Gentamicin Injection Induced Digital Necrosis in a 32-Year Old Apprentice: A Case Report

Alphonsus Udo*
Department of Family Medicine, College of Health Sciences, University of Uyo, Akwa Ibom State, Nigeria
*Corresponding author: Alphonsus udo idung, Department of Family Medicine, College of Health Sciences, University of Uyo, Akwa Ibom State, Nigeria, Tel: +234(0)8069768262; E-mail: dridung@yahoo.com

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Abstract

Gentamicin is a type of aminoglycoside produced by the fermentation of bacteria widely present in the environment. Because of its widespread use and abuse, their toxic effects have become more obvious and sometimes bizarre. Toxic effects involve the kidneys, ears and rarely neuromuscular junction as well as neurovascular bundles leading to ischemia and tissue necrosis. I present a 32-year old apprentice who developed bluish to black discoloration of the terminal phalanx of the index, middle, ring and little fingers of both hands following several injections of gentamicin. Digital necrosis arising from injection of gentamicin is rare in the scientific literature. Adverse effects of aminoglycosides can be prevented through proper regulation of its use.

Keywords: Tissue necrosis; Gentamicin injection; Aminoglycosides; Apprentice

Introduction

Gentamicin is a type of aminoglycoside which is produced by the fermentation of micromonospora purpurea, a genus of gram-positive bacteria widely present in the environment (water and soil) [1,2]. It is composed of linked ring of amino sugars and amino substituted cyclic polyalcohol (aminocyclitol). The moiety usually consists of 1 of 2 streptamine derivatives; streptidime or deoxystreptidime. The later may be subdivided into neomycin and kanamycin groups [3]. The later includes gentamicin, tobramycin and netilmicin [3].

The aminoglycosides are believed to act by binding to the 30s ribosomal subunit causing misreading of the mRNA codon, leading to errors in amino acid sequencing, disruption of polysomes, reducing the efficiency of protein synthesis, as well as inhibition of the translocation of tRNA between A and P ribosomal binding sites [4]. This process disrupts bacterial protein synthesis and eventually leads to cell death.

Gentamicin is a first-line antibiotic for many less-to-severe infections due to its fairly wide spectrum of activity, and low rates of resistance. It is a widely used and abused drug because of the ease with which it can be accessed even without prescription as well as its low cost compared to other antibiotics of comparable efficacy [5].

Because of the widespread use and abuse of the aminoglycosides, their toxic effects have become more obvious and sometimes bizarre. Examples of such toxic effects involve the ears and kidneys and rarely the neuromuscular junctions as well as neurovascular bundles leading to ischaemia and tissue death or necrosis [6-8].

Typically necrosis develops following inadvertent or deliberate direct intra-arterial injection of drugs leading to severe tissue ischaemia characterized by hyperemic, skin discoloration and haemorrhagic patch formation at or close to the injection site [9].

In this report, I present an apprentice who developed necrosis of the terminal phalanx of the index, middle, ring and little fingers of both hands following multiple intravenous injections of gentamicin in a drug dispensing shop.

Case Presentation

Thirty-two year old male furniture making apprentice presented to the out-patient clinic of University of Uyo Teaching Hospital with bluish discoloration of the terminal phalanx of the index, middle, ring and little fingers of both hands of one week duration (Figures 1 and 2).

Figure 1: Showing necrosis of terminal phalanx of fingers of left hand.
Discussion above.

There were no pulses present, but pain and touch sensations involving the fingers. Infection may then develop and spread to involve all the anatomical structures of the hand causing abscesses, cellulitis, necrotizing fasciitis, tenosynovitis, arthritis, osteitis and osteomyelitis. Destruction of the articular cartilage causes finger deformities, radial or brachial intra-arterial injections may cause hand ischemia and digital necrosis [10-13].

Blood Sugar 4.9 mmol/l (3.0–5.5 mmol/l). Haemoglobin concentration was 9.0 g/dl (11-17.5 g/dl). From the foregoing, there was no derangement in kidney function although patient was anaemic. Urine analysis did not reveal any abnormality. (Please note that numbers in parenthesis represent normal reference values used in the hospital where the index patient presented for treatment).

He initially reported the incident to the drug dispenser who promptly suggested he should seek help at the Teaching Hospital. Physical examination showed that he was in good condition and fairly well nourished but very anxious. Both hands showed dark discoloration of the terminal phalanx of the index, middle, ring and index fingers. There was associated wasting of the affected digits which were also cold to touch with no demonstrable capillary pulses were present, but pain and touch sensations involving the affected digits were lost.

Results of some laboratory investigations which the patient reluctantly agreed to do following his complaint of lack of money were as follows: Creatinine concentration: 109 mmol/l, (53–115 mmol/l); Urea: 3.4 mmol/l (2.1–7.1 mmol/l); Sodium: 139 mmol/l (135–145 mmol/l); Potassium: 4.1 mmol/l (3.2–5.0 mmol/l); Chloride: 99 mmol/l (96–108 mmol/l); Bicarbonate: 23 mmol/l (22–28 mmol/l); Fasting Blood Sugar 4.9 mmol/l (3.0–5.5 mmol/l). Haemoglobin concentration was 9.0 g/dl (11-17.5 g/dl). From the foregoing, there was no derangement in kidney function although patient was anaemic. Urine analysis did not reveal any abnormality. (Please note that numbers in parenthesis represent normal reference values used in the hospital where the index patient presented for treatment).

Discussion

Tissue necrosis caused by intramuscular injection has been reported with phenylbutazone, local anaesthetics, corticosteroids and diclofenac but report of tissue necrosis following gentamicin injection is rare in the scientific literature [14,15]. Some of the factors that have been reported to increase the risk of gentamicin toxicity include elevated trough gentamicin levels, duration of treatment on aminoglycoside, concomitant administration with other drugs such as vancomycin or frusemide, the type of aminoglycoside, and the frequency of aminoglycoside dosing and the timing of aminoglycoside administration [16]. The index patient took multiple doses of aminoglycoside, the frequency of dosing and the timing of administration was not certain since the drug was obtained from an unauthorized outlet.

Other risk factors might include inadvertent intra-arterial administration of the injection, lack of skin sepsis at the injection site, unsterile equipment as well as adulterants acting as irritant substances and foreign bodies. These factors are a result of people who are ill-trained handling medicines that ought to be properly regulated. These factors may also contribute to the disease presentation in the index patient. The index patient was referred to the surgical unit of the teaching hospital but he subsequently refused to co-operate with the surgical team and was eventually lost to follow-up. These further highlight the danger associated with wrong treatment choices that patients make.

Conclusion

Aminoglycoside-induced toxicity is avoidable. The ease with which very useful but easily abused drug like aminoglycoside can be assessed is worrisome. Agencies responsible for regulation of drug distribution should be up and doing. Education regarding indiscriminate use of drugs procured from unauthorized outlets should be stepped up. Extra steps should be made to rehabilitate victims of avoidable complications arising from improper drug use.

References


