Recurrent urachal adenocarcinoma

Primary adenocarcinoma of the bladder accounts for less than 1% of all bladder malignancies.1 Of these, 20–39% are urachal in origin. Urachal adenocarcinoma is more common in men and tends to present in the 5th or 6th decades, although it has been described in a 15 year old girl.2 The common presenting symptoms are haematuria, pain, irritative symptoms, and mucousuria.3 We report a 37 year old woman who presented with a history of irritative voiding symptoms and a suprapubic mass. Five years earlier, she had undergone a laparotomy, partial cystectomy, and excision of a urachal tumour at another centre. Cystoscopy revealed no definite intravesical lesion, apart from a small diverticulum at the site of the previous bladder resection. A bladder biopsy showed a minimal degree of oedema and chronic inflammation, but no other abnormality. A contrast computed tomography scan revealed a mass lesion deep to the anterior abdominal wall, measuring 5 cm in diameter. It was consistent with recurrent carcinoma in the region of the urachal remnant. At laparotomy, we found a mass lesion, situated 10 cm superior to the dome of the bladder, which was attached to the posterior wall of the rectus abdominus muscle. A wide excision of the mass was performed including a large ellipse of skin and rectus muscle. A portion of the dome of the bladder was excised separately to exclude microscopic involvement, and lymph nodes from the omentum were also biopsied.

Histologically, such tumours are often mucinous in phenotype, as was the case in our patient.4 The tumour was multicystic and infiltrated the muscle in an expansile fashion. The epithelium showed only minor atypia, with nuclear hyperchromasia and stratification (fig 1). Comparison of the histology with the resection specimen taken five years earlier showed an identical picture, confirming this as a recurrent urachal adenocarcinoma.

The prognosis for urachal adenocarcinoma does not differ significantly from non-urachal adenocarcinoma, and is relatively poor, with a five year survival of 37% and a 10 year survival of 17%.5 This may result from the relatively late presentation and advanced stage of these tumours. The treatment is primarily surgical, with extended partial or total cystectomy, and en bloc excision of the urachal mass, urachal tract, and umbilicus advocated.6 These tumours are radioresistant and the results of adjuvant chemotherapy are not yet clear.

Urachal adenocarcinoma is an uncommon malignancy that often presents late because of its location and relatively non-specific symptoms. This probably results in its poor prognosis. Adequate primary surgery and close follow up is the treatment of choice. Importantly, despite the poor published prognosis and presentation with stage III disease, our patient has survived with only a local recurrence after five years.

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References

An immunodiagnostic method for the detection of Shiga-like toxigenic Escherichia coli in faeces

Escherichia coli O157 is one of the most common serotypes of shiga-like toxin producing E coli, and has been associated with serious disease and several high profile outbreaks.6

The Quix™ Rapid E coli O157 Strip Test (Universal HealthWatch Inc, Columbus, North Carolina, USA) is an immunochromatographic test that detects the O157 antigen using a colloidal gold labelled antibody. We chose to evaluate the Quix™ Strip Test for the detection of E coli O157 in faecal specimens of patients with bloody diarrhoea or clinical symptoms typically associated with haemolytic uraemic syndrome.7

Two hundred and fifty eight consecutive patient specimens referred to our routine diagnostic laboratory with indications described above were entered into our study. Performance of the Quix™ Rapid E coli O157 Strip Test was compared with growth on sorbitol MacConkey agar, a method used for routine detection of E coli O157 in our laboratory.8 The test was performed according to the manufacturer’s instructions. Briefly, a sample of faeces approximately 5 mm in diameter was suspended in five drops of buffer solution. A strip was placed in the tube and the sample allowed to migrate along the strip for five minutes. The results were then read directly from the strip.

Of the 258 specimens included in our study, eight yielded E coli O157 on sorbitol MacConkey agar and all eight specimens were identified using the Quix™ Rapid E coli O157 Strip Test.

We also tested a sample previously culture positive for E coli O157 that had been stored at 4°C for six months. This produced a positive result with the Quix strip despite no growth on re-culture. This demonstrates that the strip test can function in the absence of viable bacteria, and therefore may be used for the retrospective study of specimens.

Limitations of the test include the high cost and inability to detect serotypes of shiga-like toxigenic E coli other than O157. Few studies have assessed the frequency at which non-O157 serotypes cause disease, although a two year study in Canada reported that non-O157 shiga-like toxigenic E coli were isolated from 0.7% of patient specimens.9 Further evaluation of the incidence of non-O157 shiga-like toxigenic E coli would be required to determine the value of this test.

The results of our limited study suggest that the strip test is highly sensitive and specific and larger scale studies may be warranted. The rapid turn around time is advantageous, providing clinicians with a prompt diagnosis, and the test may prove suitable for near patient testing. Overall, the Quix™ Rapid E coli O157 Strip Test was quick, accurate, and simple to use.

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The findings appeared suggestive, but not these cases had minimal granular deposits. Sections were taken from the biopsy observed initially, but was seen when further cartilage in the bone marrow biopsies; there that were not normal or had leukaemic large numbers of fine to coarse granules, preparations had purple granular deposits. Five cases were interpreted as

maps and/or bone marrow biopsy...granular deposits."...bone marrow biopsy. The odd case with apparently no cartilage results from the orientation of the bone marrow biopsy, such that a particular section may fail to include the cartilage. In contrast, the presence of only equivocal sparse granularity in a sample containing cartilage can be explained by the proportion constituted by, and the position of, the cartilage within the biopsy, and the way it has been touched on to a slide. The high frequency of cartilage in the trephine bone marrow biopsy of children has been documented previously, and these investigations were performed. However, the results of urine biochemistry obtained in the assessment of severe hyponatraemia are rarely reported.

We examined the computer records of 59 patients (44 women, 15 men; age range, 22–93 years) found in 2002 to have a serum sodium of less than 120 mmol/litre, in whom a urine osmolality was requested at around the same time (the total number of patients with a serum sodium of less than 120 mmol/litre during the same period was 304). Case notes were available for 34 of these patients. Figures 1 and 2 show the results of urine osmolality and sodium measurement, respectively. Serum osmolality confirmed a hypomolar state in 41 of the 45 patients in whom it was measured. Of the four patients in whom a hypo-osmolar state was not confirmed, three were uraemic and one was hyperglycaemic. Cortisol was measured, either randomly or as part of a short synacthen test, in 28 patients; it was greater than 560 nmol/litre in all but two, both of whom had secondary adrenal insufficiency, one as a result of treatment with high dose steroids, and the other because of failing to comply with steroid replacement after hypophysectomy. Thyroid stimulating hormone was in the hypothyroid range in eight of 36 patients, although hyponatraemia was attributed to hypothyroidism in only two of these. Clinical information sought included volaric status (for example, dehydration and oedema), postural hypotension, clues from the history about the likely mechanism of hyponatraemia, and how the patient was.

We conclude that purple granular deposits that impair the evaluation of bone marrow touch preparations are a result of the presence of cartilage in the bone marrow biopsy, and are seen more often in children.

**References**


**Results of measurement of urine osmolality and sodium in the evaluation of severe hyponatraemia**

Measurement of urine osmolality and urine sodium is often advocated in the evaluation of severe hyponatraemia. In a recent article in the *Journal of Clinical Pathology*, Saced and colleagues documented the frequency with which these investigations were performed. However, the results of urine biochemistry obtained in the assessment of severe hyponatraemia are rarely reported.

We examined the computer records of 59 patients (44 women, 15 men; age range, 22–93 years) found in 2002 to have a serum sodium of less than 120 mmol/litre, in whom a urine osmolality was requested at around the same time (the total number of patients with a serum sodium of less than 120 mmol/litre during the same period was 304). Case notes were available for 34 of these patients. Figures 1 and 2 show the results of urine osmolality and sodium measurement, respectively. Serum osmolality confirmed a hypomolar state in 41 of the 45 patients in whom it was measured. Of the four patients in whom a hypo-osmolar state was not confirmed, three were uraemic and one was hyperglycaemic. Cortisol was measured, either randomly or as part of a short synacthen test, in 28 patients; it was greater than 560 nmol/litre in all but two, both of whom had secondary adrenal insufficiency, one as a result of treatment with high dose steroids, and the other because of failing to comply with steroid replacement after hypophysectomy. Thyroid stimulating hormone was in the hypothyroid range in eight of 36 patients, although hyponatraemia was attributed to hypothyroidism in only two of these. Clinical information sought included volaric status (for example, dehydration and oedema), postural hypotension, clues from the history about the likely mechanism of hyponatraemia, and how the patient was.

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Urine osmolality was 100 mmol/kg or more in 54 of 59 patients, and was less than 600 mmol/kg in all but one patient, who had septic shock (fig 1). These data raise several issues relating to the usefulness of urine osmolality results in the assessment of hyponatraemia. First, the results obtained from the measurement of urine osmolality merely confirm the presence of impaired water excretion in most hyponatraemic patients. Formal diagnosis of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) may require the demonstration of a urine osmolality that is inappropriately high for the serum osmolality, but it should be obvious clinically when water excretion is not impaired; maximally dilute urine is associated with very high urine flow rates (in excess of 500 ml/hour). Second, impaired water excretion, however it is established, does not in itself establish the mechanism of hyponatraemia. It may signify inappropriate antidiuresis, but is equally consistent with sodium depletion (the hypovolaemia resulting from sodium and water loss is a powerful non-osmotic stimulus to antidiuretic hormone secretion). Thus, knowledge of the patient’s volemic status is essential for the meaningful interpretation of urine osmolality results. Third, the ability of urine osmolality to discriminate between mechanisms of hyponatraemia may be limited, given the relatively narrow range of urine osmolality (100 to 600 mmol/kg) observed in all but six patients. However, this is speculative, and much larger numbers are required to assess whether this is the case; only two patients were identified with clear evidence of sodium depletion.

In 27 of 45 patients, urine sodium was less than 20 mmol/litre (fig 2). Only 12 patients had urine sodium concentrations in the range typically observed in SIADH (40 mmol/litre or more). The combination of impaired water excretion and sodium retention is characteristic of effective circulating volume depletion. Other causes of true hyponatraemia and impaired water excretion are associated with higher urine sodium concentrations (greater than 40 mmol/litre). What do these findings tell us about the prevalence of hypovolaemia in severely hyponatraemic patients? The clinical information we reviewed retrospectively (not shown) was incomplete, and it was often not possible unequivocally to attribute hyponatraemia to a specific cause or mechanism. These limitations also apply in some degree to previous surveys of hyponatraemia, including that of Saeed and colleagues. Nevertheless, it may come as a surprise that we did not find a greater number of higher urine sodium concentrations in this population. Our findings underline the need for complete relevant information to be collected if the prevalence of different causes and mechanisms of severe hyponatraemia is to be established with any degree of accuracy, and, just as importantly, if the clinical usefulness of urine biochemistry in the evaluation of severe hyponatraemia is to be better defined.

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