
No elevation of blood urea level in a dehydrated patient with central diabetes insipidus

Sir,
Dehydrated patients usually present with elevated blood urea nitrogen (BUN) concentrations, reflecting a low urine flow rate and increased renal reabsorption of urea. This increased renal reabsorption of urea is thought to owe at least in part to the action of antidiuretic hormone (ADH).1

A 44-year-old Japanese man with mental retardation was admitted because of the recent onset of polyposia and polyuria. As he had taken any kind of fluids (e.g. swimming pool water) and he had not slept well due to polyuria, he was restricted fluid intake. Therefore, he showed mild hypertonenic dehydration on admission. His blood test results were as follows: Na, 151 mEq/l; K, 3.3 mEq/l; Cl, 117 mEq/l; BUN, 12 mg/dl; creatinine, 0.82 mg/dl; uric acids, 7.0 mg/dl and blood glucose, 97 mg/dl. Although plasma osmolality was elevated to 308 mOsm/kg, his urine was markedly diluted (urinary osmolality, 183 mOsm/kg). The renal reabsorption of urea was not enhanced in spite of dehydration (fractional excretion of urea: FEUN, 40.1%; reference range, less than 35% under dehydration).2 Administering desmopressin (5 μg intranasally) markedly increased urinary osmolarity to 762 mOsm/kg. Brain magnetic resonance imaging showed the absence of the hyperintense signal of the posterior pituitary on T1-weighted images without any remarkable lesions. Therefore, he was diagnosed as having idiopathic central diabetes insipidus (CDI) and was discharged with desmopressin. In the follow-up clinic, he could not take enough fluids during fever; thus, hypernatrenic dehydration occurred again (Na, 155 mEq/l), associated with a low BUN level (7 mg/dl) due to enhanced urinary excretion (FEUN, 82.7%).

In the present case, in spite of dehydration, the level of BUN was not elevated and renal reabsorption of urea was not increased. The urea permeability in the inner medullary collecting duct rises in the presence of antidiuretic hormone,3 suggesting that urea reabsorption in the distal nephron might be decreased in CDI even when the patients are dehydrated. An observation similar to ours, low BUN levels with hypertonic dehydration, has already been reported in CDI patients;4 however, it is not mentioned in any recent textbooks of internal medicine. Our observation suggests that BUN levels can be used to distinguish dehydrated CDI patients when hypernatenic dehydrated patients are seen.

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Tonsillar and lymph node tuberculosis revealing asymptomatic pulmonary tuberculosis

Sir,
Tonsillar tuberculosis is a rare presentation of extrapulmonary tuberculosis. Although tuberculosis of the tonsils is nowadays an uncommon finding, tonsillar granulomas are occasionally seen by histopathologists, and can sometimes contain tuberculous organisms. Occasionally, tonsillar tuberculosis may precede the diagnosis of pulmonary tuberculosis.1

We present the case of a 40-year-old patient admitted for evaluation of hoarseness and difficulty in swallowing, firstly diagnosed as recurrent angina. He had also had chronic bilateral cervical lymphadenopathy for the last three years. All preceding investigations were normal.

Clinical examination was normal, but head and neck examination revealed enlarged ulcerated
palatine tonsils and laryngeal oedema. Tonsillectomy and microlaryngoscopy were performed, and the histology revealed granulomas without caseous necrosis. Lung X-ray showed some bilateral non-specific mediastinal lesions. Angiotensin convertase was normal.

Despite the normality of these standard assessments, thoracic tomodensitometry and bronchial fibroscopy with bronchopulmonary washing were done, and apical and mediastinal pulmonary lesions were observed. Pathological findings included caseous granuloma and positive culture for acid-fast bacilli (Mycobacterium tuberculosis), permitting the diagnosis of pulmonary tuberculosis with tonsillar involvement. Moreover, the presence of acid-fast bacilli in the bronchial lavage suggested the diagnosis of a possibly reactivated pulmonary tuberculosis. Anti-tuberculous therapy resulted in rapid improvement of the pulmonary lesions and resolution of the pseudo-anginal symptoms. Cervical lymphadenopathy progressively regressed.

Tuberculosis is occasionally found in the head and neck, where it generally presents as tonsillitis, laryngitis, or pharyngitis.\(^2\)\(^,\)\(^3\) In a retrospective series of 22 tonsillar granulomas, tuberculosis was diagnosed only in three cases (14%).\(^4\) Nowadays, this localization is thought to appear mostly in immunodeficient patients, as with the co-infection with tuberculosis in the upper respiratory/digestive tract and human immunodeficiency virus recently described.\(^5\) The presenting symptoms and abnormal tonsillar findings of tonsillar tuberculosis are similar to those of malignant tumours, and hence it is sometimes difficult to differentiate the two pathologies. Sporadically, tonsillar tuberculosis could represent the first manifestation of pulmonary tuberculosis.\(^1\) Chronic lymphadenopathy could also occasionally be the inaugural symptom of tuberculosis, either isolated, or in association with other clinical manifestations.\(^6\) In a series of 16 cases of chronic lymphadenopathy disclosing tuberculosis, other associated symptoms were observed in 12 patients (75%).\(^6\)

In our patient, who was immunocompetent, histopathological and bacteriological lung investigations established a diagnosis of tonsillar and lymph node tuberculosis associated with asymptomatic pulmonary localization. This is further evidence that tonsillar tuberculosis and/or chronic lymphadenopathy may represent the first manifestation of tuberculosis. The possibility of tonsillar tuberculosis should be considered when unexplained enlarged tonsils are observed, and should be remembered for the differential diagnosis of atypical recurrent angina, even in immunocompetent patients.

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