INPLASY PROTOCOL

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None declared.

Secondary IgA Nephropathy without IgA vasculitis: a systematic review of case reports

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Review question / Objective: **To determine the novel putative underlying diseases and to identify etiologic factors of secondary IgA nephropathy**
Condition being studied: **Primary (pIgAN), secondary IgA nephropathy (sIgAN), and IgA-associated nephropathy can be distinguished. Various etiologies may lead to sIgAN. Amongst them are liver, gastrointestinal, oncological, dermatological, autoimmune, and respiratory diseases, as well as iatrogenic, infectious, and environmental factors. The most common sIgAN triggers are liver diseases, in particular liver cirrhosis. Whether secondary forms of the disease share common pathways activated by underlying conditions or separate processes that result in comparable pathologic findings remains to be determined. Recent reports suggest that secondary IgAN shares a similar galactose-deficient IgA1-oriented pathogenesis with primary IgAN, at least for IgA vasculitis. Wang et al. showed that circulating IgA1 and glomerular IgA1 displayed galactose deficiency of O- glycans in patients with secondary IgAN.**
Although both sIgAN and IgA-associated nephropathy are derivatives of preceding disease entities, we pinpoint the difference between the two as long as management differs.
In sIgAN, renal problems are predominant, significantly deteriorating patients' life quality, while in IgA-associated nephropathy, alteration in kidneys is not that severe (oligosymptomatic or asymptomatic).

INPLASY registration number: This protocol was registered with the International Platform of Registered Systematic Review and Meta-Analysis Protocols (INPLASY) on 05 February 2023 and was last updated on 05 February 2023 (registration number INPLASY202320022).

INTRODUCTION

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METHODS

Participant or population: Patients with secondary IgA nephropathy or IgA-associated nephropathy without IgA vasculitis

Intervention: IgAN phenotype (subnephrotic (<3.5g/day)/nephrotic (≥ 3.5g/day) proteinuria/no proteinuria; microscopic/gross hematuria; introduced treatment, response to treatment).

Comparator: Not applicable.

Study designs to be included: Case reports.

Eligibility criteria: Inclusion criteria: studies on sIgAN/ IgA-associated nephropathy concerning humans written in English. The exclusion criteria were: articles regarding primary IgAN, IgA vasculitis and data presumed as insufficient or irrelevant.

Information sources: Electronic databases (MEDLINE, EBSCO, Embase).

Main outcome(s): Gain knowledge about etiology, pathophysiology and management of secondary IgA nephropathy without IgA vasculitis. Measures of effect: response to treatment (remission/improvement/deterioration).

Quality assessment / Risk of bias analysis: PRISMA.

Strategy of data synthesis: Table and flow diagram.

Subgroup analysis: Subgroups will be distinguished based on the underlying disease, analysis of different therapeutic approaches within groups.

Sensitivity analysis: None.

Country(ies) involved: Poland.

Keywords: IgA associated; gastrointestinal; infection; dermatological; cancer; liver; autoimmune; drug-induced; treatment; pathophysiology.

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