Pitfalls in non-utilization of basic clinical methods in the detection of kidney disease: a case report

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Abstract

Background: Chronic kidney disease (CKD) is a devastating problem worldwide. Early detection and intervention usually lead to more favourable outcomes. However, CKD is sometimes under diagnosed except in its very late stages where little can be done. This is partly because a lot of interns and physicians do not apply basic clinical methods of history, physical examination and laboratory investigations to evaluate patients.
Methods: A case report involving a 32 year old woman who presented to a health facility with complaints of abdominal pain and vomiting. A proper medical history was not taken, basic physical examinations were not done and baseline investigation were not carried out or interpreted properly.
Results: This caused the patient to undergo needless laparotomy.
Conclusion: There is need for medical doctors to acquaint themselves with knowledge of basic medical skills needed for detection of kidney disease. This will aid early detection and institution of appropriate measures.

Keywords; Chronic kidney disease, misdiagnosis, basic clinical methods, early detection

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Introduction

The magnitude of chronic kidney disease (CKD) is enormous and its prevalence is increasing[1]. Data from around the world suggest that the prevalence is between 10-16% with an annual growth rate of 8%[1-2].The criteria for its definition are objective and can be ascertained by means of simple laboratory tests without identification of the cause of disease, thereby enabling detection of CKD by non-nephrologist physicians and other health professionals[3]. Despite this, CKD is still under diagnosed and under recognized all over the world[4]. Recent studies have shown that the knowledge of CKD is poor among the general populace, [5-6]trainee doctors[7] and specialist physicians[8]. It can manifest in subtle ways, with myriad of complications and sometimes in atypical manners; thereby slipping beneath the watchful eyes of the health care worker[9]. Hence, the importance of proper utilization of basic clinical methods in the detection of CKD cannot be overemphasized.

Case Presentation

Patient is a 32 year old female trader who was referred from a peripheral private hospital post laparotomy on account of a deranged electrolytes and urea result. The history at the referral hospital was that of abdominal pains of 3 days duration prior to presentation and vomiting also for a period of 3 days. Physical examination showed that patient was pale. No other general examination findings were noted. There was marked epigastric tenderness, no flank tenderness and no masses were palpable per abdomen. Abdominal ultrasound scan was done which was said to have shown nothing of significance. Patient was then diagnosed as a case of acute abdomen and an exploratory laparotomy with no significant findings at laparotomy. Patient was transfused on account of a packed cell volume of 18%.The vomiting persisted in the patient and patient developed fever post operatively. Post-operatively the urea was 300mg/dL (15-50 mg/dL) and creatinine was 6.7mg/dL (0-1.3 mg/dL). Potassium was also elevated, 5.7mmol/L (3.5-5.5mmol/L) and bicarbonate was 12mmol/L (20-25mmol/L). On account of this result and the persistent vomiting, patient was referred to the University of Benin Teaching Hospital (UBTH), Nigeria.

At the UBTH, past medical history taken from patient revealed that patient is a known chronic kidney disease(CKD) patient diagnosed 2 years earlier during her last pregnancy. Patient was also told 2 years earlier that she was suffering from kidney disease due to hypertension and subsequently had 3 sessions dialysis. She however defaulted on medications and follow up. Physical examination revealed a young woman who was chronically ill-looking, pale with bilateral leg swelling up to the knees. Her blood pressure was 160/100mmHg;

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there was a locomotor brachialis, displaced apex beat and a fourth heart sound. Dilated funduscopy showed papilloedema (Grade 4 hypertensive retinopathy). There was asterixis. All other examination findings were unremarkable. Her weight was 65kg. A bedside urinalysis showed 2+ for proteins. Results of urgent investigations showed a blood urea nitrogen of 279 mg/dl, serum creatinine 10 mg/dl, potassium 6.3 mmol/l, bicarbonate 9 mmol/l and haemoglobin 6 g/dl. Renal Ultrasound Scan done in UBTH showed bilaterally shrunken hyper echogenic kidneys with loss of cortico-medullary differentiation. The renal sizes were 6.0cm X 2.9cm and 6.5cm X 2.5 cm for the right and the left kidneys respectively. The estimated glomerular filtration rate (eGFR) at presentation was 8.3mls/min./1.73m^2 (based on the modification of diet in renal disease formula[10]); consistent with end stage renal disease. The patient was therefore diagnosed of an end stage renal disease secondary to hypertension complicated by uraemia, anaemia and hyperkalemia. She received an urgent session of haemodialysis, admitted into the ward and managed in line with the diagnosis.

Discussion

Patient was diagnosed of CKD two years before presentation to the peripheral hospital and had received three sessions of haemodialysis. The history taken at the peripheral hospital did not inquire about the patients' past medical. She presented with acute abdomen and vomiting from uremic gastritis. This was mistaken for a surgical abdomen and exploratory laparotomy was performed. Nothing was found on surgery. The possibility of uremic gastritis was not considered as a cause for vomiting, Patient was anemic at presentation in the peripheral private hospital, the possibility of anaemia of renal origin was not entertained at the same hospital. Past medical history of renal disease was not obtained at the peripheral hospital where this patient was evaluated. Pre-anaesthetic renal function tests and urinalysis were not done. If a simple urinalysis had been done, the proteinuria would have been detected and the possibility of renal disease would have been suspected. Abdominal ultrasound scan done did not take note of renal sizes, echogenicity or cortico-medullary differentiation. The renal ultrasound scan done at UBTH showed evidence in keeping with a chronic renal parenchymal disease. Serum biochemistry also showed markedly deranged urea, creatinine and electrolyte values.

This underscores the need for physicians to be aware of the multiplicity of ways uraemia may present. There is need to follow the time-honoured structured approach of basic clinical medicine in every patient assessment. A good history, physical examination and laboratory investigations which includes basic bedside urinalysis and ordering of appropriate laboratory results and judicious interpretation of those results would have averted a laparotomy in this patient. These basic clinical methods were not applied in this patient and caused remarkable morbidity. There is need therefore to continue to acquaint doctors and other medical personnel with the myriad ways in which CKD may present

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