The role of Factor-VII activating protease in the generation of bradykinin in patients with hereditary angioedema

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1) Does FSAP activation in plasma of HAE patients contributes to bradykinin formation? 2) Study the inhibitory efficacy of c1-inhibitor towards kallikrein and FSAP towards bradykinin formation in a endothelial cell-based system. 3) test the efficacy…

Ethical review	Approved WMO
Status	Will not start
Health condition type	Skin and subcutaneous tissue disorders NEC
Study type	Observational invasive

Summary

ID

NL-OMON37687

Source
ToetsingOnline

Brief title
Factor-VII activating protease in hereditary angioedema/HAE study

Condition

- Skin and subcutaneous tissue disorders NEC

Synonym
hereditary angioedema

Research involving
Human

Sponsors and support

Primary sponsor: Academisch Medisch Centrum
Source(s) of monetary or material Support: Ministerie van OC&W,Viropharma

Intervention

Keyword: Bradykinin, FSAP, hereditary angioedema

Outcome measures

Primary outcome

Investigate the role of factor-VII activating protease (FSAP) in the generation

of bradykinin in pateints with hereditary angioedema

Secondary outcome

not applicable

Study description

Background summary

Patients with hereditary angioedema (also known as Quincke edema) are known to have a protein deficiency called C1-esterase inhibitor in their blood. During angioedema which can be caused for example by stress, fever or infection, there is a shortage of this inhibitor and an excess of another protein called bradykinin. This results in a high concentration of bradykinin in the bloodstream. Bradykinin is an important factor in the permeability in the blood vessels. The high concentration of the bradykinin in the blood causes high permeability of water within the blood vessels which results in oedema. Through scientific experiments it has recently been discovered that a protein factor VII activating protease (FSAP) may cause the production of bradykinin within the body. Normally the FSAP is inactive. When there is trauma or inflammation in the body the blood cells die off and FSAP can be activated and produce bradykinin.

Study objective

1) Does FSAP activation in plasma of HAE patients contributes to bradykinin formation? 2) Study the inhibitory efficacy of c1-inhibitor towards kallikrein and FSAP towards bradykinin formation in a endothelial cell-based system. 3) test the efficacy of plasma inhibitors other than c1-inhibitor toward activated FSAP and kallikrein with regard to bradykinin formation in a endothelial cell-based system . These experiments will be performed with plasma of HAE patients (n=40) and of healthy controls (n=40)

Study design

At patients with HAE the concentration of FSAP and bradykinine will be measured in blood. Also we will investigated whether genetic material in patients with HAE have a tendency to produce active forms of FSAP compared to healthy people.

Study burden and risks

one blood sampling (40 ml) through venapunctures and questionnaire. Risks are not to be expected.

Contacts

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Trial sites

Listed location countries

Netherlands

Eligibility criteria

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

Inclusion criteria

Patients suffering from hereditary angioedema Age >/ 18 yr

Exclusion criteria

none

Study design

Design

Study type: Observational invasive

Masking:	Open (masking not used)
Control:	Uncontrolled
Primary purpose:	Other

Recruitment

NL

Recruitment status:	Will not start
Enrollment:	30
Type:	Anticipated

Ethics review

Approved WMO

Date:	26-07-2012
Application type:	First submission
Review commission:	METC Amsterdam UMC

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	NL40131.018.12