Unusual presentation of more common disease/injury

Two unusual cases of hypothyroidism with renal dysfunction

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Summary

We describe two cases in which the initial finding of renal impairment guided further investigation leading to the diagnosis of hypothyroidism. In the first case, a 40-year-old female patient presented with a complaint of facial puffiness. On investigation, her serum creatinine was 1.86 mg/dl, glomerular filtration rate (GFR) by Cockcroft-Gault equation was 40 ml/min, free thyroxine (FT4) was <1 mg/dl and a thyroid stimulating hormone (TSH) concentration >80 µl. In the second case, a 45-year-old male patient presented with complains of indigestion, weight gain and lethargy. On examination, his blood pressure was 160/100 mm Hg and there was facial puffiness and mild pedal oedema. On investigation, her serum creatinine was 1.5 mg/dl and estimated GFR by Cockcroft-Gault equation was 55 ml/min. Thyroid function was abnormal, FT4 2.99 mg/dl and TSH 80 µl. In both the cases, thyroid hormone replacement therapy brought about complete recovery of renal function.

BACKGROUND

Renal derangement in a patient is of high concern for physician as well as relatives of patient. This is mostly because of fear that it may progress to chronic renal failure. In these two cases, renal impairment was attributed to hypothyroidism which fully recovered after giving hormone replacement therapy.

CASE PRESENTATION**Case 1**

A 40-year-old female patient presented with chief complain of facial puffiness. There was no significant medical history. On examination, her blood pressure was 150/100 mm Hg, pulse rate 56/min and there was facial puffiness. Rest of the examination was within normal limit.

Case 2

A 45-year-old male patient presented with complaints of indigestion, weight gain and lethargy for last 6 months. There was no significant medical history. On examination, his blood pressure was 160/100 mm Hg, pulse rate 70/min, there were facial puffiness and mild pedal oedema. Rest of the examination was within normal limit.

INVESTIGATIONS**Case 1**

Routine investigation includes haemoglobin 10.5 g/l, white cell counts 6500/dl, S. Sodium 137 mEq/l serum potassium 3.7 mmol/l. Serum creatinine 1.86 mg/dl, estimated glomerular filtration rate (GFR) by Cockcroft-Gault equation 40 ml/min. Urea 21 mg/dl, Cholesterol 390 mg/dl, SGOT 95 IU/l, SGPT 42 IU/l, alkaline phosphatase 85 IU/l, albumin 4.5 g/dl, calcium 9.7 mg/dl and glucose 90 mg/dl. Twenty-four hours urinary protein was 400 mg.

Ultrasonography revealed both kidneys were normal in size and echotexture. Renal biopsy was undertaken, 15–18

glomeruli were analysed by light microscopy, immuno-histochemistry and electromicroscopy was inconclusive. On 2-D Echo cardiography small pericardial effusion was present, magnetic resonance angiography showed no evidence of renal artery stenosis.

Thyroid function tests showed a free thyroxine (FT4) concentration of < 1 mg/dl and a thyroid stimulating hormone (TSH) concentration >80 µl, consistent with severe hypothyroidism.

Case 2

Routine investigations revealed haemoglobin 13.5 g/dl, white cell count 7200/dl, sodium 135 mEq/l, potassium 4.2 mEq/l, serum creatinine 1.5 mg/dl, estimated GFR by Cockcroft-Gault equation was 55 ml/min, urea 20 mg/dl, calcium 9.4 mg/dl, cholesterol 255.2 mg/dl, SGOT 94 IU/l, SGPT 42 IU/l, alkaline phosphatase 72 IU/l, serum bilirubin 0.4 mg/dl, serum albumin 4.5 g/dl and random blood glucose 140 mg%, 24 h urinary protein was 455 mg. Ultrasonography revealed that both kidneys were normal in size and echotexture. Thyroid function was abnormal, FT4 2.99 mg/dl and TSH 80 µl.

TREATMENT

Both patients were started with levothyroxine 100 µg/day.

OUTCOME AND FOLLOW-UP**Case 1**

She was asked to come in follow-up fortnightly. Her symptoms improved gradually. After 3 months, her blood pressure and serum urea and creatinine were all normalised.

Case 2

He was also asked to come in follow-up fortnightly. Over a period of 4 months, his symptoms improved and his renal function was normalised (serum creatinine 0.6 mg/dl).

DISCUSSION

We describe two cases in which the initial finding of renal impairment guided further investigation leading to the diagnosis of hypothyroidism. In both cases, thyroid hormone replacement therapy brought about complete recovery of renal function.

The pathophysiology of impaired renal function in hypothyroidism is multifactorial, the reduction in GFR due to the lower cardiac output and renal blood flow is likely to be the predominant mechanism.¹ It has also been suggested that thyroxine may mediate tubular secretion of creatinine.² Furthermore, hypothyroidism, may increase creatinine release from muscle.³ Although freely filtered, additional tubular secretion of creatinine renders it a poor marker of GFR. In patients with hypothyroidism, clarification of whether an elevated serum creatinine represents true renal impairment (ie, reduced GFR) or simply increased generation, and tubular secretion, of creatinine therefore requires further analysis by the way of isotope GFR studies.

Hypothyroidism has also been described as the consequence, rather than the cause, of renal dysfunction, thyroxine is heavily protein bound and is lost via the urine in nephrotic syndrome.⁴ Sub-clinical hypothyroidism (elevated TSH levels with normal FT4 levels) and clinically apparent hypothyroidism occur in 18–20% of patients with chronic kidney disease, not requiring renal replacement therapy, with the prevalence rising as the degree of renal impairment worsens.^{5 6}

To the best of our knowledge, four such cases are earlier reported.^{7 8} In all cases, renal function improved after hormone replacement therapy.

Although further studies are required to consider hypothyroidism to be a reversible cause of renal

dysfunction, we recommend that thyroid function be assessed early in patients with renal impairment.

Learning points

- Whenever you encounter a patient with deranged renal function without apparent cause, investigate for thyroid function also.
- In such case, treating hypothyroidism may revert renal derangement.

Competing interests None.

Patient consent Obtained.

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