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Signs and symptoms of dehydration in the elderly

Odwodnienie u osób w wieku podeszłym – objawy przedmiotowe i podmiotowe

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Keywords

dehydration diagnosis, dehydration staging, hypovolaemia, elderly

Słowa kluczowe

rozpoznanie odwodnienia, nasilenie odwodnienia, hipowolemia, wiek podeszły

Summary

Evaluation of the hydration status is probably the most difficult task in clinical examination. The clinical and objective determinations of hydration status frequently differ. The intracellular water volume remains stable when the acute water deficit is less than 2% of body weight (BW), and in the supine position dehydration of less than 5% may by clinically undetectable. Hypovolaemia ensues only from the mild-to-lethal dehydration, and the signs and symptoms of each are frequently wildly mixed up by the doctors. Signs of dehydration in the elderly precede the nonspecific symptoms, with the exception of thirst, which reaches maximum at water deficit of 1%, and is often suppressed by ageing, medication or both. The early detection of dehydration in the elderly is vital to prevent hypovolaemia and health deterioration and should be made by the caregivers, family members or medical professionals. The signs and symptoms can also help in water deficit quantitative evaluation which may support taking decision as to the proper intervention. The author details the pathophysiological background and the dehydration staging, to serve this purpose.

Streszczenie

Ocena stanu nawodnienia jest prawdopodobnie najtrudniejszą częścią badania klinicznego. Wyniki obiektywnej oceny nawodnienia często się różnią od klinicznej. Objętość wody wewnątrzkomórkowej nie zmienia się, jeśli tylko jej ostry deficyt nie przekracza 2% ciężaru ciała, ale w pozycji leżącej odwodnienie rzędu 5% ciężaru ciała może być klinicznie niewykrywalne. Hipowolemia towarzyszy zwykle odwodnieniu co najmniej łagodnemu, ale objawy odwodnienia i hipowolemii są bardzo często kompletnie mylone przez lekarzy. Oznaki odwodnienia wyprzedzają jego niespecyficzne objawy, z wyjątkiem pragnienia, które osiąga maksimum przy ubytku wody powodującym utratę 1% ciężaru ciała, ale które może być znacznie zmniejszone z powodu starzenia się, przyjmowania leków lub obu. Wczesne rozpoznanie odwodnienia u osób w wieku podeszłym jest dla nich życiowo ważne, bo pomaga zapobiec hipowolemii i pogorszeniu stanu zdrowia. Powinno to być dokonane przez opiekunów, członków rodziny lub personel medyczny. Objawy podmiotowe i przedmiotowe odwodnienia mogą być pomocne przy określaniu deficytu wody i wspomagać decyzję co do właściwego postępowania. Autor podaje szczegółowe podstawy patofizjologiczne i sposoby oceny stopnia odwodnienia mogące służyć temu celowi.

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INTRODUCTION

Evaluation of the hydration status is probably the most difficult task in clinical examination. Clinical judgment of water content is based mainly on the results of skin, oral mucosa and jugular veins examination. This is why the clinical and objective determinations of hydration status frequently differ (1). The aged skin is thinner, stiffer, less tense, and flexible than the young one (2). The surface of such a skin appears dry and rough due to the reduced epidermal proliferation accompanied by slower desquamation of the stratum corneum (SC), which makes it thicker and deficient in water binding substances (3). This partly explains why the transepidermal water loss (TEWL) in individuals aged 65 years and above is lower than in younger adults – lowest for the breast skin, and highest for the axilla (4). Up to 60% of population aged 70+ may show some laboratory or clinical signs of dehydration (5).

INTRACELLULAR VOLUME STABILITY

The volume and mineral content of both the ICW and ECW is controlled both by the cells and by numer-

ous body regulatory mechanisms, which stabilize the TBW in a healthy person within 0.22% (± 165 mL) of body weight (BW) (6).

Each of the 10^{14} cells of the body precisely regulates its own volume by controlling water exchange with the surrounding extracellular fluid. The ICW remains stable when the acute water deficit is less than 2% of BW (1-2 L) (7).

The marked and rapid "pure" water loss from the extracellular compartment, exceeding 2% BW, causes intracellular dehydration, which results in folding and denaturation of intracellular proteins and eventually cellular death – by necrosis or apoptosis. Similar acute isotonic changes in the ECW initially do not affect it, at least in haemodialysed patients with chronic renal insufficiency (8). However, the acute isotonic decrease of plasma or blood volume may cause water shift into the cells and increase the ICW (9, 10).

EXTRACELLULAR VOLUME CHANGES

Almost all extracellular fluid is distributed between the blood plasma (16-17%) and the interstitium – the extracellular space located extravascularly (83-84%). The equilibrium between plasma and interstitium volumes can be affected by many factors, which result in volume shift, either direction.

The hypohydrated interstitium shrinks and becomes hyperoncotic, whereas the hyperhydrated one expands. The first adsorbs water from the cells, which takes time, the second makes cell breathing, nutrition and detoxication more difficult.

The excessive interstitial water may form visible oedema – usually when fluid retention exceeds 4% BW. The loose connective tissues, lungs, gastrointestinal mesentery and mucosa can accumulate even more water – e.g. in pulmonary oedema the interstitial fluid expands fivefold from the normal 500 ml (11).

Due to the resulting adaptive morphological changes in the intracellular space in chronic overhydration, the chronic expansion of up to 8% of the ECW (1.6% of body weight, or 1.2 L in the "average" man) cannot be clinically diagnosed (12).

TISSUE HYDRATION EVALUATION

It is convenient, and correct, to assume for practical purposes, that the daily changes in hydration are limited to the ECW and do not affect the ICW. Thus, the changes in hydration status (ΔHS) can be calculated from the difference between the measured ECW and the normal ECW expected. The Relative Change in Tissue Hydration ($R\Delta TH = A\Delta TH/ECW$) greater than -7% of the ECW (reduction) is a cut off for the moderate-to-lethal dehydration. The $R\Delta TH$ of +7% (expansion), can be classified as "fluid overload" (13). Because the ECW forms 25% of body weight, the aforementioned 7% of ECW makes 1.75% of BW. This is why the BW changes of less than 1.5% from the individual's optimal weight (only 1 kg in a person of 70 kg BW), should not be, from the clinical point of view, alarming to the medical and social staff.

OSMOTICALLY DRIVEN INTRACOMPARTMENTAL FLUID SHIFTS

Intrinsic mechanisms keep plasma osmolality within a narrow range of 275-295 mOsm/kg; sodium is the principal osmolality determinant, at 135 to 145 mEq/L. Regulation of the extracellular volume prevails over the regulation of its composition, as it can be clearly seen in SIADH – when slight hyperhydration, mainly intracellular, accompanies severe hypotonicity second to hyponatraemia (14).

HYDRATION AND BLOOD VOLUME

Mild to severe dehydration (lack of water), acute or chronic, is accompanied by hypovolaemia (lack of blood), and so the diagnostic criteria for each of them are usually and incorrectly mixed up and presented together. Blood in the venous part of the vascular system is of lesser importance to hydration regulation as compared to that inside big arteries ("effective blood volume"). Blood volume insufficient to fill the big arteries and perfuse the body organs is called hypovolaemia. It results either from severe hypohydration, from decreased left heart contractitlity (heart failure), or from relaxation of small arteries (e.g. septic shock).

DEHYDRATION IDENTIFICATION

There is no single sign or symptom that would be pathognomonic to dehydration. Thus, the diagnosis of dehydration has to relay mainly on signs actively sought for.

Loss of "pure" water causes hyperosmolar dehydration – it affects exclusively the ECW when less than 2% of body weight is lost, and both, ICW and ECW at more advanced water loss stages.

Loss of water and sodium e.g. in sweat, after diuretics, in hyperglycaemia, results in hypohydration and concomitant hypovolaemia.

Dehydration may be isotonic, hypertonic or hypotonic, depending on the proportion of water and sodium lost.

Depending on the pure water deficit the dehydration can be divided, into:

- imminent: 0.22-1% of body weight,
- mild: 1-2% of body weight,
- moderate: 2-5% of body weight,
- severe: 5-10% of body weight,
- extreme: > 10% of body weight,
- fatal: > 15% of body weight.

Mild to moderate dehydration could be treated at home or in the nursing home (15).

CLINICAL SYMPTOMS BY STAGE OF DEHYDRATION

Hypohydration initially leads to increased urine concentration, which normally results in the output of lower than normal amounts of dark-yellow urine. The volume, osmolality, specific gravity, and colour of the urine are all very sensitive indicators of the extent of the imminent water deficit (16) provided the renal function is normal and the water deficit does not exceed 1% of

body weight. The darkening of urine from straw-yellow to yellow, and less frequent visits to the toilet can be usually noticed as early as the water deficit reaches 0.5% of body weight (300-400 ml), and adults attain the maximum urine concentration when the body water deficit arrives at 1% (600-900 ml). In the elderly, the ability of the kidneys to excrete concentrated urine deteriorates, thus the pale urine colour can be deceiving in assuming euhydration, because the urine can remain yellow, even in mild and severe hypohydration, when the dark yellow urine would be expected.

Along with the increasing urine concentration decreases saliva production (below 0.1 ml/min) causing the very unpleasant feeling of dry mouth (xerostomia), which can be partly supressed by its long duration. However, the same effect is seen when the person respires through the mouth, or takes numerous medications (there are over 500 of them, mainly cholinolytics, sympaticomimetics, painkillers, diuretics). Thus the leading symptoms of the imminent dehydration (< 1% BW) are limited:

- darkening of urine,
- less frequent visits to the toilet and
- xerostomia.

These are seldom perturbing to the patient and frequently not reported, even during systematic anamnesis taking.

In extreme to lethal dehydration, (body weight loss > 10%) symptoms are inconclusive, because are masked by confusion and even lethargy.

The clinical meaning of dehydration symptoms is limited and very individual, some may appear at imminent-mild dehydration and some may not be present even at very severe dehydration. The same holds true for the clinical signs of dehydration.

Clinical signs of dehydration

The signs of mild to moderate dehydration precede the majority of its symptoms, and for this reason should be sought for actively. This is of special importance in the elderly, because the unspecific symptoms of imminent to mild dehydration can be absent or not noticed, ignored, misinterpreted, or even neglected by them. Thus, it is important that everyone who takes care for the patient pays attention to the signs of dehydration.

Clinical signs usually accompanying each stage of dehydration are collected in the table below (tab. 1).

The chronic moderate dehydration usually is accompanied by the signs of imminent hypovolaemia:

- blood pressure decreased only upon raising up (orthostatic hypotension, systolic pressure drop by 10-15%),
- pulse weak, thread, rapid (especially increasing at rest by 15 bpm after upright position),
- capillary refill time 2-4 s.

In severe to lethal dehydration the signs of hypovolaemia usually dominate the clinical picture.

LABORATORY FINDINGS IN DEHYDRATION

The lack of water proportionally grater than that of sodium, increases plasma sodium concentration and osmolality, above 145 mmol/L and 300 mOsm/kg H_2O, respectively. Should the moderate or severe dehydration result in hypovolaemia, the plasma protein and albumin and blood haemoglobin increase. The accompanying rise in plasma BUN, creatinine and uric acid concentrations are indicative of concomitant kidney hypoperfusion.

HYPOVOLAEMIA

It is important for clinical evaluation to detect if the hypohydration is accompanied by hypovolaemia. The

Table 1. Emergence of symptoms and signs indicative of dehydration by stage.

< 1%	1-3%	3-5%	5%-10%	> 10%
Darkening of urine, Less urine, Less frequent visits to the toilet, Xerostomia	Thirst, Tiredness, Headaches, Sore throat, Decreased attention, Reduced short-term memory, Irritability, Decreased appetite, Sleeplessness, Tingling or discomfort during urination	Thinning of the fingers, Skin dryness, Localised pruritus, Anorexia	Dizziness and tachycardia at adopting the upright position, Fainting, Fatigue, Severe headaches, Nausea, Constipation, Decreased sweating, Low or none urine output, Tingling or numbness in extremities, Muscle cramps, Extreme sleepiness, Apathy, Lethargy	Confusion
Urine yellow or dark yellow, Saliva thick	Dark-yellow to brown urine, Saliva thick and sticky, Dry oral and nasal mucosa, Cracked lips, Dry skin on hands and on forehead	Sudden loss of 2-5% body weight, Recent change in consciousness or in muscle strength, White or yellow tongue, Halitosis, Speech incoherence, Talkativeness, Complaining, Dry sunken eyes, Decreased skin turgor, Dry axilla, Tachypnoe (breathing rate > 28/min), Imminent hypovolaemia	See stage 2 hypovolaemia	See stage 2 hypovolaemia

loss of 1 L of total body water reduces the body weight of the average individual by 1.33% indicating mild dehydration accompanied by the decrease in blood volume, jointly plasma and erythrocytes, by 120 ml (2.4%). The same amount of isotonic fluid lost exclusively from the extracellular compartment results in 180 ml reduction of blood volume (3.6%) exclusively from plasma (6.5%), because the total volume of all red blood cells remains initially unchanged. These are far lesser changes when compared to the loss of 1 L of blood (20%), which initially does not affect the interstitium volume nor the extravascular ICW. The course of blood volume loss can be exacerbated by heart failure – i.e. the volume loss results in higher than normal class of hypovolemic shock. Based on the reaction to the acute blood loss one can characterise four stages of acute hypovolaemia, depending on the volume lost (tab. 2). Some authors reduce the number of haemorrhagic shock stages to three.

The causes of hypovolaemia are numerous:

- sodium and water loss (vomiting, diarrhea, excessive sweating, diuretics),
- haemorrhage (external or internal, from the ruptured aneurysm, varix, or injured vessel),
- third spacing (fluid shifts in sepsis or trauma),
- other (inadequate fluid treatment, darenal insufficiency, hypocalcaemia).

There are no pathognomic symptoms of hypovolaemia, and it is diagnosed based on symptoms and signs of cardiovascular adaptation to decreased effective blood volume, which are presented in the table below (tab. 3).

CLINICAL SYMPTOMS OF HYPOVOLAEMIA

Hypovolaemia is most easy diagnosed in the upright position – usually dizziness or lightheadedness, tachycardia or cardiac arrhythmia, appearing or worsening in standing up position.

More pronounced hypovolaemia results in tachycardia also in recumbent position, and getting into the vertical position may cause fall and loss of consciousness. The patient may also feel cold and report “chills”, cold feet, hands, nose tip and ears.

CLINICAL SIGNS OF HYPOVOLAEMIA

The most typical signs of hypovolaemia result from the adaptive contraction of vascular bed and blood centralisation to keep the proper blood inflow to the vitally most important organs – brain, heart and kidneys. These include cold skin, tachycardia, low urine volume, and dizziness presenting or increasing in standing position. In the supine position the blood pressure and the heart rate can remain normal despite the loss of up to 4% of body weight (7% of TWB). Should the hypovolaemia result from dehydration, the skin is dry, and other signs of dehydration may accompany. Beta blocker medications, taken for high blood pressure, heart disease, or other indications, may cease the ability to increase the compensatory heart rate.

LABORATORY FINDINGS INDICATIVE OF HYPOVOLAEMIA

Hypovolaemia, depending on the relative amounts of water and sodium losses can result in elevated markers of tissue hypoperfusion. The most typical and earliest to be present is the rise in plasma BUN/creatinine ratio > 20 (BCR, both values are expressed in mg/dl). Again, this cannot serve as a single diagnostic criterion of hypovolaemia, because the BCR can be also increased by:

- pre-renal failure,
- congestive heart failure,
- bleeding,
- increased protein intake,
- licorice excessive intake,
- lack of muscle tissue (which lowers the plasma creatinine concentration),
- glucocorticoids.

Table 2. Staging of blood losses.

Haemorrhagic shock class/stage	Stage 1	Stage 2	Stage 3	Stage 4
Blood loss	< 15% (< 750 ml)	15-30% (750-1500 ml)	30-40% (1500-2000 ml)	> 40% (> 2000 ml)

Table 3. Clinical signs by stages/classes of haemorrhage at presentation, subject to the increase on further treatment course (modified from MCS guidelines (17)).

Signs\stage of haemorrhagic shock	Stage 1	Stage 2	Stage 3	Stage 4
Constriction of vascular bed	Cardiac output =	Cardiac output ↓	Hypovolemic shock	Hypovolemic shock
Blood pressure Systolic Diastolic Pulse pressure	 = = =	 = ↑ Narrow	< 105 mmHg	< 70 mmHg
Heart rate	< 100 bpm	> 100 bpm	> 120 bpm	> 140 bpm
Respiratory rate	=	↑	> 30 rpm	Pronounced
Skin	Pale	Pale, cold	Pale, cold, wet	Pale, cold, wet
Mental status	Normal/slight anxiety	Mild anxiety	Confusion, agitation	Lethargy, coma
Capillary refill	=	↓	↓	0
Urine output	=	500-700 ml/24 h	500 ml/24 h	< 500 ml/24 h

= – normal; ↓ decreased; ↑ increased

More severe hypoperfusion induced tissue damage may be manifested by an increase in plasma lactic dehydrogenase (LDH), creatine kinase (CK) or even free myoglobin.

Usually, in chronic hypovolaemia the patient presents also hyponatraemia, hyposmolality, alkalosis, hyperuricosaemia, and metabolic alkalosis.

CLINICAL EVALUATION OF HYDRATION STATUS

As one can see from the data presented above, the evaluation of hydration status should be always accompanied by estimation of blood volume and plasma composition (mainly sodium and albumin concentrations). The chronicity of the condition, comorbidities and medication should be also taken into account. In the elderly the responsibility for detecting the early stages of dehydration are the responsibility of the staff and the family, because the signs precede symptoms, and the last are very unspecific and easy to be misinterpreted. Any sudden change in body weight, behaviour, mood or wellbeing of the elderly person should always raise a presumption of dehydration.

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received/otrzymano: 02.09.2015
accepted/zaakceptowano: 26.09.2015