Peran nitric oxide (no) terhadap terjadinya abnormalitas daerah sfingter gastroesophageal junction sebuah kajian tentang pengaruh inflamasi, dan berkurangnya antioksidan endogen dengan tikus sebagai model = The role of nitric oxide (no) in the development of gastroesophageal junction (gej) sphincter abnormalities. a study of the effects of inflammation, and reduction of endogenous antioxidants using rat model.

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Abstrak

Gastro Esophageal Reflux Disease) adalah suatu kondisi terjadinya refluks isi lambung ke dalam esophagus yang menyebabkan berbagai gejala klinis. Penyebab dari GERD sudah banyak diketahui namun patofisiologi densitas saraf pleksus Meissner dan pleksus Aurbach di daerah gastroesofagus junction (GEJ) akibat pemberian senyawa nitrat (NO₃) sehingga menyebabkan GERD belum diketahui. Metode : Membuat model hewan GERD dan menilai variabel-variabel pengaruh senyawa nitrat di daerah GEJ menggunakan tikus wistar usia 10-12 minggu dengan berat badan 200-300 gram yang dibagi dalam 4 kelompok : kontrol (n=12) dan kelompok perlakuan (n=36). Pada kelompok perlakuan dilakukan pemberian senyawa nitrat masing kelompok (n=12) sebanyak 1 ml, 1.5 ml dan 2 ml NaNO₃ . Pada hari ke 2,4,6 dan 8 setelah puasa dan diberikan senyawa nitrat, sebanyak 3 tikus dari setiap kelompok dianalisis menggunakan pemeriksaan biokimia, histologi, histokimia dan imunohistokimia (IHK).
Hasil: Tikus model GERD berhasil dibuat. Dimana pada hari ke 2 terdapat korelasi antara NO luminal dengan fibroblast, NO jaringan dengan perpanjangan lamina propria, penebalan sel basal dengan limfosit, hiperplasi sel basal dengan IHK IL6 dan perpanjangan lamina propria dengan limfosit. Pada hari ke 4 didapat korelasi antara NO luminal dengan penebalan sel basal, NO luminal dengan GSH, penebalan sel basal dengan GSH, dan korelasi limfosit dengan IHK IL6. Pada ke 6 terdapat korelasi antara NO luminal dengan FGF₂. Pada hari ke 8 didapati korelasi antara NO luminal dengan densitas saraf pleksus Meissner dan pleksus Auerbach didapat korelasi kuat dan bermakna (r = 0,758 dan p = 0,004) , penebalan sel basal dengan fibroblas , limfosit dengan fibroblast, IHK IL6 dengan fibroblast dan IHK FGF₂ dengan penebalan sel basal.
Kesimpulan: Pemberian senyawa NO₃ meningkatkan kadar NO luminal yang mengakibatkan perubahan morfologi makrokopis dan mikroskopis, penurunan antioksidan endogen, inflamasi serta peningkatan densitas saraf pleksus Meissner dan pleksus Auerbach didaerah sfingter GEJ sehingga menyebabkan terjadinya GERD.
<hr>Background: GERD (Gastro Esophageal Reflux Disease) is a condition with reflux of gastric contents into the esophagus which causes various clinical symptoms. The causes of GERD have been known but the pathophysiology of the density of the Meissner plexus nerve and the Aurbach plexus in the gastroesofagus junction region (GEJ) due to administration of nitrate (NO₃) compounds is not known unknown.
Methods: GERD animal models were prepared to asses the variables affected by nitrate compounds in the GEJ area using wistar mice aged 10-12 weeks with a weight of 200-300 grams divided into 4 groups:

control (n = 12) and treatment group (n = 36). In the treatment group, nitrate compounds were given as NaNO3 in each group (n = 12) with the doses of 1 ml, 1.5 ml and 2 ml. On days 2,4,6 and 8 after fasting and gavage of nitrates, 3 rats from each group were sacrificed, and esophageal tissue was taken for biochemical, histological, histochemical and immunohistochemical (IHC) examinations.
Results: GERD model rats were successfully made. On day 2, there was a significant correlation between luminal NO level with fibroblasts, tissue NO with extension of lamina propria, thickening of basal cells with lymphocytes, basal cell hyperplasia with IL6 IHC and extension of lamina propria with lymphocytes. On day 4, there was a correlation between luminal NO and basal cell thickening, luminal NO with GSH, basal cell thickening with GSH, and lymphocyte with IL6 IHK. On day 6, we found a significant correlation between luminal NO and FGF2. On day 8, there was a correlation between luminal NO and the density of Meissner plexus nerve and Auerbach plexus with a strong and significant correlation ($r = 0.758$ and $p = 0.004$), thickening of basal cells with fibroblasts, lymphocytes with fibroblasts, IL6 IHC with fibroblasts and FGF2 IHC with thickening of basal cells.
Conclusion: The administration of NO₃ compounds increases luminal NO levels which results in changes in macroscopic and microscopic morphology, decreased endogenous antioxidants, inflammation and increased density of Meissner plexus nerve and Auerbach plexus in the area of the GEJ sphincter leading to development of GERD.