The present case describes a patient with rhabdomyolysis due to hypothyroidism. Other known causes of rhabdomyolysis include collagen diseases, intake of massive amounts of alcohol, vigorous exercise, trauma, infections, seizures, medications like statins and electrolyte imbalances. If severe, rhabdomyolysis may be life threatening, especially when it is complicated by multiple organ failure. Muscle involvement is common in hypothyroidism and its myopathy is usually manifest with delayed relaxation of tendon jerks, proximal muscle weakness, myalgia and cramps. But, rhabdomyolysis is quite rare. Only a few cases of rhabdomyolysis due to hypothyroidism have been reported.\[1-5\]

The present case describes a patient suffering from rhabdomyolysis due to hypothyroidism, with no other precipitating factor. Diagnosis of rhabdomyolysis was carried out based on muscle weakness, grossly elevated CPK and elevated serum creatinine.

The exact cause of rhabdomyolysis in hypothyroidism is unclear, but both impaired glycogenolysis and impaired mitochondrial oxidative metabolism may be responsible.\[4,5\]

Hypothyroidism should be considered as one of the causes of rhabdomyolysis. Rhabdomyolysis manifests with muscular symptoms and severely elevated serum levels of muscle enzymes. Thyroid hormone replacement therapy improves thyroid and renal functions and reverses rhabdomyolysis.

---

PARACETAMOL INDUCED ANGIOEDEMA: MORE DETAILS REQUIRED

Sir,

I have read with keen interest the case report of a 4-year old boy who supposedly developed angioedema due to paracetamol.\[1\] The authors have attempted to justify with reasons that the angioedema was most likely due to paracetamol. The patient was treated for presumed viral infection; yet, the viral infection was not considered a possible cause of the angioedema. The fact that the reaction occurred within an hour of receiving the first dose of paracetamol and that it happened on just a single occasion made the reaction acute angioedema.\[2\] The commonest cause for a single isolated attack of angioedema is probably viral infection. Viruses are usually blamed because immune response to environmental
microbes may take an odd course to produce angioedema. This explanation is thought to be likely when this kind of reaction occurs in children.\[^2\]

A good past medical and drug history is a cornerstone to preventing and managing adverse drug reactions.\[^3\] Unfortunately, it was not properly explored in the patient. A previous report of hypersensitivity to paracetamol has shown a high incidence in children with personal/familial history of atopy or previous reaction to non-steroidal anti-inflammatory drugs (NSAIDs), acetylsalicylic acid (ASA) and antimicrobials.\[^4\] One wonders if this was the first episode of fever in this patient's lifetime. If not, how were they treated? Previous fever episodes were likely to have been treated with paracetamol or NSAIDs. Self-medication is a recognized problem in India\[^5\] and other developing countries.\[^6\] Paracetamol remained one of the most commonly self-medicated medicines for children and the medicine mostly kept at home by parents,\[^6\] therefore the likelihood of use of paracetamol or other medicines with potential for hypersensitivity reactions is high in the patient reported. The previous use of paracetamol, NSAIDs, ASA or antimicrobials might have sensitized the patient to produce the reported angioedema in a manner similar to type I allergic reaction or an aberrant non-allergic reaction, otherwise called idiosyncratic reaction.\[^7\] The only way to establish paracetamol hypersensitivity in this patient is to take a good clinical history or do oral challenge tests for NSAIDs, ASA and paracetamol when clinically stable.\[^3\] Unfortunately these were not done. The oral challenge tests should be performed in the hospital, under strict supervision. The formulation and source of the paracetamol is equally important in this case. The shelf life and stability of medicines kept at home, especially on a warm climate, are known to decrease over time, thereby increasing loss of potency and possible toxicity.\[^6\] The use of contaminated and adulterated paracetamol for children was responsible for multiple adverse reactions culminating in deaths in Nigerian children.\[^8\]

Overall, the case report was interesting but left so many questions unanswered. Publication of case reports describing suspected adverse reactions to drugs and medical products should provide sufficient details for either a differential diagnosis or provisional assessment of cause-effect association, or a reasonable pharmacological or biological explanation for the reaction.

KAZEEM A OSHIKOYA
Department of Pharmacology and Paediatrics, Lagos State University College Medicine, Ikeja, Lagos, Nigeria

Correspondence: Dr Kazeem A Oshikoya,
Academic Division of Child Health, University of Nottingham,
The Medical School, Derbyshire Children’s Hospital,
Uttoxeter Road, Derby DE22 3DT, United Kingdom.
E-mail: med_modhospital@yahoo.com
DOI: 10.4103/0019-5359.48557

REFERENCES
4. Boussetta K, Ponvert C, Karila C, Le Bourgeois M,
AUTHORS’ REPLY

Sir,

We are grateful to Dr. Oshikoya for his interest[1] in our report of a case of paracetamol angioedema.[2] We offer the following responses:

We agree that viral infection could be a possible cause for angioedema in children. However, as detailed in the case report, the morphological characteristics of the initial rash and the rash that developed following the exposure to paracetamol were different, suggesting separate etiologies. More importantly, the attack of angioedema was not an isolated one. The child developed angioedema (day 3) which did not subside through days 4 and 5, when the child continued to receive paracetamol from his general practitioner. The child was admitted to our facility on day 5. A thorough past medical history did not reveal any past history of angioedema. The fact that angioedema subsided as soon as administration of paracetamol was suspended supports our conclusion of a probable adverse reaction. We would like to point out that the initial rash that the child had developed due to a presumed viral infection subsided four days after the angioedema, again pointing to a possible role of paracetamol in this adverse reaction. We agree that a supervised oral challenge test is the definitive means of diagnosing paracetamol-induced angioedema. This test, however, could not be performed as parents refused permission.

Paracetamol is readily available in India in packaged formulations and hence storage at home resulting in increased toxicity and adverse reactions is unlikely though possible. By eliciting a discussion, we seem to have succeeded in our objective of creating and increasing awareness amongst general practitioners about the possibility of adverse reactions with paracetamol, which is known to have an excellent safety record.

TANMAY S. PANCHABHAI, NITHYA J. GOGTAY, SANDEEP B. BAVDEKAR
Departments of Clinical Pharmacology and 1Pediatrics, Seth GS Medical College and KEM Hospital, Mumbai, India
Correspondence:
Dr. Tanmay S Panchabhai,
Department of Clinical Pharmacology,
Seth GS Medical College and KEM Hospital,
Mumbai, India.
E-mail: tspanchabhai@hotmail.com
DOI: 10.4103/0019-5359.48558

REFERENCES