Recurrent Hypoglycaemia and Seizures in an HIV-patient .

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A young male patient recently diagnosed with HIV presented to us with a septic tibia. He developed recurrent seizures and hypoglycaemia. Terminally he developed a clinical picture of Addison's crisis and disseminated intravascular coagulation. Addison's crisis must always be borne in mind in patients with HIV who are subjected to stressful conditions like surgery.

Intrduction

It is estimated that the number of people with HIV worldwide is 33.2 million[1]. HIV is a serious health issue, particularly in developing countries. There is great controversy whether HIV per se predisposes to higher incidence of post - operative infection[2,3,4,5]. Serious musculo-skeletal infection occurs in advanced or WHO stage 3 disease. Seizures can be the presenting symptoms in 2- 20% of HIV-positive patients.[1] The cause can be infective or non-infective. Hypoglycaemia can be due to drugs, metabolic or hormonal disturbances. We report on a HIV - positive patient who developed recurrent hypoglycaemia, seizures and Addison's crisis during treatment for a septic united tibia.

Case Report

A 43 year-old male presented to the orthopaedic trauma unit with a clinical problem of septic united tibia. He had intramedullary nail three years ago. He was recently diagnosed with HIV. The CD4 count was $78x10^6$/litre and he was not on anti-retroviral treatment. Clinically he looked well. He was apyrexial. The only clinically relevant findings were cervical lymphadenopathy.

The nail was removed and reaming sent for microscopy, culture and sensitivity. Culture results isolated Proteus Mirabilis and Streptococcus pyogenes sensitive to cloxacillin, ampicillin and bactrim (trimethoprim plus sulphamethoxazole). Intravenous treatment was started with the first two drugs and continued for 35 days. Oral therapy with bactrim (160mg trimethoprim + 800mg sulphamethoxazole) two tablets twice daily for four days was commenced.

A day after stopping oral therapy with bactrim, he developed hypoglycaemia with a blood glucose of 1.6 mmol/l (normal = 4.1- 59 mmol/l) and coma. He was resuscitated with 50% dextrose intravenously and the infusion continued with 10% dextrose - normal saline. Eight hours later he developed generalizedtonic clonic seizures. Blood glucose level was 2.4 mmol/l. Resuscitation was done using the same regime as before. The following morning, approximately five hours later, he had a second generalized tonic clonic seizures which lasted a minute. The glucose level was normal. His clinical condition stabilized. The last episode of hypoglycaemia (blood glucose = 1,1 mmol/l) with no convulsions occurred four hours later, was accompanied by low blood pressure (90/60 mmHg) and hypoventilation. The third seizure occurred six hours later.

Table 1. Laboratory Data

Test	Normal values	22/8/8	5/9/8	17/9/8	2/10/8	2/10/8
RCC	4.89-6.11 x 10^{12}/l	3,56			2,71	
HB	14.3-18.3 g/dl	9,0			6,4	
HCT	0.43-0.55 l/l	0,287			0,186	
MCV	79.1-98.9 fl	80,6			68	
MCH	27.0-32.0 pg	25,3			23	
MCHC	32.0-36.0 g/dl	31,4			34	
RCDW	11.6-14.0 %	15,4%			18 %	
PLT	137-373 x 10^9/l	222			80	
WCC	4.0-11.0 x 10^9/l	3,11			2,12	
NEUT		57 %				
LYMP		19 %				
MONO		22,5%				
EOSIN		0,6 %				
BASO		0,3 %				
NA	135-147 mmol/l	131	133	131	128	120
K	3.3-5.3 mmol/l	3,8	3,1	3,2	5,1	5,4
CL	99-113 mmol/l		94	93	88	83
UREA	2.6-7.0 mmol/l	4,1	3,9	5,2	40	39
CREAT	60-120 ųmol/l	79	66	117	734	677
AG	7-17 mmol/l		16		30	
CRP	0-10 mg/l	83,3			279	
CD4	500-2010 x 10^6/l				2	
PT	10-14 seconds				21	
INR	0.9 -1,2				1,74	
PTT	26-36 seconds				60	

Abbreviation:
RCC = Red cell count, HB = Haemoglobin, HCT = Haematocrit, MCV = Mean corpuscular volume, MCH = Mean corpuscular haemoglobin, MCHC = Mean corpuscular haemoglobin concentration, RCDW = Red cell distribution width; PLT = Platelets, WCC = White cell count, NEUT = Neutrophil, LYMP = Lymphocytes, MONO = Monocytes, EOSIN = Eosinophils, BASO = Basophils, NA = Sodium, K = Potassium, CL = Chloride, CREAT = Creatinine, AG = Anion gap, CRP = C-reactive protein, PT = Prothrombin time, INR = International normalized ratio, PTT = partial thromboplastin time

Three hours after the hypoglycaemia and shock he developed generalized seizures and severe hypoglycaemia (blood glucose = 0.6 mmol/l). The final hypoglycaemic episode (blood glucose = 0,9 mmol/l)occurred an hour later. Resuscitation was unsuccessful. The blood results show a picture of Addisonian crisis plus disseminated intravascular coagulation. Laboratory data and normal ranges are shown in Table 1.

Discussion

Recurrent hypoglycaemia in this patient could be due to either cotrimoxazole therapy or Addisonian crisis. Seizures could be due to metabolic (hypoglycaemia) or infective causes. Cerebral infection was unlikely because the patient recovered well between episodes of seizures. He had no evidence of neurological deficit. Addisonian crisis showed a full-blown picture in the terminal stage: hypotension, hyperkalaemia, hyponatraemia and hypoglycaemia.

Cotrimoxazole is known to cause hypoglycaemia. Hypoglycaemia may be prolonged: lasting for more than 12 hours.[6] The drug can induce demand-related or over-use hypoglycaemia.[6] Patients at risk are those with renal failure.

Subclinical adrenal dysfunction is common in HIV-positive patients.[7] Patients have marginal adrenal reserves.[8,9] Clinically significant adrenal insufficiency is not common.[9] Adrenal failure is the most serious complication in these patients. It is not clear from the literature whether adrenal insufficiency should always be excluded in patients with HIV, especially if they are

subjected to stress; like surgery. Mohsin Saley Eledrisi et al.[8] state that identification of adrenal insufficiency in HIV-positive patients is imperative.

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