Tertiary hyperparathyroidism presenting as posterior reversible encephalopathy syndrome

A man with hereditary hypophosphatemic rickets presented with seizures and coma. Brain MRI revealed edema typical of posterior reversible encephalopathy syndrome (PRES) and ECG showed ST elevations (figure). Coronary angiography was normal. Blood pressure was 165/90 mm Hg on admission; other known causes of PRES were absent. Severe hypercalcemia (3.83 mmol/L) was detected while parathyroid hormone was highly elevated (1,119 pg/mL). Sonography revealed an enlarged parathyroid gland. Subtotal parathyroidectomy led to prompt normalization of parathyroid hormone and calcium. The patient recovered fully.

Tertiary hyperparathyroidism and hypercalcemic crises can be rare sequelae of long-term substitution treatment in hereditary hypophosphatemic rickets.¹ Severe hypercalcemia has been recognized as a trigger for PRES (and ST elevations), yet the pathophysiologic mechanisms involved are unknown.²

Stoyan Popkirov, MD, Anja Figge, MD, Uwe Schlegel, MD, Sabine Skodda, MD

From University Hospital Knappschaftskrankenhaus Bochum, Ruhr-University Bochum, Germany.

Author contributions: Dr. Popkirov: study concept/design, analysis/interpretation of data, drafting/revising the manuscript for content, including medical writing for content. Dr. Figge: analysis/interpretation of data, drafting/revising the manuscript for content, including medical writing for content. Prof. Dr. Schlegel: analysis/interpretation of data, drafting/revising the manuscript for content, including medical writing for content. Dr. Skodda: analysis/interpretation of data, drafting/revising the manuscript for content, including medical writing for content.

Study funding: No targeted funding reported.

Disclosure: The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

Correspondence to Dr. Popkirov: stoyan.popkirov@kk-bochum.de

References


Tertiary hyperparathyroidism presenting as posterior reversible encephalopathy syndrome

Stoyan Popkirov, Anja Figge, Uwe Schlegel, et al.

Neurology 2016;86;695-696
DOI 10.1212/WNL.0000000000002378

This information is current as of February 15, 2016

Updated Information & Services
including high resolution figures, can be found at:
http://www.neurology.org/content/86/7/695.full.html

References
This article cites 2 articles, 0 of which you can access for free at:
http://www.neurology.org/content/86/7/695.full.html##ref-list-1

Subspecialty Collections
This article, along with others on similar topics, appears in the following collection(s):
Coma
http://www.neurology.org/cgi/collection/coma
Electrolyte
http://www.neurology.org/cgi/collection/electrolyte
Endocrine
http://www.neurology.org/cgi/collection/endocrine
Metabolic disease (inherited)
http://www.neurology.org/cgi/collection/metabolic_disease_inherited

Permissions & Licensing
Information about reproducing this article in parts (figures,tables) or in its entirety can be found online at:
http://www.neurology.org/misc/about.xhtml#permissions

Reprints
Information about ordering reprints can be found online:
http://www.neurology.org/misc/addir.xhtml#reprintsus

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 2016 American Academy of Neurology. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.