Neutropenia as a Probable Side Effect of Regular Insulin Therapy: A Case Report

ABBAS M ABBAS¹, WALID G BABIKR^{2*}, AARIF ALHASSAN³, NAFAA ALNAFAA⁴, ABDULLAH I. AEDH² and HAMDAN ALSHEHRI²

 ¹Endocrinologist, Domat Al-gendal Hospital, Aljouf-Saudi Arabia.
²Assistant Professor of Internal Medicine, Faculty of medicine, Najran University, Saudi Arabia.
³Cosultant Physician, the diabetes center, Al-jouf, Saudi Arabia.
⁴Medical Specialist, Domat Al-gendal Hospital, Aljouf-Saudi Arabia. Corresponding auther: Dr. Walid G Babikr
*Corresponding author E-mail: walidbabikr@yahoo.com

http://dx.doi.org/10.13005/bpj/1133

(Received: February 14, 2017; accepted: March 02, 2017)

INTRODUCTION

Neutropenia or [agranulocytosis] means reduced number of circulating neutrophils. It is commonly defined as an absolute neutrophil count [ANC]more than two standard deviations below the normal mean.¹ Neutropenia may be classified as mild (1.0 to 1.5 \times 109/L), moderate (0.5 to 1.0 \times 109/L), or severe (<0.2 \times 109/L), ² the later can markedly increase susceptibility to devastating bacterial infections and a significant increase in mortality risk.³ The association between neutropenia and medical drug use has been recognized since earlier in the twentieth century by investigators.⁴ Drug-induced American hematological disorders can span almost the entire spectrum of hematology, aûecting red cells, white cells, platelets, and the coagulation system.⁵ Druginduced neutropenia can be a side effect of many analgesics, psychotropics, anticonvulsants, antithyroid drugs, antihistaminics, antirheumatics, GI drugs, antimicrobials, cardiovascular drugs, and, as expected, with chemotherapy drugs.6,7 In the literature, reports about insulin induced neutropenia are very scarce. This is an interesting case report of regular insulin induced neutropenia that has completely resolved with shifting to insulin analogues.

Case report

A 60-year-old Saudi male patient with history of long standing uncontrolled type 2 diabetes mellitus presented to the accident and emergency department of Domat Al-gandal hospital in Al-Jouf -Saudi Arabia on the 23rd of September 2016 complaining of polyuria and polydipsia associated with weight loss for the last 2 months. He stopped his oral hypoglycemic on his own 2 years prior to his presentation to the hospital due to his beliefs that they are harmful. One year ago, he was started on insulin therapy but he again stopped it shortly after initiation owing to the frequent hypoglycemic attacks he experienced. Since then he has remained off treatment. He is not known to have hypertension or any other chronic medical illness. He is not known to have any of the blood dyscrasias. He was a febrile at the time of presentation and there was no evidence of any viral or bacterial infection. He was not taking any medications. No family history of any blood disorder or other inherited illnesses. On examination, no abnormalities were detectable apart from being dehydrated. No odor of acetone was detectable and he was breathing quite normally. All vital signs were stable. The chest was clear and the abdomen was soft without any tenderness. CNS examination revealed features of peripheral neuropathy and fundoscopy showed non-proliferative diabetic retinopathy. His laboratory investigations were as follows: Random blood sugar[RBS] was 847 mg/dl, serum creatinine was 214 mg /dl, serum sodium was 127 mg/dl, serum osmolality was 310. CBC was quite normal. Arterial blood gases[ABGS] were within normal limits. The chest x ray was normal and infection screening was non-revealing. He was admitted to the medical ward with the diagnosis of diabetic-non ketotic-hyperosmolar state and was started on the protocol of management including regular insulin. The patient responded well to the treatment but on day 3 of admission his CBC showed a drop of his white blood cell count and specifically the neutrophils[0.3x109/L]. We thought of regular insulin as the culprit. We then shifted the patient to short acting insulin analogues. His neutrophils then went back to normal within 3 days.

DISCUSSION

Many drugs are well known to cause agranulocytosis, although other causes like

immune mediation or direct inhibition of the bone marrow precursors are also common during clinical practice. Drug-induced neutropenia was first reported in 1931 by Kracke, attributed to administration of an analgesic (pyramiden) in patients with agranulocytosis.⁸ In the literature, as far as we know scarce was written about regular insulin as a cause of neutropenia.

CONCLUSION

Drug induced neutropenia is common during clinical practice and is encountered quite often with certain drugs among which regular insulin does not exist. The case well describes that regular insulin may induce neutropenia thus CBC should be monitored while patients are on treatment.

AKNOWLEDGEMENT

Special thanks go to the administration of Domat Al-Gendal General hospital,Al-Jouf –Saudy Arabia for endless co-operation

REFERENCES

- 1. [Dale DC. Neutropenia. eLS. 1990.]
- Haddy TB, Rana SR, Castro O. Benign ethnic neutropenia: what is a normal absolute neutrophil count?. *Journal of Laboratory and Clinical Medicine.* 133(1):15-22 (1999).
- Shapiro S, Issaragrisil S, Kaufman DW, Anderson T, Chansung K, Thamprasit T, Sirijirachai J, Piankijagum A, Porapakkham Y, Vannasaeng S, Leaverton PE. Agranulocytosis in Bangkok, Thailand: a predominantly drug-induced disease with an unusually low incidence. Aplastic Anemia Study Group. *The American journal of tropical medicine and hygiene.;* 60(4):573-7 (1999).
- Kiatboonsri P, Richter J. Unethical trials of dipyrone in Thailand. *The Lancet.*; 332(8626-8627):1491 (1988).
- Mintzer DM, Billet SN, Chmielewski L. Druginduced hematologic syndromes. *Advances in hematology* ;(2009).
- Bhatt V, Saleem A. Drug-induced neutropenia-pathophysiology, clinical features, and management. *Annals of Clinical & Laboratory Science*; 34(2):131-7 (2004).
- Mintzer DM, Billet SN, Chmielewski L. Druginduced hematologic syndromes. *Advances in hematology.* (2009).
- Kracke RR. Recurrent agranulocytosis. *Am J Clin Pathol;* 1:385 (1931).

480