

Conclusion

The gallbladder agenesis is certainly a rare entity, but has to be kept in mind in doubtful situations in order to further investigations before surgery. MRC-MRI can confirm the diagnosis and rule out a gallstone disease. Unnecessary surgery and its risks, would be avoided.

References

1. Toufeeq Khan TF, Baqai FU. Agenesis of the gall bladder with duplication cysts of the hepatic flexure - A case report and literature review. Singapore Med J 1991;34:181-82.
2. N. Peloponissios, M. Gillet, R. Cavin, and N. Halkic, "Agenesis of the gallbladder: a dangerously misdiagnosed malformation," World Journal of Gastroenterology 2005;11:6228-31.
3. Valeria Fiaschetti, Giovanna Calabrese, Silvia Viarani and al. Gallbladder agenesis and cystic duct absence in an adult patient diagnosed by magnetic resonance cholangiography: report of a case and review of the literature. Case Reports in Medicine, 2009.

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An exceptional association: ulcerative colitis, amyloidosis and type-2 diabetes mellitus

The association of inflammatory colitis (Crohn's disease and ulcerative colitis) to diabetes let us think of suspected auto-immune disorders, the association to amyloidosis is recognised and this is due to the inflammatory state.

The association of ulcerative colitis, amyloidosis and diabetes was not described until now in the literature.

Islet amyloid has been implicated as a pathological entity in type 2 diabetes since the turn of the century. It has as its unique component the islet beta-cell peptide islet amyloid polypeptide (IAPP), or amylin, which is co-secreted with insulin (1, 2).

The progressive accumulation of IAPP-containing fibrils allows the eventual replacement of beta-cell mass by amyloid and contributes to the development of hyperglycaemia (3, 4).

The aim of this study is to discuss if the association of ulcerative colitis, diabetes and amyloidosis is fortuitous and to focus on the role of IAPP in the occurrence of type-2 diabetes.

Case Report

We report the case of a 49 years old man having an ulcerative colitis diagnosed since the age of thirty years when the patient had diarrhoea and rectal haemorrhage.

The diagnosis was confirmed by the anatomo-pathological exams. The treatment consisted of salazopyrine and then total large bowel resection.

A type-2 diabetes was revealed ten years after. The patient was treated by oral anti-diabetics then insulin.

Six years after, he was hospitalized to explore generalised oedema. The physical examination found orthostatic hypotension and oedema of inferior legs.

He had also nephrotic syndrome and renal insufficiency.

The diagnosis of amyloidosis was confirmed by accessory salivary glands biopsy. The type of amyloidosis was AA.

The patient refused to practice a biopsy of the pancreas.

The outcome of the patient was characterised by disturbance of renal function implicating extra-renal purifying, a high persistent proteinuria, and an activated ulcerative colitis.

After necessary liquid supply, arrest of conversion enzyme inhibitor and treatment by colchicine and salazopyrine; the digestive signs, the renal function and the glycaemia were improved.

Nevertheless, the patient dead in terminal renal stage insufficiency.

The pancreatic biopsy wasn't done in our case. Nevertheless, the literature results permit to us to think about the hypothesis of pancreatic amyloid deposits explaining the type 2 diabetes installation. Then, in our case the association of ulcerative colitis to amyloidosis is classic, but the occurrence of a type 2 diabetes let us think about its pathophysiology.

Conclusion

Beta-Cell dysfunction is an important factor in the development of hyperglycemia of type-2 diabetes mellitus, and pancreatic islet amyloidosis has been postulated to be one of the underappreciated contributors to impaired insulin secretion. The comprehension of the mechanism(s) involved in islet amyloidogenesis allow the development of therapeutic agents that inhibit amyloid fibril formation, with the goal being to preserve beta-cell function and improve glucose control in type 2 diabetes.

References

- 1- Höppener JW, Nieuwenhuis MG, Vroom TM et al. Islet amyloid and diabetes mellitus type 2. Ned Tijdschr Geneesk. 2000; 144:1995-2000.
- 2- Jaikaran ET, Clark A. Islet amyloid and type 2 diabetes: from molecular misfolding to islet pathophysiology. Biochim Biophys Acta. 2001; 1537: 179-203.
- 3- Powell DS, Maksoud H, Chargé SB et al. Apolipoprotein E genotype, islet amyloid deposition and severity of Type 2 diabetes. Diabetes Res Clin Pract. 2003; 60: 105-10.
- 4- Butler AE, Janson J, Soeller WC et al. Increased beta-cell apoptosis prevents adaptive increase in beta-cell mass in mouse model of type 2 diabetes: evidence for role of islet amyloid formation rather than direct action of amyloid. Diabetes. 2003; 52: 2304-14.

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Métastase testiculaire bilatérale d'un adénocarcinome prostatique

Les métastases testiculaires sont rares, ayant le plus souvent pour origine la prostate [1]. Les métastases testiculaires du cancer de la prostate sont caractérisées par leur latence clinique et leur découverte est habituellement fortuite [2,3]. Les atteintes