

HIRSCHSPRUNG'S DISEASE: A LECTIN HISTOCHEMISTRY STUDY

Nogueira, E.V.M.¹; Fittipaldi-Júnior, H.M.²; Teles, N.³; Beltrão, E.I.C.^{1,4*}

¹ Laboratório de Imunopatologia Keizo Asami, LIKA/UFPE; ² Serviço de Anatomia Patológica, IMIP; ³ Departamento de Patologia, CCS/UFPE; ⁴ Departamento de Bioquímica – CCB – Universidade Federal de Pernambuco, Pernambuco, Brazil.

Hirschsprung's disease is a congenital anomaly characterized by partial to complete colonic obstruction associated with the absence of intramural ganglion cells in the distal alimentary tract. The present work aims to evaluate the expression of carbohydrates of aganglionic distal intestine wall using lectin histochemistry. Rectal biopsies and rectosigmoidal resections were sliced (4 µm), treated with trypsin and methanol-H₂O₂ solutions and incubated with lectins conjugated to horseradish peroxidase - HRP (peanut agglutinin, PNA-HRP; *Lotus tetragonolobus* agglutinin, LTA-HRP, concanavalin A, Con A-HRP; *Ulex europaeus* agglutinin, UEA-I-HRP; and *Triticum vulgare* agglutinin, WGA-HRP, 100 µg/mL). Peroxidase was visualized with diaminobenzidine-H₂O₂ solution and slices were counter-stained with haematoxylin and analysed by light microscopy. Lectins binding was inhibited with their respective carbohydrate (glucose for Con A, galactose for PNA, N-acetyl-D-glucosamine for WGA, L-fucose for LTA and UEA-I, 300mM). Con A and WGA recognized residues of glucose and/or mannose and N-acetyl-D-glucosamine in the cytoplasm of gland cells but not your membrane. LTA and UEA-I recognized residues of L-fucose in the membrane of gland cells. PNA failed to recognize residues of galactose in analyzed biopsies. Results indicated that there is a variety of expression of glycoconjugates in aganglionic distal intestine wall in Hirschsprung`s disease.

Supported by CNPq.

Keywords: lectin, histochemistry, Hirschsprung`s disease

