and osmolality kits [17]. Regarding paraclinical diagnosis of DI in case of patient number 8, the clinical indicators were negative. Although there may be some error in the kits used by the laboratory, we cannot conclude that the patients in the present study had PPS due to DI. Another case report of a patient with transient polydipsia-polyuria associated with SCI described a psychogenic cause [18]. Therefore, we acknowledge the hypothesis that PPS in SCI patients might have a psychogenic cause. Consistent with this hypothesis, we observed that all patients in the present study had depressed mood or a clinical depression diagnosis.

It is important to underline the similarities and differences between PPS and the other pathologies like DI [19], SIADH [20, 21] and SWS [22] (Table 6). All have in common polydipsia and polyuria. Regarding hyponatremia PPS is similar with SWS and SIADH. Considering treatment, PPS has similarities with DI and SWS. So, PPS could be a new cause of altered ADH secretion.

TABLE 5. Patients' associated comorbidities.

No.	Associated comorbidities
1	mild TBI, UTI, depression, obesity
2	mild TBI, UTI, depression, insomnia, pneumonia
3	mild TBI, UTI, dyslipidemia, depressed mood
4	mild TBI, UTI, fracture of left tibia, depressed mood
5	mild TBI, ankylosis spondylitis, obesity, depressed mood
6	mild TBI, UTI, depressed mood
7	mild TBI, right leg thrombophlebitis, pleurisy, depressed mood
8	mild TBI, UTI, depression, pressure sores
9	mild TBI, UTI, depression, pressure sores
10	mild TBI, depression, COPD, pneumonia, pressure sores
11	mild TBI, depression, pneumonia, HT

TBI, Traumatic Brain Injury; UTI, Urinary Tract Infection; COPD, Chronic Obstructive Pulmonary Disease; HT, Hypertension.

TABLE 6. Main characteristics of Diabetes insipidus, Syndrome of inappropriate antidiuretic hormone, Salt-wasting syndrome and Polyuria polydipsia syndrome.

Pathology	Symptoms	Main causes	Paraclinical	Treatment
Diabetes insipidus	Polyuria	Central nervous system disturbances	Hypernatremia	Desmopressin
		Nephrogenic		
		Dipsogenic	Hyperosmolarity	
		Psychiatric		
	Polydipsia	Iatrogenic	Low ADH	
		Gestational		
Syndrome of inappropriate antidiuretic hormone	Polyuria	Central nervous system disturbances	Hyponatremia	Fluids restricted
		Neoplasia	Hypoosmolality	
		Pulmonary diseases Iatrogenic		
	Polydipsia	Genetic	ADH increased	
		Others		
Salt-wasting syndrome	Polyuria	Aneurysmal subarachnoid hemorrhage	Hyponatremia	Fluids and sodium supplementation
		Brain infection	Hypovolemia	
	Polydipsia	Brain tumor	Urine hyperosmolarity	
		Traumatic brain injury	Elevated urine sodium	
Polyuria polydipsia syndrome	Polyuria	Spinal cord injury	Hyponatremia	Desmopressin
	Polydipsia		Normal values of osmolarity	Fluids and sodium supplementation

4.2 Demographic data

All patients in the present study were male, but there are reported cases of female SCI patients with PPS [3]. Thus, we cannot conclude that male gender is a causal factor. However, SCI occurs more frequently in males [23], and, consequently, the association between transient DI and SCI is also more common in males.

4.3 Neurological level

SCI patients with injuries at the cervical and upper thoracic neurological level might have associated transient polyuria polydipsia because of the route of the vagus nerve, which descends from the medulla oblongata through the chest and abdomen [24]. It is possible that activity of the vagal nerve might be impaired in this group of patients, but there is insufficient information to determine if this is the case. However, SCI patients with injuries at the lower thoracic level (T10, T11, T12) or lumbar level did not exhibit symptoms of transient DI.

4.4 Neurosurgical intervention

There was a weak correlation between neurosurgical intervention and the occurrence of polydipsia-polyuria and, thus,

insufficient evidence to conclude there is a relationship between the two. However, Kuzeyli maintains that there is a potential effect of neurosurgical intervention on occurrence of polydipsia-polyuria after SCI [3, 25].

Desmopressin is a relatively safe drug [15]; however, its major adverse effect is hyponatremia [15]. Frisbie cited the association of low level of plasma Na and polydipsia for SCI patients with the injuries at a high neurological level [26]. In the present study, only one patient had hyponatremia at the beginning of desmopressin treatment. Nevertheless, hyponatremia could play a specific role in the polydipsia-polyuria syndrome that occurs in SCI patients treated with desmopressin. Thus, maintenance of plasma Na within the normal range is very important [26].

4.5 Quality of life, kinetic program and medication

Both the occurrence and resolution of PPS are strongly correlated with the patients' mobilization/kinetic program. On the other hand, the number of days of desmopressin treatment and the number of days of polydipsia-polyuria were also strong correlated, taking into account that all the patients received kinesitherapy and medication. Overall, the patients' quality of life was significantly improved with this therapeutic approach. It is possible that all components of this successful treatment strategy—the mobilization of the patient, physical exercises, and desmopressin treatment—contribute to be the best management approach for these patients [25].

Patients received desmopressin due to its clinical efficacy in symptom reduction [15]. However, all patients required adjustment of their low sodium levels induced by desmopressin to maintain Na within the normal range [15]. Desmopressin treatment remains important for patients' QoL despite this continuous medical intervention to adjust Na levels [25, 27], taking into account the pulsatile secretion of vasopressin after SCI, as highlighted by monitoring of plasma copeptin in a recent study [28].

From our point of view, a medical World Health Organization International Classification of Disease (ICD) code [29] should be established for this specific pathology: transient polyuria polydipsia associated with SCI. This would allow studies of cases from multiple medical centers.

In a brief, the major limitation of this case series is the lack of paraclinical evaluation to permit diagnosis of DI. Another limitation is the small number of cases. However, considering the literature regarding the association of SCI with polydipsia-polyuria consisting mainly of case studies [2, 3, 30], the small number of patients is acceptable. On the other hand, the beneficial role of kinesitherapy and desmopressin medication in the patients' QoL are the main conclusions of our research, and we consider this to be our "take home medical message".

5. Conclusions

The mechanisms responsible for the transient PPS associated with SCI are not entirely known. Hypothetical mechanisms include mild TBI or vagus nerve dysfunction. Clincal depression or depressed mood might influence the appearance of transient polyuria polydipsia. There is no evidence that neurosurgical interventions have a negative impact on development of polydipsia-polyuria. Mobilization programs appear to promote resolution of PPS. Desmopressin treatment is beneficial and, together, medication and kinesitherapy significantly elevate patients' QoL. The association between SCI and transient PPS requires additional investigation in additional patients.

ABBREVIATIONS

SCI, Spinal Cord Injury; TBI, Traumatic Brain Injury; QoL, Quality of Life; PPS, Polydipsia-Polyuria Syndrome; DI, Diabetes Insipidus; ADH, Antidiuretic Hormone; SWS, Like Salt-Wasting Syndrome; SIADH, Syndrome of Inappropriate Antidiuretic Hormone; GCS, Glasgow Coma Scale; CT, Computed Tomography; AIS, American Spinal Injury Association Impairment Scale; FIM, Functional Independence Measure; Na, Natrium; ICD, International Classification of Diseases.

AUTHOR CONTRIBUTIONS

Conceptualization: CD, GO; Methodology: CD, CM, IA, AS, CP, CT; Formal analysis and investigation: CD, CM, IA; Writing - original draft preparation: CP, CT, AS; Writing - review and editing: CD, GO,CM, IA; Resources: AS, CP, CT, Supervision: CD.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This case study was approved by the Ethics Commission of the Clinical Emergency Hospital "Bagdasar Arseni", Bucharest, Romania, No. 29307/18.11.2020.

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CONFLICT OF INTEREST

The authors declare no conflict of interest. Cristina Daia, Constantin Munteanu, Cristina Popescu, and Gelu Onose are co-Guest Editors of this journal.

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