The role of PVAT in vascular ageing in chronic kidney disease and type 2 diabetes.

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PVAT plays an important role in driving vascular ageing manifested by medial smooth muscle cell (SMC) dedifferentiation into an osteogenic phenotype that induces intimal and/or medial calcification. I furthermore hypothesize that this process of...

Ethische beoordeling	Positief advies
Status	Werving gestart
Type aandoening	-
Onderzoekstype	Observationeel onderzoek, zonder invasieve metingen

Samenvatting

ID

NL-OMON23667

Bron
NTR

Verkorte titel
Vascular Ageing Study

Aandoening

Accelerated vascular ageing
Chronic kidney disease
Type 2 diabetes

Ondersteuning

Primaire sponsor: University Medical Center Groningen
Overige ondersteuning: Astellas

Onderzoeksproduct en/of interventie

Uitkomstmaten

Primaire uitkomstmaten

• Characterization of the pro-inflammatory and pro-calcifying environment of PVAT as compared to subcutaneous fat (SAT)
• Identification of potential differences in inflammatory profile between PVAT obtained from ‘healthy’ and calcified arterial wall
• Assessment of the effects on SMC calcification, dedifferentiation and contractile function in vitro of PVAT (compared to SAT)

Toelichting onderzoek

Achtergrond van het onderzoek

Chronic kidney disease (CKD) is associated with a strong increase in cardiovascular risk, which is a consequence of accelerated vascular ageing. This process is hallmarked by vascular remodeling, chronic low-grade inflammation, calcification, and increased vascular stiffness. Vascular ageing is more pronounced in CKD patients who are also suffering from diabetes. The majority of type 2 diabetes (T2D) patients are obese with visceral adipose tissue (VAT) playing a central role in causing insulin resistance and metabolic syndrome. VAT is distributed through the abdominal cavity and is present surrounding the abdominal organs and the vasculature, the latter also called perivascular adipose tissue (PVAT). PVAT may be protective at some sites but it may also promote vascular ageing at other vascular sites because of its pro-atherogenic effects. This deranged function of PVAT may serve as a link between accelerated vascular ageing in CKD and T2D. I hypothesize that CKD and/or T2D derange PVAT function results in aggravated vascular ageing including development of atherosclerosis and calcification. In the current proposal, I will assess the pro-atherogenic environment of PVAT in patients with CKD with or without T2D.

In this study we will assess the role of T2D and CKD (end stage renal disease and pre emptive) in PVAT dysfunction.

Doel van het onderzoek

PVAT plays an important role in driving vascular ageing manifested by medial smooth muscle cell (SMC) dedifferentiation into an osteogenic phenotype that induces intimal and/or medial calcification. I furthermore hypothesize that this process of vascular inflammation and calcification is most severe in patients with both chronic kidney disease and Type 2 diabetes (diabetic nephropathy).

Onderzoeksopzet

T0: informed consent

T1: venapuncture 1 day before transplantation

Onderzoeksproduct en/of interventie

Not applicable.

Contactpersonen

Publiek

Wetenschappelijk

Deelname eisen

Belangrijkste voorwaarden om deel te mogen nemen (Inclusiecriteria)

Kidney donors:

- Men and women

- Age above 17 years

Kidney recipients:

- Men and women

- Age above 17 years

- Kidney failure leading to transplantation

Belangrijkste redenen om niet deel te kunnen nemen (Exclusiecriteria)

- Inadequate spreaking of Dutch language

- Age below 18 years

- Incompetent

Onderzoeksopzet

Opzet

Type:	Observationeel onderzoek, zonder invasieve metingen
Onderzoeksmodel:	Parallel
Toewijzing:	N.v.t. / één studie arm
Blindering:	Enkelblind
Controle:	N.v.t. / onbekend

Deelname

Nederland

Status:	Werving gestart
(Verwachte) startdatum:	01-01-2018
Aantal proefpersonen:	0
Type:	Verwachte startdatum

Ethische beoordeling

Positief advies

Datum:	01-10-2018
Soort:	Eerste indiening

Registraties

Opgevolgd door onderstaande (mogelijk meer actuele) registratie

Geen registraties gevonden.

Andere (mogelijk minder actuele) registraties in dit register

Geen registraties gevonden.

In overige registers

Register	ID
NTR-new	NL7442
NTR-old	NTR7684
Ander register	Research register UMCG : 201500869

Resultaten

Samenvatting resultaten
None yet.