CASE REPORT

Recurrent bilateral frontal lobe lesion with maladaptive schema modes and post-traumatic stress disorder symptoms: a case study

Sabir Zaman,[1] Kehkashan Arouj,[2] Shahid Irfan[3]

Abstract

The frontal lobe is responsible for high-order functioning, such as memory, attention, decision-making, and personality. Lesions in the frontal lobe may lead to different physical and psychological problems. The current study was conducted to examine the emotional, cognitive, and behavioural states and coping strategies of a patient with recurrent bilateral frontal lobe lesion. It also attempted to determine post-traumatic stress disorder (PTSD) symptoms in a patient. This study described the case of an adult with recurrent bilateral frontal lobe tumour. It covered the clinical presentation, administration of Urdu translation of the Schema Mode Inventory (SMI) and Clinician-Administered PTSD Scale for DSM-5 (CAPS-5), and analysis of the case. The results of the study showed that the recurrent bilateral frontal lobe brain tumour patient engaged in child mode and had a dysfunctional coping style and a maladaptive punitive parent mode. Furthermore, the patient also had moderate PTSD symptoms.

Keywords: Maladaptive Schema Modes, PTSD, Frontal Lobe, Lesion.

DOI: https://doi.org/10.47391/JPMA.1071

Introduction

The frontal lobe is one of the most developed and largest lobes of the brain and lies next to the central sulcus. Lesions in the brain affect an individual's physical and psychological health, particularly when they are in the frontal lobe. The frontal lobe controls executive functioning like memory, schema, attention, decision-making, speech production, social behaviour, and personality.[1] It is one of the most advanced parts of the brain and is divided into three areas: premotor, pre-central, and prefrontal.[2] Damage and injury to the frontal lobe is due to the location of the frontal skull and the size of the tumour. Frontal lobe is the region that is most frequently damaged and sensitive to ethanol effects compared to other parts of the brain.[3] Schema is related to our cognition and thought processes and is imposed on reality or experiences, which helps to guide and explain an individual's behaviour and responses. Schema modes are a core concept of schema focus therapy and consist of a collection of thoughts, behaviours, and responses.[4] Schema modes are self-perpetuating and extremely difficult for an individual to change. Individuals adjust their schema modes according to their situations. They are grouped into four main categories including: healthy modes, dysfunctional child modes, dysfunctional parental modes, and dysfunctional coping modes which are further sub-grouped into 14 categories. According to a study, the frontal lobe is involved in different functions, such as memory, decision-making, language, and impulse control.[5] The current study reported an individual with a brain tumour in the frontal lobe. The purpose of the case report was to investigate the schema modes and coping styles along with PTSD symptoms in patients with frontal lobe lesion.

Case Report

A 42-year-old man was brought to the neurosurgery

Table-1: SMI of 42-year-old male patient with recurrent bilateral frontal lobe lesion (meningioma).

SMI	Case Report	Healthy means	Discrepancy
VC	3.5*	1.47	2.03
AC	4.3*	1.81	2.47
EC	2.7*	1.20	1.5
IC	3.1*	2.15	0.95
UC	3.3*	2.27	1.03
HC	4.5*	4.52	0.03
CS	2.1(ns)	2.51	-0.41
DPT	2.9*	1.59	1.31
DSS	2.2*	1.93	0.27
BA	4.6*	1.72	2.88
SA	2.5(ns)	2.31	1.09
PP	3.8*	1.47	2.33
DP	2.7(ns)	3.06	-0.36
HA	4.9*	4.60	0.3

*Shows significant, ns= not significant Note: VC=Vulnerable Child, AC=Angry Child, EC=Enraged Child, UC= Undisciplined Child, IC=Impulsive Child, HC=Happy Child, DP=Detached Protector, CS=Complaint Surrenders=Self Aggrandizer, DSS=Detached Self-Soother, BA=Bully and Attack, DP=Demanding Parent, PP=Punitive Parent, HA=Healthy Adult.

[1,2]Department of Psychology, International Islamic University, Islamabad, [3]Department of Psychology, Foundation University, Islamabad, Pakistan.
Correspondence: Sabir Zaman. Email: sabir.zaman@iiu.edu.pk

Table-2: Summary sheet of PTSD symptoms of the patient with recurrent Bilateral Frontal lobe lesion on CAPS-5.

Criterion "B" Intrusive Symptoms	**Past month**	
Symptoms (1 for diagnose	Sev	Sx (Sev ≥ 2) 0= No, 1=Yes
1) Intrusive memories B1	2	1
2) Distressing dreams B2	1	0
3) Dissociative reaction B3	0	0
4) Psychological distressB4	1	0
5) Physical reactions B5	2	1
B total	**6**	**# B Sx= 2**
Criterion "C" Intrusive Symptoms	**Past month**	
Symptoms (1 for diagnose)	Sev	Sx (Sev ≥ 2) 0= No, 1=Yes
6) Avoidance feeling and memories C1	3	1
7) Avoidance of external reminder C2	1	0
C total	**4**	**# C Sx= 1**
Criterion "D" Intrusive Symptoms	**Past month**	
Symptoms (2 for diagnose	Sev	Sx (Sev ≥ 2) 0= No, 1=Yes
8) Unable to recall important aspect D1	3	1
9) Exaggeration of negative belief D2	2	1
10) Distortion of cognition leading blame D 3	1	0
11) Negative emotional state D4	2	1
12) Diminished Interest D5	0	0
13) Detachment from others D6	2	1
14) Lack of positive emotion D7	1	0
D total	**11**	**# D Sx= 4**
Criterion "E" Intrusive Symptoms	**Past month**	
Symptoms (2 for diagnose	Sev	Sx (Sev ≥ 2) 0= No, 1=Yes
15) Angry and irritable behaviour E1	3	1
16) Self-destructive behaviour E2	2	1
17) Hypervigilance E3	1	0
18) Exaggerated startle Responses E4	0	0
19) Concentration problems E5	3	1
20) Sleep disturbance E6	3	1
E total	**12**	**# E Sx= 4**
PTSD Totals	**Past month**	
Total	Total Sev	Total # Sx
Sum of Sub Totals (B+C+D+E)	**6+4+11+12=33**	**2+1+4+4=11**
F duration of disturbance	Current	
22) Duration of disturbance ? 01 month	1=Yes	
Impairment or distress (1 for diagnose)	**Past month**	
Criterion	Sev	Cx (Sev ≥ 2) 0= No,1=Yes
23) Subjective distress	01	0
24) Social impairment	02	1
25) Occupational functional impairment	02	1
G total	**05**	**# G Cx= 2**
Global rating		
26) Global Validity	Good	
27) Global Severity	Moderate	
28) Global Improvement	No	
Dissociative symptoms (1 for Sub-types)	**Past month**	
Symptoms	Sev	Cx (Sev ≥ 2) 0= No, 1=Yes
29) Depersonalisation		0
30) Derealisation		0
Dissociative sub total		# Diss Sx= 0
PTSD Diagnosis	**Past month**	
Present all criteria (B-G)	1=Yes	
With dissociative symptoms	0= No	
21(Delay onset ≥ months	0= No	

Note: Sev=Sevrity, Sx=Symptoms numbers of time, Diss=Dissociative, CAPS-5 severity score 0-10 few symptoms,11-22 mild PTSD, 23-34 moderate PTSD, 35-46 severe PTSD, above 46 Extreme PTSD.

department at the Pakistan Institute of Medical Sciences, Islamabad, in April 2019 with complaints of severe headache, dizziness, vomiting, irrational behaviour and talk, body tremors, insomnia, lack of appetite, and consistent sadness, accompanied by fits and loss of consciousness for the past month. He had no family history of psychiatric illness, no major physical trauma, and no history of any medical conditions or alcohol or substance abuse. The patient's history revealed that he had had a frontal lobe lesion in the right hemisphere, which was operated upon successfully in March 2016, after which the patient was referred to another hospital for chemotherapy. However, he did not adhere to the procedure for chemotherapy. After three sessions of chemotherapy, he discontinued the treatment. After three years, he returned to the hospital with the above complaints. He had lost all his previous treatment documents, and was re-admitted to the hospital's neurosurgery ward. Physical and neurological examinations showed some abnormalities, and the physician advised all related investigations. Biochemistry, and haematology results were within the normal range. An MRI revealed a well-defined extradural axial mass dense lesion in the right hemisphere that also involved part of the left frontal lobe. The thick mass in both the right and left frontal lobes was determined to be most likely a meningioma. The patients' caregivers were counselled about the treatment, prognoses, and future complications. Surgical excision was essential, and radiation therapy was advised after surgery. Routine follow-up (once a month for one year) was also advised. History was taken a week before the surgery. The intention of the current study was to examine the PTSD symptoms, emotional, and cognitive conditions, and coping strategies of a patient with a history of a recurrent bilateral frontal lobe lesion. A psychometric Schema Mode Inventory (SMI) was administered to assess the cognitive, emotional, and behavioural, and coping style, whereas Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) was used to measure the PTSD symptoms of the patient.[6,7] These scales were used for clinical and educational purposes and had good internal consistency Cronbach's range 0.76-0.96, and mean score of 0.86. Furthermore, CAPS-5 is a semi structured interview, which measures the essential features of PTSD, and five point symptom severity scale labelled from "0-4".[8] Before starting the test, the patient was instructed to read all the items carefully and then respond to each question. The patient was also instructed not to leave any items blank. Before giving the questions, the patient was informed and ensured that his data would be kept confidential and used only for academic purposes. After a proper informed consent was secured from the patient, a short Urdu version of the SMI was administered. Furthermore, a written consent was also secured from the patient for publishing the paper; permission was also taken from Institutional Review Board (IRB). The results of the current study showed that a patient with a bilateral frontal lobe lesion engaged more in child modes, maladaptive coping style, and maladaptive punitive parent as shown in Table-1.

The characteristics of child modes include, intense anger, harm to people and objects, like an uncontrollable child. In addition, child modes may also have characteristics such as strong feelings of aggression and an inability to show responsibility, lack of autonomous development, and feelings of helplessness. Furthermore, a patient with a history of bilateral frontal lobe lesions may also engage in dysfunctional coping styles as shown in Table-1. These maladaptive coping styles consist of avoidance, surrender, and overcompensation. The features of maladaptive coping strategies include being emotionally and psychologically withdrawn from pain. Patients with bilateral frontal lobe lesions engage in maladaptive coping style like emotional shut-off and behaviour that include gambling, risky sports events, immoral sexual activities, and drug abuse. Another dysfunctional coping style is overcompensation, which includes bullying and attacking and self-aggrandising. The person with frontal lobe lesion engages in bullying and attack activities, such as openly damaging and hurting people socially, verbally, and sexually, and engaging in antisocial activities. In addition, results in Table-1 revealed that a patient with bilateral frontal lobe lesion may also engage in maladaptive punitive parent modes including demanding and punitive parents. These are the negative learned thoughts about oneself and others. These thoughts may be based on fear (demanding) or it can be nasty thoughts (punitive). Moreover, healthy adult mode performs perfect functioning, such as problem solving, appropriate working, social responsibility, maintaining relationship, taking part in sporting and social activities. Results in Table-1 showed that bilateral frontal lobe brain lesion patients lack such qualities. The current study showed that a patient of recurrent bilateral frontal lobe lesion with no current history of trauma had moderate PTSD symptoms as described in Table-2.

The sum of total score on CAPS-5 is 33 which is moderate PTSD symptoms.

Discussion

Several studies have shown the association between brain lesions and psychological symptoms. One study

revealed that tumours on the dorsolateral region of the brain cause difficulty in planning, and organisation. Lesions on the orbito-frontal side lead to disinhibition, while tumours on the medial frontal area might be responsible for apathy and abulia. The schema and belief changes and depressive symptoms occur when the tumour exists in the frontal lobe particularly in the diencephalic region.[9,10] A study provided support to the result of the present study, that PTSD symptoms were more severe in men without criterion A1, than A1 events, whereas the severity of PTSD symptoms were the same in women after non-A1 and A2 events.[11] Treatment and diagnosis of patients with brain tumours specifically on the frontal lobe are challenging tasks for the physicians and neuropsychologists. Clinical and physical examinations and neuro imaging such as MRI and CT scan play an important role in the diagnosis and psychological assessment along with intervention can also boost the recovery of such patients.

Conclusion

The present study showed that patient with a history of recurrent bilateral frontal lobe lesions engaged in maladaptive schema modes, and maladaptive coping strategies. He lacked healthy schema modes qualities, such as maintaining relationship, sporting social responsibilities, and social activities. These findings may help psychologists, psychiatrists, neuro-physicians, care givers, and close family members in the treatment, and care of the individual. The study also provided literature support in the field of neuropsychology and psychiatry and help psychologists in schema focus.

Disclaimer: None to declare.

Conflict of Interest: None to declare.

Funding Disclosure: None to declare.

References

1. Schneider B, Koenigs M. Human lesion studies of ventromedial prefrontal cortex. Neuropsychologia. 2017;107:84-93.
2. Perecman E. The frontal lobes revisited: Psychology Press; 2019.
3. Sachs B, Dodson K. Addictive Substances and Neurological Disease. Elsevier; 2017.
4. Young, Klosko JS, Weishaar ME. Schema therapy: A practitioner's guide: Guilford Press; 2003.
5. Firat RB. Opening the "Black Box": Functions of the Frontal Lobes and Their Implications for Sociology. Front Sociology. 2019;4:3.
6. Young J, Arntz A, Atkinson T, Lobbestael J, Weishaar M, Van Vreeswijk M, et al. The schema mode inventory. New York: Schema Therapy Institute; 2007.
7. Riaz MN, Khalily T. Translation, adaptation, and cross language validation of short Schema Mode Inventory (SMI). Pak J Psychological Res. 2013;28.
8. Weathers FW, Bovin MJ, Lee DJ, Sloan DM, Schnurr PP, Kaloupek DG, et al. The Clinician-Administered PTSD Scale for DSM–5 (CAPS-5): Development and initial psychometric evaluation in military veterans. Psychological Asses. 2018;30:383.
9. Pirau L, Lui F. Frontal Lobe Syndrome. StatPearls Publishing; 2018.
10. Pressman P, Rosen HJ. Disorders of frontal lobe function. Neurobiology of Brain Disorders. Elsevier; 2015, pp 542-57.
11. van den Berg LJ, Tollenaar MS, Spinhoven P, Penninx BW, Elzinga BM. A new perspective on PTSD symptoms after traumatic vs stressful life events and the role of gender. Eur J Psychotraumatol. 2017;8:1380470.

CASE REPORT

Potential effect of sodium thiosulfate in calciphylaxis: remission of intractable pain

Canlin Yang,[1] Yuqiu Liu,[2] Haifeng Ni,[3] Xiaomin Li,[4] Hong Liu,[5] Xiaoliang Zhang[6]

Abstract

Calciphylaxis, a rare disease mainly seen in patients with chronic kidney disease, is characterised by ischaemic skin damages and excruciating pain. Calciphylaxis has poor prognosis which often results in amputation and high mortality. Although guidelines for the management of calciphylaxis are not available, sodium thiosulfate has shown efficacy in many clinical reports. We report the case of a 64-year-old advanced calciphylaxis male patient who had two amputations due to intolerable pain manifested as deteriorating ulcer. After he was treated with intravenous sodium thiosulfate (STS), his pain was significantly relieved with a healing trend of the big wound. One more amputation for the remission of intractable pain was avoided. The treatment experience indicates that sodium thiosulfate is of great value in quick pain relief and reducing suffering of calciphylaxis patients.

Keywords: Calciphylaxis, Calcific uremic arteriolopathy, Painful skin ulcer, Maintenance haemodialysis, Sodium thiosulfate.

DOI: https://doi.org/10.47391/JPMA.1244

Introduction

Calciphylaxis, also known as calcific uraemic arteriolopathy, is a rare but fatal vascular calcific disease characterised by occlusion of subcutaneous adipose tissue and microvascular in the dermis, which causes severe pain and ischaemic skin damage.[1] Calciphylaxis is typically reported in end-stage renal disease patients with poor prognosis, which has amputation rate of 66.7% and the patient feels incurable pain even with basic treatments such as antisepsis, analgesia and dressing change.[2] The major risk factors are chronic kidney disease, hypercalcaemia, hyperphosphataemia, high serum intact parathyroid hormone (iPTH) level, long-term use of Warfarin and calcium supplement.[3,4] Treatment options including medication, wound care and hyperbaric oxygen are still at an exploratory stage. Sodium thiosulfate (STS) was the first to be reported effective in calciphylaxis[5] and is still most frequently used. STS, a calcium chelator, can inhibit adipocyte-induced vascular calcification with antioxidative and vasodilatory properties.[6] Comprehensive treatment based on intravenous STS is recommended and is routinely used off-label in calciphylaxis.

We report the case of an advanced calciphylaxis patient who had recurrent acral skin necrosis and a third amputation seemed to be inescapable. However, STS administration significantly relieved the sharp pain and delayed deterioration of skin ulcer so that the third amputation was avoided.

Case Report

A 64-year-old Chinese, non-diabetic male patient with a 23-year history of haemodialysis presented with calciphylaxis. He had been diagnosed with secondary hyperparathyroidism (SHPT) with high iPTH level (>600 ng/L) and had been on oral calcium supplement for about five years. Since 2015, there were recurrent and progressively worsening skin ulcers along with severe pain in the first and third fingers of his left hand. His condition was such that he could not sleep, which had seriously affected his quality of life. Hence, his two fingers had been amputated, while three toes of the right foot met the same fate one year later. Although he accepted amputation of the right lower limb again, there was also an evolving ulcer on his left heel (Figure-1a) which caused sharp pain. At the same time, the previous operated site of amputation showed poor healing. The pain was difficult to control even with two or more painkillers. He had already received treatment for ulcers and wounds in other hospitals with wound care, debridement and repetitive intravenous antibiotics. He was admitted to our hospital (Department of Nephrology, Zhong Da Hospital) on March 12, 2018 for the dusky discoloured foot ulcer accompanied by growing pain and had agreed to undergo amputation.

Physical examination showed a dark purple necrotic lesion on his left heel without exudates (Figure-1a). Tenderness on the left dorsum pedis was significant, instep skin temperature was lower than proximal part and

[1-3,5,6]Department of Nephrology, [4]Department of Clinical Pharmacy and Pharmacology, Zhong Da Hospital, Affiliated to Southeast University School of Medicine, No.87, Dingjiaqiao, Gulou District, Nanjing, Jiangsu Province, China.
Correspondence: Xiaoliang Zhang. Email: tonyxlz@163.com

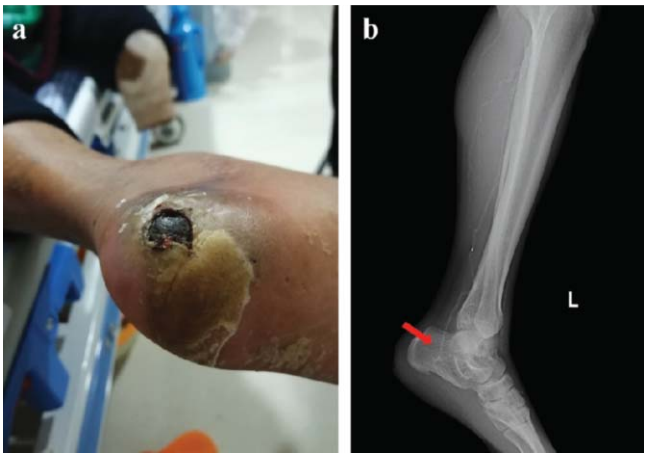

Figure-1: (a) Ulcer on the left heel with black- sunken necrosis surrounded with dark purple colour skin (Photographed in March 2018); (b) X-ray of the left lower extremity. The red arrows indicate the calcified vascular which suspected of supplying blood at the necrotic site.

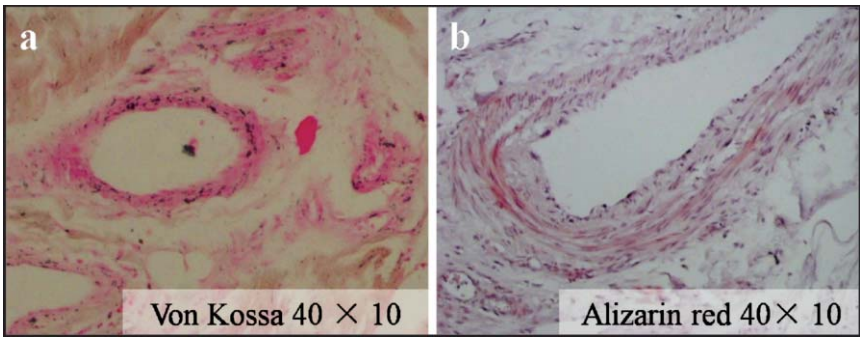

Figure-2: Skin Pathological Examination: (a) Von Kossa: positive with black stained calcium deposition in soft tissue, arterioles and middle artery wall of dermal tissue; (b) Alizarin red: positive with orange stained calcium in the vessel wall.

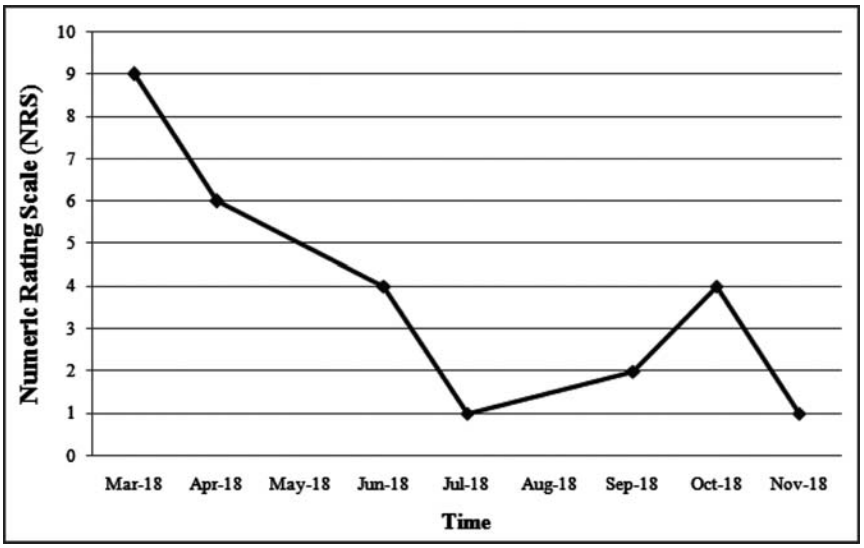

Figure-3: Highest pain scores measured in numeric pain rating scale during admission period.

left dorsalis pedis arteries pulsation was weak. The left thumb, middle finger and right extremity were missing. The right thigh amputated wound was poorly healed with obvious redness and swelling. Laboratory evaluation revealed an elevated iPTH of 474.3pg/mL (12~88 pg/mL), serum phosphorus of 1.83mmol/L (0.85~1.51mmol/L) and a normal serum calcium of 2.23mmol/L (2.11~2.52mmol/L). His highest pain score of numeric pain rating scale (NRS) was 9. Radiography (Figure-1b) of left lower extremities showed successive vascular calcification in tibial and dorsalis pedis arteries.

A skin biopsy was performed, which confirmed calcium deposition in the vessel walls with von Kossa and Alizarin red staining (Figure-2). Combined with the risk factors, clinical manifestations, imaging performance and pathological examination, the patient was diagnosed with calciphylaxis. Besides low calcium dialysis, oral calcitriol and wound care, he was given daily intravenous infusion of STS (starting dose: 5g and maintenance dose: 10g i.v. each day) for six months. In the initial week of medication, there was an increase in the dusky area on the left heel but the patient felt the pain was relieved as he did not wake up with pain at night. Within three months, ulcer progression had halted and the amputated wounds in his right lower extremity began to slowly heal. Six months later, serum calcium and phosphorus level were 2.35mmol/L and 1.21mmol/L, respectively. His NRS pain score gradually decreased within the initial five months and fluctuated between 1~4 since the onset of medication (Figure-3). On account of the above treatment, there was no indication for amputation, and the patient received STS treatment periodically.

Discussion

Calciphylaxis is gradually gaining clinicians' attention with more literature published while the mechanism remains unclear. It's reported that the one-year mortality is 45~80%[3,4] and calciphylaxis in chronic haemodialysis patients has nearly three times higher mortality.[7] Approximately 50% of survivors are bedridden or wheelchair-bound, and more than 70% require hospitalisation because of severe ulcers.[8] Sustained pain

and insomnia could further affect their quality of life.[9] It's important to relive pain and reduce disability rate in calciphylaxis patients.

Our patient was diagnosed with severe calciphylaxis who was characterised by recurrent peripheral gangrene of fingers, toes and heel. His risk factors included chronic haemodialysis, SHPT, high serum levels of phosphate and long-term oral calcium treatment. The patient had a history of two amputations due to unbearable pain. However, the acral-skin necrosis deteriorated continuously and seriously affected his life.

STS (Na2S2O3) has certain efficacy on calciphylaxis owing to its calcium chelating property.[6] The calcium deposited in vascular wall can be dissolved by sodium ions, which synthesise highly soluble calcium thiosulfate. The latter can be further removed by haemodialysis. STS also functions as an anti-oxidation and vasodilatation agent.[6,10]

Nigwekar SU. et al[11] proposed a STS medication regimen in 2015, for an average 70kg person on thrice haemodialysis therapy, they recommended administering 25g STS (Na2S2O3·5H2O) intravenously in 100mL of normal saline during the last half-hour of each haemodialysis session. The same regimen was applied to our patients and obvious adverse reactions including nausea and vomiting were reported. This may stem from racial differences. After repeated attempts, a dosage regimen for Chinese calciphylaxis patients was developed which worked well with fewer side effects. The starting dose of STS (Na2S2O3·5H2O) is 5g with an increase of 1g daily and maintenance dose is 10g. STS is delivered by intravenous drip in 250mL normal saline solution every day. It shows that reduction of phosphorus and NRS are significantly correlated with STS treatment courses. Although incidence of adverse events is up to 40% (nausea/vomiting 10%, hypotension 10%, infection 20%), no one's treatment was interrupted by mild discomfort.

Conclusion

The case of an advanced calciphylaxis with typical acral-gangrene and history of amputations is presented. The patient responded well to treatment with STS. It's suggested that STS also has a certain therapeutic effect among calciphylaxis patients in advanced stage, which mainly reflects in relieving pain and delaying the progression of skin ulcers. We are applying STS medication regimen in more calciphylaxis patients and hope to see more authoritative treatment guidelines.

Informed Consent Form: The patient signed an informed consent and authorised the publication of data and photographs of the case.

Disclaimer: None to declare.

Conflict of Interest: None to declare.

Funding Disclosure: This work received support from the National Natural Science Foundation of China (No. 81570612 and No.81870497) and Clinical Medical Science Technology Special Project of Jiangsu Province (BL2014080).

References

1. Nigwekar SU, Thadhani R, Brandenburg VM. Calciphylaxis. N Engl J Med 2018; 378: 1704-14.
2. Torregrosa JV, Sanchez-Escuredo A, Barros X, Blasco M, Campistol JM. Clinical management of calcific uremic arteriolopathy before and after therapeutic inclusion of bisphosphonates. Clin Nephrol 2015; 83: 231-4.
3. McCarthy JT, El-Azhary RA, Patzelt MT, Weaver AL, Albright RC, Bridges AD, et al. Survival, Risk Factors, and Effect of Treatment in 101 Patients With Calciphylaxis. Mayo Clin Proc 2016; 91: 1384-94.
4. Nigwekar SU, Zhao S, Wenger J, Hymes JL, Maddux FW, Thadhani RI, et al. A Nationally Representative Study of Calcific Uremic Arteriolopathy Risk Factors. J Am Soc Nephrol 2016; 27: 3421-9.
5. Cicone JS, Petronis JB, Embert CD, Spector DA. Successful treatment of calciphylaxis with intravenous sodium thiosulfate. Am J Kidney Dis 2004; 43: 1104–8.
6. Chen NX, O'Neill K, Akl NK, Moe SM. Adipocyte induced arterial calcification is prevented with sodium thiosulfate. Biochem Biophys Res Commun 2014; 449: 151-6.
7. Nigwekar SU, Solid CA, Ankers E, Malhotra R, Eggert W, Turchin A, Thadhani RI, Herzog CA. Quantifying a rare disease in administrative data: the example of calciphylaxis. J Gen Intern Med 2014; 29 Suppl 3: S724-31.
8. Weenig RH, Sewell LD, Davis MD, McCarthy JT, Pittelkow MR. Calciphylaxis: natural history, risk factor analysis, and outcome. J Am Acad Dermatol 2007; 56: 569-79.
9. Riemer CA, El-Azhary RA, Wu KL, Strand JJ, Lehman JS. Underreported use of palliative care and patient-reported outcome measures to address reduced quality of life in patients with calciphylaxis: a systematic review. Br J Dermatol 2017; 177: 1510-8.
10. Generali JA, Cada DJ. Sodium Thiosulfate: Calciphylaxis. Hosp Pharm 2015; 50: 975-7.
11. Nigwekar SU, Kroshinsky D, Nazarian RM, Goverman J, Malhotra R, Jackson VA, Kamdar MM, Steele DJ, Thadhani RI. Calciphylaxis: risk factors, diagnosis, and treatment. Am J Kidney Dis 2015; 66: 133-46.