

Endoscopic findings and the prevalence of Helicobacter pylori in Patients with Nephrotic syndrome & Uremia complaining of dyspepsia

These is for partial fulfillment of master degree in internal medicine

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INTRODUCTION

A study suggest that the presence of a specific antigen in the glmeruli of patients with membranous nephropathy and helicobacter pylori infection may be involved in the pathogenesis of membranous nephropathy.

(Nagashima R, ET AL., ۱۹۹۷)

Patients with Nephrotic syndrome & Uremia frequently suffer from dyspeptic complaints such as nausea, vomiting, abdominal distension, early satiety and anorexia.

(Van Vlem B, ET AL., ۲۰۰۱)

Peptic ulcer disease (PUD) occurs in up to one fourth of patients with chronic renal failure.

, ET AL., ۲۰۰۰) Karari EM (

PUD in Chronic renal failure patients seems to have some unique features-namely, lack of pain and higher associations with bleeding, with post-bulbar location, and with multiple ulcers.

(Fallone CA, ET AL., ۲۰۰۱)

Prevalence's of Helicobacter Pylori infection were highest in Haemodialysis patients () and predialysis patients () compared to Peritoneal Dialysis patients ()

, ET AL., ۲۰۰۲) Schoonjans R (

AIM OF WORK

To evaluate the upper gastrointestinal tract endoscopic findings and to determine the prevalence of H. pylori in Patients with Nephrotic syndrome & Uremia with dyspepsia.

Patient & Methods

The study will be conducted on ٣٠ Patients with Nephrotic syndrome & Uremia & ١٠ non Uremic patients as a control Group, all of them complain of dyspeptic symptoms.

-- The ٣٠ Patients with Nephrotic syndrome & Uremia will be divided into three groups, group (A), group (B) & group (C)

-- Group (A) will include ١٠ Nephrotic Syndrome patients & group (B) will include ١٠ **CRF** patients in Predialysis stages & group (C) will include ١٠ **CRF** patients in regular haemodialysis

-- All patients & controls will be subjected for:

١-Full history taking.

٢-Full clinical examination.

٣-Laboratory tests include : S.Creatinine , BUN , Na , K , CBC , ESR , ALT , AST , HBsAg , HCVAb, RBS, Albumin in urine, Pregnancy test for female patients.

٤- ECG & Chest X Ray.

- Abdominal Ultrasound.
- ↯- Upper GIT Endoscope & Biopsies will be taking from Antrum & Bulb.

Exclusion Criteria

- ↯- Pregnancy.
- ↯- Patients with Chronic liver disease.

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List of abbreviations

ACE	Angiotensin-converting enzyme
ANA	Antinuclear antibodies
BMT	bismuth, metronidazole, and tetracycline
CrCl	Creatinine clearance
CRF	Chronic renal failure
CRI	Chronic renal insufficiency
ECL	Enterochromaffin like cells
EGD	Esophagogastroduodenoscopy
ESRD	End-stage renal disease
FSGS	focal and segmental glomerulosclerosis
GBM	glomerular basement membrane
GERD	gastroesophageal reflux disease
GFR	Glomerular filtration rate
HD	hemodialysis
HIV	human immunodeficiency virus
HLOs	Helicobacter-like organisms
HP	Helicobacter pylori
HUS	hemolytic-uremic syndrome
IBS	Irritable bowle syndrome
LAC	lansoprazole, amoxicillin, and clarithromycin
LPS	Lipopolysaccharide
MIG	monokine induced by interferon-gamma
OAC	omeprazole, amoxicillin, and clarithromycin
PTH	parathyroid hormone
ROS	reactive oxygen species
TTP	thrombotic thrombocytopenic purpura
UBT	urea breath test
VCUG	Voiding cystourethrogram

Helicobacter Pylori Infection

Background: In 1982, Warren (a biologist) and Marshall (a clinician) described *Helicobacter pylori* (HP). At first, they named the bacterium *Campylobacter pyloridis*. Later, it was named *Campylobacter pylori* (Ceponis PJ et al., 1995). Since then, a large number of reports have been produced on HP and its pathogenetic potential. In fact, although peptic ulcer disease is the most studied disease related to HP infection, this bacterium is seemingly involved in the pathogenesis of several extra gastric diseases, such as mucosa-associated lymphoid tissue lymphomas (MALTomas), coronaritis, gastro esophageal reflux disease, iron deficiency anemia, skin disease, and rheumatological conditions. However, at present, many of these associations remain largely uncertain, and the debate to confirm or refute causality related to these associations is still open (Demirel A et al., 2000) & (Fallone CA et al., 2000).

The association of chronic HP infection with alterations in gastric mucosal cell proliferation is recognized worldwide. In addition, HP can produce and release several bioactive factors that may directly affect the stomach's parietal cells, which produce hydrochloric acid, and enterochromaffin like (ECL) cells (i.e., G cells and D cells), which produce gastrin and somatostatin, respectively. Evidence suggests that HP inhibits D cells and stimulates G cells. HP has some control mechanisms able to switch the transcription of different genes on or off when needed (Demirel A et al., 2000).

A strong association has been reported between HP infection and gastric lymphoma and adenocarcinoma of the body and antrum of the stomach (Alexander GA et al., 2000). Some cofactors may play a key role in determining such diseases. Currently, whether HP eradication can decrease the risk of cancer remains unknown (Ceponis PJ et al., 1995) & (Fischbach W et al., 2000).