Case Report

Recovery from Cogwheel Rigidity and Akinesia and Improvement in Vibration Sense and Olfactory Perception following Removal of an Epoxy-Oleic Acid DNA Adduct

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1. Introduction

A DNA adduct is the covalent binding of a substance to DNA. The effect would depend on the position of the adduct on the genome. At a gene location, expression of the gene may be suppressed. On the promoter region of a gene, overexpression is a possibility.

We present the first case to be published of how removal of an epoxy-fatty acid from the locus of the N-formyl peptide receptors was associated with recovery from cogwheel rigidity and akinesia as well as with improvement in vibration sense and olfactory perception.

2. Case Report

A 51-year-old man presented with a two- to three-month history of episodes of akinesia affecting his hands. It was noted that his employment entailed regular and frequent flights, both long haul and short haul. The history was otherwise unremarkable.

Physical examination revealed cogwheel rigidity in the upper limbs and impaired vibration sense which was absent in the lower limbs, reduced rostrally to the level of the anterior superior iliac spine. He was positive on both the Unterberger test and Romberg’s test. Important negative neurological signs were no evidence of abnormality of gait, no tremor, no nystagmus, and no pain. Clinically, his long-term memory appeared to be excellent. The clinical examination was otherwise unremarkable.

Routine blood tests were insignificant. Investigation for DNA adducts by analysis of genomic DNA from leucocytes by gas-liquid chromatography showed the presence of epoxy-oleic acid, which was localised to the N-formyl peptide receptors (FPRs) using DNA microarrays. Other investigations were carried out to exclude other possible causes of the patient’s symptomatology; these proved negative and included testing for gonorrhoea, hepatitis B core antibodies, hepatitis B surface antigen, hepatitis C antibodies, HTLV-1, IgG and IgM antibodies to syphilis, and antibodies to chlamydia species, as well as the HIV duo...
doctors were blinded to the nature of his treatment and the
also limited by the fact that neither the patient nor his
efficacy of the described treatment for the DNA adducts are
above. The conclusions which may be drawn regarding the
adducts with the neurological symptomology described
limitations. A much larger study needs to be carried out in
cerebral inflammatory processes associated with this form of
Alzheimer cerebral tissue and so may be involved in the
inflammatory cells infiltrating neuritic plaques in postmortem
for FPR-like-1 (FPRL1); FPRL1 is strongly expressed by in-
Epoxy-fatty acids occur naturally in some plant seed oils,
as vernolic acid, considered in this paper is present in
human food uses. The cis-12,13-epoxy-oleic acid, also known as
vernolic acid, considered in this paper is present in the seed oils of Vernonia anthelmintica and Vernonia gal-
amensis. In the latter case, it can account for as much as
60 percent of the total fatty acids. Small amounts of both
hydroxyl and epoxy fatty acids can be generated during long-
term storage of many seed oils, including oils used for
culinary purposes; frying oils and fried foods are the major
source of cis-12,13-epoxy-oleic acid in the human diet [2].
This includes heated olive and sunflower oils [3]. Epoxy-fatty
acids have been shown to be absorbed by healthy women [4].
The means by which an epoxy-fatty acid can adduct to
genomic DNA has been explored in detail [5–7]. It is well
established that DNA adduct formation with some poten-
tially dangerous chemical intermediates can be promoted by
epoxy-group molecules [8].

At the time of writing, three FPRs are known; FPR1,
FPR2, and FPR3 are located in close proximity at 19q13.3.
While most members of the G-protein-coupled FPR family
are now known to be expressed by murine vomeronasal
sensory neurons [9], the above case report suggests that FPRs
may also have a role in early Parkinson’s disease. Indeed, the
amyloid-beta peptide Aβ42 has been shown to be an agonist
for FPR-like-1 (FPRL1); FPRL1 is strongly expressed by in-
flammatory cells infiltrating neuritic plaques in postmortem
Alzheimer cerebral tissue and so may be involved in the
cerebral inflammatory processes associated with this form of
dementia [10].

As this report is based on a single case, there are clearly
limitations. A much larger study needs to be carried out in
order to ascertain the extent of the association of DNA
adducts with the neurological symptomology described
above. The conclusions which may be drawn regarding the
efficacy of the described treatment for the DNA adducts are
also limited by the fact that neither the patient nor his
doctors were blinded to the nature of his treatment and the
fact that there was no control group which received placebo
treatment.

This case highlights the clinical value of checking for
and removing DNA adducts and calls attention to the
need for further research into the possible roles of FPRs in
neurodegenerative diseases.

4. Conclusion

In conclusion, this case report demonstrates that the
presence of DNA adducts should be considered in cases of
neurological presentations involving movement and/or
perceptual disorders when no other causes can be found.
Furthermore, the presence of such DNA adducts need not
be permanent; removal may be associated with clinical
improvement.

Abbreviations

Aβ42: Amyloid-β42
DNA: Deoxyribonucleic acid
FPR: N-formyl peptide receptor
HIV: Human immunodeficiency virus
HTLV-1: Human T-lymphotropic virus 1
IgG: Immunoglobulin G
IgM: Immunoglobulin M
VDRL: Venereal Disease Research Laboratory.

Consent

Written informed consent was obtained from the patient
for publication of this case report.

Conflicts of Interest

Jean A. Monro is Medical Director of Breakspear Medical
Group Ltd., which is a family-owned business, and John
McLaren-Howard is a director of Acumen Medical Ltd.
There are no other conflicts of interest.

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