Diabetic hyperosmolarity: a consequence of loss of autonomy

T Constans

SUMMARY

Diabetic hyperosmolarity is a serious acute metabolic disorder mainly occurring in the frail elderly subject presenting age-related favoring factors (reduced sensation of thirst, altered endocrine regulation), disease-related favoring factors (cognitive impairment, poor nutritional status and/or loss of autonomy), and a triggering factor, generally infection. Diabetic hyperosmolarity can occur in a previously non-diabetic patient. Intense dehydration dominants the clinical picture. The prognosis depends largely on the underlying chronic disease.

Key-words: Diabetes mellitus · Elderly · Hyperosmolar non-ketotic coma · Co-morbidity . Frailty.

T Constans. Diabetic hyperosmolarity: a consequence of loss of autonomy.
Diabetes Metab, 2005;31:5S62-5S66

RÉSUMÉ

L'hyperosmolarité : une conséquence de la perte d'autonomie

L'hyperosmolarité diabétique (DH) est un désordre métabolique aigu sévère survenant principalement chez un patient âgé fragile du fait de facteurs favorisants liés à l'âge (réduction de la sensation de soif, altération des régulations endocriniennes), de facteurs favorisants pathologiques (détérioration des fonctions cognitives, dénutrition et/ou perte d'autonomie) et d'un facteur déclenchant, habituellement une infection. La DH peut survenir chez un patient préalablement non-diabétique. La déshydratation est intense et domine le tableau clinique. Le pronostic est largement conditionné par les maladies chroniques sous-jacentes.

Mots-clés : Diabète · Personne âgée · Hyperosmolarité · Comorbidité · Fragilité.

Faculté de Médecine de Tours, Université François-Rabelais,
F-37032 Tours Cedex 1, France

Address correspondence:
T Constans
Faculté de Médecine de Tours, Université François-Rabelais,
F-37032 Tours Cedex 1
t.constans@chu-tours.fr

Diabetic hyperosmolarity is the usual way to decompensate type 2 diabetes in aged patients. Despite its high mortality rate, no recent publications on this pathology are currently available, and its relatively low incidence does not permit to correctly evaluate the various therapeutic protocols proposed so far.

Definition

Davidson [1] proposed the association of severe hyperglycaemia (8 g/l) and osmolarity > 350 mOsm/l. The *Manual of Medical Therapeutics* [2] proposed glycaemia > 6 g/l and osmolarity > 320 mOsm/l, as reflecting sufficiently severe metabolic degradation to warrant hospitalization in an intensive care unit. If serum osmolarity cannot be measured directly, it can be calculated from the following equation:

Plasma osmolarity = 2(Na + K) + glycaemia (mmol/l) + BUN (mmol/l)

Na, K, glucose, and blood urea nitrogen (BUN) values at 145, 5, 10 and 9 mmol/l respectively, give a calculated osmolarity of 319 mOsm/l. An upper limit of 320 mOsm/l thus clearly establishes a state of diabetic hyperosmolarity accepted by all authors. However, a blood glucose value ≥ 16.5 mmol/l (≥ 3 g/l) [3, 4], ≥ 30.0 mmol/l (≥ 5.4 g/l) [5] or even ≥ 33.0 mmol/l (≥ 6.0 g/l) [6] is mandatory to affirm the diagnosis of diabetic hyperosmolarity. Thus, there is currently no definite consensus on the definition of diabetic hyperosmolarity.

Clinical diagnosis

The clinical manifestations of diabetic hyperosmolarity can be viewed as an intense dehydration with impaired consciousness. The clinical and biological signs have not varied since the early descriptions [7] *(Table I)*. Like ketotic acidosis, diabetic hyperosmolarity is not systematically associated with coma, although impaired consciousness is much more frequent due to the severity of the dehydration. The term of hyperosmolar coma is not always appropriate. The general signs are associated with signs specific to the cause of the diabetic dehydration, usually an infection.

Table I
Clinical presentation and laboratory tests in diabetic hyperosmolarity.

Clinical presentation	Laboratory tests
Dehydration	**Osmolarity > 320 mOsm/l**
Hypovolemia: low ABP, low VBP	Plasma glucose > 16,5 mmol/l
Weight loss	(or 30, or 33 mmol/l)
Altered consciousness: from	Plasma Na > 145 mmol/l
lethargy to coma	Blood urea nitrogen > 9 mmol/l
Delirium	*To compare to previous values*
Hyperthermia	- High plasma creatinine
Neurologic signs	- High hematocrit
Seizures	- High plasma proteins
	Parameters with normal values
	- Arterial blood gases: pH > 7.30
	- Plasma lactate level: < 2 mmol/l
	- Plasma beta-hydroxybutyrate: normal
	- Ketones bodies in urine: 0 or +

Epidemiology

Diabetic hyperosmolarity is almost always observed in an old or old-old patient, often with dementia. Few epidemiological studies are available. Wachtel reported an incidence of about 17.5 cases/100,000 inhabitants/year, an incidence which is close to that of diabetic ketotic acidosis observed in the same Rhodes Island population in 1986 [7]. A more recent French study [3] conducted in the admissions room of a short-stay geriatric unit reported an incidence of 48 patients with hyperosmolarity (> 320 mOsm/l) among 436 admissions during a six-month period (11%). Among these 48 patients, 21 presented with a blood glucose level above 7.0 mmol/l and eight above 16.0 mmol/l at admission, thus fulfilling the diagnosis criteria for diabetic hyperosmolarity (mean glycaemia 20.8 ± 3.0 mmol/l). None of these patients presented a blood glucose level above 30.0 mmol/l. The sex-ratio (M/F = 1/3) was the same as that observed in age-matched patients hospitalized for other reasons. The two main associated factors were loss of autonomy and infections. Loss of autonomy was mainly related to impaired cognition (relative risk = 2.4, 95% CI: 2.2-3.3, P < 0.001) [3]. It was thus not surprising that many patients hospitalized for diabetic hyperosmolarity are nursing home residents.

Any infection can lead to glucose imbalance, or severe hyperglycaemia, in a patient with a well-controlled diabetes. Diabetic hyperosmolarity can also occur in a patient without any history of diabetes, and, according to Wachtel [7] and Bourdel-Marchasson *et al.* [3], frequently (one-third of all cases for Wachtel). Infections are found in 30 to 62% of patients [3, 7], mainly involving the respiratory tract (30 to 50%), but also the urinary tract, the digestive tract, the skin, etc. The infection is almost always an acute condition, generally with a major inflammatory component.

Other factors favoring the onset of diabetic hyperosmolarity are related to the multiple co-morbid conditions observed in the elderly patients: hypertension, ischemic stroke, myocardial infarction, heart failure, surgery, gastrointestinal bleeding, and any factor able to provoke a significant stress. Among drugs, diuretics and corticosteroids were often accused of favoring diabetic hyperosmolarity. The mechanism is easy to understand for these drugs, but for antihypertensives and anticonvulsants the mechanism is more obscure.

Indeed, patient-related factors, including co-morbid conditions, and institutionalization, must not be overlooked: protein-calorie malnutrition due to insufficient intake, and thus insufficient fluid intake; dependence for food intake and