



## Transient Diabetes Insipidus Following Thermal Burn; A Case Report and Literature Review

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**Received:** March 14, 2017  
**Revised:** July 19, 2017  
**Accepted:** August 1, 2017

### ▶ ABSTRACT

Diabetes insipidus is a disease characterised by increased urine production and thirst. Neurogenic diabetes insipidus following head trauma, autoimmune disease and infection is quite common but diabetes insipidus following thermal burn injury is a rare complication. We should know about this complication as its management need a comprehensive approach for satisfactory outcome. Thermal burn can cause different complications in early post burn period like electrolyte imbalance, dehydration, acute renal failure, but diabetes insipidus is a very rare and unusual complication that may come across in thermal burn. We should be aware about this condition to prevent and treat mortality and morbidity in burn patients. We have reported a case of transient diabetes insipidus in a patient of thermal burn in early post burn period. Patient was treated accordingly, leading to complete recovery.

**Keywords:** Diabetes insipidus; Thermal burn; Polyuria.

Please cite this paper as:

Dash S, Ghosh S. Transient Diabetes Insipidus Following Thermal Burn; A Case Report and Literature Review. *Bull Emerg Trauma*. 2017;5(4):311-313. doi: 10.18869/acadpub.beat.5.4.454.

### Introduction

Thermal burn as a cause of DI is very rare and only few cases have been reported so far in literature. The pathophysiology of these cases suspected to be hypoxic brain insults, which leads to diabetes insipidus (DI). Management of these cases needs proper history assessment, supportive measure and close monitoring of renal function and electrolytes, urine and plasma osmolality, specific gravity. Most these cases have spontaneous remissions [1, 2]. DI a condition characterized by

excretion of abnormally large amount 24 hr urine volume (>50ml/kg/body weight and osmolality is <300 mosml/L). This increases the plasma osmolality (>300) slightly leads to increase in thirst. Overt signs of dehydration are absent unless the fluid intake is impaired [3]. Deficient secretion of anti-diuretic hormone (ADH) is the cause of DI. It can be of central or renal origin. Renal DI causes are due to drugs, metabolic vascular, genetic. While central DI is due to head trauma, neoplasm, central nervous system (CNS) infection, and genetic [4].

## Case Presentation

A 35-years-old woman who sustained homicidal (approximately 40% body surface area) mixed thermal burn injury with facial burn and inhalational burn injuries with suspected inhalational injuries, following domestic quarrel. There is no history of taking any anti-psychotic medications or other drugs. This patient was treated at first in the local hospital for first few days, referred to us after 7 days to our emergency. At the time of admission, she was conscious and her vitals were stable. Her weight was 41 kg. She suffered major burns over her trunk, arms, thighs and back. She was resuscitated with ringer lactate and dextrose saline. Other supportive measures like analgesia, tetanus prophylaxis and pantoprazole was given. She was put on high protein diet. Her weight measurement was done regularly. Complete haematocrit, electrolytes, urea and creatinine sent for. Wound swab culture sent for sensitivity. Wound care with 1% silver sulphadiazine cream and closed dressing done with colloidal nano silver dressing. Nebulization and chest physiotherapy was done. Complete haemogram such as haemoglobin, total white cell count and albumin, were shown in Table 1. Fluid intake output monitored everyday along with electrolytes and renal function test are done in every 2-3 days, deficit electrolytes are replaced accordingly (Table 2).

From the mid of second week she started passing

large amount of urine. The amount of urine has increased subsequently. At first we tried to restrict the fluid intake both oral and IV, to concentrate the urine, but it failed. A MRI of brain was done to rule out any intracranial pathology found to be normal study. Urine specific gravity was done found to be 1.003. Urine osmolality was 190 mosm/l, plasma osmolality 398 mosm/l (Table 3). We have started carbamazepine 200 bd due to easy availability and based on literature. It showed improvement after 10 days of therapy. She was treated with amoxicillin clavulanic acid at first. The patient wound culture found to be pseudomonas aeruginosa, sensitive to amikacin. About end of third week the urine began to concentrate and the daily urine output began to contract, reaching about 2000 ml at the time of discharge. Her sense of thirst was normal to slightly increased throughout her illness. Examination urine for glucose and protein was negative. She has undergone multiple dressings. Most of the burned areas show epithelisation. By the end of first month she gained weight (45 kg) and wounds are healthy. Split skin grafting was done for non-healing wounds. We couldn't measure the serum vasopressin level due to unavailability of these test in our setup.

## Discussion

Deficient secretion of ADH leads to DI, which can be primary or secondary. Primary is also called as

**Table 1.** Values of haemoglobin, total leukocyte count and albumin on different days.

Day	Hb (g/dL)	WBC (cells/mm <sup>3</sup> )	Albumin (g/dl)	Remarks
11	7.1	9800	2.8	Blood transfused
20	8.7	15300	2.6	Antibiotics changed
24				Blood transfused
27	9.8	10100		
29	9.2			
31	10.1	9700	2.9	Pt improved

**Table 2.** The Urine pH, protein and sugar in days 14 and 24 of the admission.

Day	Urine PH	Urine Glucose	Urine Protein
14	7.4	Nil	Nil
24	7.5	Nil	Nil

**Table 3.** Urine amount, electrolytes and renal function tests on different days

Day of admission	Urine output in ml	Sodium mEq/L	Potassium mEq/L	Urea mg/dl	Creatinine mg/dl	Remark
11	4700	133	3.8	28	1	
14	6540	138	3.3	30	0.8	Fluid restriction started,
15	8560					
18	9820	142	4.7	32	0.7	Carbamazepine started
20	8120	140	3.9	31	0.8	
22	9830					
24	7690					Urine amount started decreasing
27	5510					
29	3400	137	4	27	0.7	
31	2400					

central DI. DI following burn injury is very rare, the pathophysiology of this is largely unknown but Chang in his article stated that it may be due to carbon monoxide poisoning causing diffuse cerebral damage. Carboxy haemoglobin causes damage in supraoptic nucleus leading to decrease in ADH secretion. There is other combustion product like cyanide have some role in the process [5]. When there is increase urinary volume, frequency, possibility of diabetes insipidus should be suspected after evaluating for urinary glucose. If volume exceeds >50 ml/kg/body weight and urine osmolality <300 mosm/L. In this case the renal function is normal throughout the course, only little change in the electrolytes [3, 4].

In this case we measured the osmolality and specific gravity of urine. We could not measure the blood ADH and carboxyhaemoglobin level due to non-availability of these test in our institute. MRI of brain was done to rule out intracranial pathology. We used carbamazepine for treatment, the patient improved after one week similarly used by Gende in his case [6, 7]. We have also planned for using desmopressin but since patient showed improvement we did not use it. Thermal burn has different complications among these DI is one of the rarest one. These patients need multimodal management and through evaluation.

**Conflicts of Interest:** None declared.

## References

1. Urquhart CK, Craft PD, Nehlawi MM. Transient diabetes insipidus following electrical burns in two patients. *South Med J.* 1994;**87**(3):412-3.
2. Ozdemir A, Seymen P, Yurekli OA, Caymaz M, Barut Y, Eres M. Transient hypothalamic hypothyroidism and diabetes insipidus after electrical injury. *South Med J.* 2002;**95**(4):467-8.
3. Capatina C, Paluzzi A, Mitchell R, Karavitaki N. Diabetes Insipidus after Traumatic Brain Injury. *J Clin Med.* 2015;**4**(7):1448-62.
4. Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J, editors. *Harrison's Principles of Internal Medicine* 19<sup>th</sup> ed. McGraw-Hill Medical; 2014.
5. Chang MY, Lin JL. Central diabetes insipidus following carbon monoxide poisoning. *Am J Nephrol.* 2001;**21**(2):145-9.
6. Gende G, James S, Garo M. Case report of a thermal burns patient with diabetes insipidus. *P N G Med J.* 2011;**54**(1-2):56-8.
7. Wales JK. Treatment of diabetes insipidus with carbamazepine. *Lancet.* 1975;**2**(7942):948-51.