Innate immunity in the Guillain-Barré syndrome

Published: 21-08-2017
Last updated: 13-04-2024

This protocol is aimed at investigating three research questions related to innate immunity in GBS:1. Do patients with viral GBS have a different innate response to microbial triggers compared to healthy controls?2. Do patients with recurrent GBS...

Ethical review	Approved WMO
Status	Recruiting
Health condition type	Peripheral neuropathies
Study type	Observational invasive

Summary

ID

NL-OMON45725

Source
ToetsingOnline

Brief title
Innate immunity in GBS (iGBS)

Condition

- Peripheral neuropathies

Synonym
acute inflammatory demyelinating polyneuropathy, no lay-term

Research involving
Human

Sponsors and support

Primary sponsor: Erasmus MC, Universitair Medisch Centrum Rotterdam
Source(s) of monetary or material Support: GBS-CIDP Foundation International

Intervention

Keyword: Guillain-Barré syndrome, Innate immunity, Leukocytes, Viruses

Outcome measures

Primary outcome

The primary study parameter is the innate response of leukocytes to microbial triggers as determined by the type I interferon production in response to stimulation of Toll-like receptor 3, 7 or 9.

Secondary outcome

The production of other cytokines and the expression of maturation/activation markers will be determined at the cell surface of leukocytes. Additionally, genetic variants that influence the innate response of leukocytes to microbial triggers will be determined.

Study description

Background summary

The Guillain-Barré syndrome (GBS) is a life-threatening acute polyneuropathy typically preceded by a gastro-intestinal or a respiratory infection. Campylobacter jejuni is the most frequent cause of GBS, but also viruses can precede the development of GBS. The factors that determine the development of this severe post-infectious complication are unclear. Activation of the innate immune system is critical for the development of an antibody response that damages the nerves. Our recent data show that a strong innate immune response was present in former C. jejuni-associated GBS patients. Importantly, this response was correlated with long-term residual disability. We hypothesize that the innate response to microbial triggers is key to the development of post-infectious GBS.

Study objective

This protocol is aimed at investigating three research questions related to innate immunity in GBS:

1. Do patients with viral GBS have a different innate response to microbial triggers compared to healthy controls?
2. Do patients with recurrent GBS have a different innate response to microbial triggers compared to non-recurrent GBS patients?
3. Which genetic variants determine the response to microbial triggers leading to (recurrent) GBS?

Study design

case-control study

Study burden and risks

Subjects will be asked to visit the Outpatient clinic neurology. Blood will be drawn to isolate white blood cells and DNA. Clinical characteristics will be assessed using questionnaires. The visit will take less than 45 minutes and will carry negligible risks.
The identification of host factors that cause GBS will mainly benefit future patients since persons could be identified with a high risk of developing GBS after an infection. For the participants themselves, the study is of less benefit, although possibly, we may be able to identify persons with a high risk of recurrent disease or determine whether family members of patients have an increased risk for GBS.

Contacts

Public
Erasmus MC, Universitair Medisch Centrum Rotterdam

Wytemaweg 80
Rotterdam 3015 CN
NL
Scientific
Erasmus MC, Universitair Medisch Centrum Rotterdam

Wytemaweg 80
Rotterdam 3015 CN
NL

Trial sites

Listed location countries

Netherlands

Eligibility criteria

Age
Adults (18-64 years)
Elderly (65 years and older)

Inclusion criteria

Healthy controls:
- Age 18 years or older
- Written informed consent given by the subject

Former GBS patients:
- Fulfilling the diagnostic criteria for GBS (Asbury, 1990), or its variant Miller Fisher syndrome (MFS).
- Current age 18 years or older
- Written informed consent given by the patient;Recurrent GBS patients:
- Occurrence of at least two episodes of GBS, as determined by the diagnostic criteria for GBS (Asbury, 1990), or its variant Miller Fisher syndrome (MFS).
- Age 18 years
- Written informed consent given by the patient

Exclusion criteria

Healthy controls:
- No serological evidence for exposure to a virus, as determined by IgG serology for EBV, CMV, HEV or influenza antigens.;All groups:
Additional diseases or disorders at time of blood sampling that may influence the endpoints:
- autoimmune diseases (like multiple sclerosis, psoriasis, Crohn*s disease, ulcerative colitis, rheumatoid arthritis, SLE and other systemic diseases)
- acute and chronic infectious diseases (like infectious mononucleosis, HIV/AIDS)
- malignancies (not in remission)
Medicines at time of blood sampling that may affect endpoints (i.e. inflammatory processes):
- NSAIDs, oral corticosteroids, cyclosporine
- Cytostatic compounds
- Cytokines (analogues) and biologicals
- Intravenous immunoglobulins.

Study design

Design

Study type:	Observational invasive
Intervention model:	Other
Allocation:	Non-randomized controlled trial
Masking:	Open (masking not used)
Control:	Active
Primary purpose:	Basic science

Recruitment

NL

Recruitment status:	Recruiting
Start date (anticipated):	01-11-2017
Enrollment:	124
Type:	Actual

Ethics review

Approved WMO Date:	21-08-2017
Application type:	First submission
Review commission:	METC Erasmus MC, Universitair Medisch Centrum Rotterdam (Rotterdam)

Study registrations

Followed up by the following (possibly more current) registration

No registrations found.

Other (possibly less up-to-date) registrations in this register

No registrations found.

In other registers

Register	ID
CCMO	NL59611.078.17