Gene expression and eosinophils/IL-31 in bullous versus nonbullous pemphigoid: what makes the blister? - a pilot study

Published: 07-03-2018
Last updated: 11-07-2024

The aim is to investigate the differences in pathogenesis of nonbullous and bullous pemphigoid by comparing gene expression, and the presence of activated and apoptotic eosinophils and IL-31 expression in skin, blood and blister fluid of both…

Ethical review	Approved WMO
Status	Recruitment stopped
Health condition type	Autoimmune disorders
Study type	Observational invasive

Summary

ID

NL-OMON46837

Source
ToetsingOnline

Brief title
Bullous versus nonbullous pemphigoid: what makes the blister?

Condition

- Autoimmune disorders
- Epidermal and dermal conditions

Synonym
bullous pemphigoid, pemphigoid

Research involving
Human

Sponsors and support

Primary sponsor: Dermatologie
Source(s) of monetary or material Support: research fonds van International pemphigus

en pemphigoid foundation (IPPF).

Intervention

Keyword: blister formation, bullous pemphigoid, eosinophils, genexpression, IL31, nonbullous pemphigoid, RNA sequencing

Outcome measures

Primary outcome

1. Differences in gene expression levels in the skin of healthy persons,

nonbullous pemphigoid patients and bullous pemphigoid patients by RNA

sequencing.

2. Differences in the number of activated and apoptotic eosinophils and IL-31

expression in the skin and blood between healthy controls, nonbullous

pemphigoid and bullous pemphigoid by immunofluorescence staining and

flowcytometry .

Secondary outcome

not applicable

Study description

Background summary

Pemphigoid is the most common autoimmune bullous disease and typically presents with severe itch and bullae on the skin (bullous pemphigoid). Pemphigoid most commonly affects the older patients and is associated with an increased risk of mortality, as well as a significant decline in quality of life and psychological well-being. Besides the typical bullous presentation, pemphigoid can also present without blisters, named nonbullous pemphigoid. Nonbullous pemphigoid can be difficult to recognize for doctors and results in a delay of treatment. To date the exact pathogenesis of pemphigoid is still not completely unravelled, and it is unknown what causes the differences in phenotype.

Study objective

The aim is to investigate the differences in pathogenesis of nonbullous and bullous pemphigoid by comparing gene expression, and the presence of activated and apoptotic eosinophils and IL-31 expression in skin, blood and blister fluid of both disease phenotypes.

Study design

Observational study

Study burden and risks

Three skin biopsies of nonbullous pemphigoid and bullous pemphigoid will be obtained by means of a 4mm punch biopsy under local anesthesia. It is a generally safe procedure with minimal burden to the patient. Possible complications of bruising, bleeding, infection and scarring rarely occur. Skin samples of healthy controls will be obtained from left over skin of breast reduction surgery. Furthermore two extra blood samples will be derived from nonbullous and bullous pemphigoid patients during a venapunction for drawn for standardcare purposes. Blood from healthy subjects for control is available in our laboratory. In the patients with bullous lesions we will extract blister fluid by using a seringe. This is a painless procedure and there are no expected complications as blisters are punctured more often in pemphigoid patients for pain relief.

Contacts

Public
Selecteer

Hanzeplein 1
Groningen 9700RB
NL
Scientific
Selecteer

Hanzeplein 1
Groningen 9700RB
NL

Trial sites

Listed location countries

Netherlands

Eligibility criteria

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

Inclusion criteria

1. Written informed consent.
2. >=18 years old.
3. Patients that are recently diagnosed with pemphigoid (bullous or nonbullous) or pemphigoid patients that were in complete remission without therapy and experience a relapse can be included.
[The following diagnostic criteria are used for cutaneous pemphigoid: a positive DIF with linear IgG and/or C3c along the BMZ, and/or positive IIF on SSS or monkey esophagus, in combination with compatible clinical presentation, histopathological findings, or other immunoserological tests.]
If the criteria are fulfilled, patients will be categorized into the nonbullous phenotype (no history and no current blistering on the skin) or the bullous phenotype.
4. Active disease with skin lesions.

Exclusion criteria

1. The use of systemic immunosuppressive medication, such as prednisolone (>0.3mg/kg/day), methotrexate, azathioprine or dapsone (see guideline Feliciani et al)5 within the last 4 weeks before the sample collection. Prednisolone in a dosage <= 0.3 mg/kg/day is allowed.*
2. Application of topical potent corticosteroids on the skin within the last week.
3. Incapacitated (psycho)geriatric patients;* the use of a topical or oral corticosteroid treatment by the general practitioner in patients presenting with an acute pruriginous skin eruption, before the diagnosis of bullous pemphigoid is unfortunately quite frequent. Furthermore, 0.3mg/kg/d of oral prednisone has been previously demonstrated to be ineffective in controlling BP (Guillaume JC et al, 1993) and therefore doses upon to 0.3mg/kg/day are allowed.

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:	Recruitment stopped
Start date (anticipated):	07-01-2019
Enrollment:	10
Type:	Actual

Ethics review

Approved WMO Date:	07-03-2018
Application type:	First submission
Review commission:	METC Universitair Medisch Centrum Groningen (Groningen)
Approved WMO Date:	01-08-2018
Application type:	Amendment
Review commission:	METC Universitair Medisch Centrum Groningen (Groningen)

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	NL63814.042.17