PREVALENCE OF HELICOBACTER PYLORI AMONG HEALTH CARE WORKERS IN ENDOSCOPY UNITS

Thesis
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Medicine

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List of Contents

Title	Page No.
Introduction	1
Aim of the Work	3
Review of Literature	
Chapter (I): Helicobacter pylori	4
Chapter (II): Infection Control Measures	51
Patients and Methods	93
Results	103
Discussion	115
Summary and Conclusion	129
Recommendation	131
References	132
Arabic Summary	

List of Tables

Table No.	Title	Page No.
Table (1):	Descriptive data of the total prevalence H. pylori	
Table (2):	Comparison between the studied groas regard general data	-
Table (3):	Comparison between the studied gro as regard presenting symptoms	
Table (4):	Comparison between the studied gro	_
Table (5):	Comparison between the studied groas regard H-pylori antibodies	
Table (6):	Comparison between the negative a positive H-pylori as regard general damong endoscopic group	ata
Table (7):	Comparison between the negative a positive H-pylori as regard general damong outside group	ata
Table (8):	Comparison between the negative positive H-pylori as regard general damong control group	lata
Table (9):	Comparison between the negative a positive H-pylori as regard clinical damong endoscopic group	ata
Table (10):	Comparison between the negative a positive H-pylori as regard clinical damong outside group	ata
Table (11):	Comparison between the negative a positive H-pylori as regard clinical damong control group	ata

List of Tables (cont...)

Table No.	Title	Page No.
Table (12):	Comparison between the negative positive H-pylori as regard history among endoscopic group	data
Table (13):	Comparison between the negative positive H-pylori as regard history among outside group	data
Table (14):	Comparison between the negative positive H-pylori as regard history among control group	data
Table (15):	The results of infection control measurvey in endoscopy units	

List of Figures

Fig. No.	Title	Page No.
Fig. (1):	Helicobacter pylori	4
Fig. (2):	Management of outbreak of infect agents in manual reprocessing cycles	
Fig. (3):	Management of outbreak of infectious a in automated reprocessing cycles	_
Fig. (4):	Comparison between the studied group regard presenting symptoms	
Fig. (5):	Comparison between the studied group regard history	
Fig. (6):	Comparison between the studied group regard H-pylori antibodies	
Fig. (7):	Comparison between the negative positive H-pylori as regard clinical among endoscopic group	data
Fig. (8):	Comparison between the negative positive H-pylori as regard clinical among control group	data

List of Abbreviation

AER	Automated Endoscopic Reprocessing
ASGE	The American Society for Gastrointestinal Endoscopy
ATD	Autoimmune thyroid disease
BABA	Blood group antigen binding adhesion
BL	Burkitt's lymphoma
BSGE	British Society of Gastroenterology Endoscopy Committee
CAG A	Cytotoxin associated gene A
CLO	Campylobacter-like organism test
CRP	C reactive protien
CSX	Cardiac syndrome X
DM	Diabetes mellitus
EIA	Enzyme immunoassay
ERCP	Endoscopic retrograde cholangiobancreatography
ESGE	European Society of Gastrointestinal Endoscopy
ESGENA	The European Society of Gastroenterology and Endoscopy nurses and associates
FDA	Food and drug administration
GERD	Gastrosoephagaol reflux disease
HBV	Hepatitis B virus
HCW	Health care worker
HCV	Hepatitis C virus
HIV	Human acquired immunodeficiency virus
HLD	High level disinfection
HPSA	Helicobacter pylori stool antigen
H. PYLORI	Helicobacter pylori
IAMM	International Association of Healthcare Central Service Material Management.

IG	Immunoglobulin
IL	Interleukin
INF	Interferon
ITP	Idiopathic thrombocytopenic purpura
LCGS	Liquid chemical germicides
LDL	Low density lipoprotein
MALT	Mucosa associated lymphoid tissue
MEC	Minimal effective concentration
MHC	Major histocompatibility complex
OEM	Original equipment manufacturer
OMPS	Outer membrane proteins
OPA	Ortho-phthalaldehyde
OSHA	The Occupational Safety and Health Administration
PAA	Para acetic acid
PAI	Pathogenecity Island
PCR	Polymerase chain reaction
PHPT	Para hyperparathyroidism
PPE	Personnel protective equipment
PPI	Proton pump inhibitor
RUT	Rapid urease test
SGNA	Society of Gastroenterology Nurses and Associates
SPSS	Statistical program for social science version 12
TMB	Tetramethylbenzidine
TNF	Tumor necrotizing factor
UBT	Urea breath test
USA	United states of America
VAC	Vacuolating toxin A

Introduction

elicobacter pylori, a gram-negative bacterium found on the luminal surface of the gastric epithelium, was first isolated by Warren and Marshall in 1983. It induces chronic inflammation of the underlying mucosa. The infection is usually contracted in the first few years of life and tends to persist indefinitely unless treated. Its prevalence increases with older age and with lower socioeconomic status during childhood and thus varies markedly around the world. The higher prevalence in older age groups is thought to reflect a cohort effect related to poorer living conditions of children in previous decades (McColl, 2010).

It is estimated that 50% of the world's population is infected by H. pylori. Although most infections are not associated with clinical disease, a significant proportion will go to develop some of the commonest problems in gastroenterology: gastritis, peptic ulcer disease, and gastric cancer. Although less than one percent of infected persons will develop gastric cancer, this is the fourth most common malignancy in the world. It is these strong disease associations which establish Н. pylori infection leading as gastroenterological public health problem (Mandeville, 2009).

Several modes of transmission of H. pylori have been described in the literature these include direct contact between subjects which is considered the most common mode,

1

contaminated water sources and food and less commonly iatrogenic transmission (during endoscopies and dental care) (*Salih*, 2009).

The potential for transmission of infection during a gastro intestinal endoscopy is a matter of concern to both physicians and patients. Fortunately, such transmission is very rare (*Banerjee*, 2008).

Some evidence exists that H. pylori infection may be more of a hazard for gastroenterologists and certain categories of nursing staff, but occupational risk factors for transmission remain incompletely elucidated (*Schryver*, 2009).



AIM OF THE WORK

To assess the prevalence of H. pylori infection among health care workers in endoscopy unit and evaluation of Infection control measures in endoscopy units.

Chapter (I)

HELICOBACTER PYLORI

elicobacter pylori (H. pylori) is a curved or S-shaped gram-negative, non-capsulated, non-spore forming bacillus with a length of about 3 µm and a width of about 0.5 µm (figure 1). H. pylori is surrounded with cytoplasmic membrane which in turn is covered by an outer membrane. Both the outer and inner face of the membranes is covered with dot-like structures presumably corresponding to incorporated proteinacious structures. Helicobacter pylori is predominantly an extracellular organism but intracellular penetration can rarely occur (*Navid et al.*, *2010*).

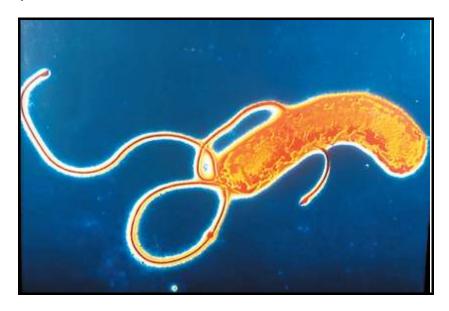


Fig. (1): Helicobacter pylori (Navid et al., 2010).

Helicobacter pylori is oxidase and catalase positive and hippurate negative. It is actively motile by hanging drop preparation with 4 to 6 lophotrichous sheathed flagella which are the most distinguishing structural characteristic of H. pylori. H. pylori is very similar to Campylobacter jejuni but is different in being urease positive while Campylobacter jejuni is urease negative. Campylobacter jejuni is motile with one polar flagellum while H. pylori has multiple polar flagella (*Fangrat et al.*, 2009).

Helicobacter pylori: the bacterium and its virulence factors:

Helicobacter pylori is a Gram-negative spiral-shaped rod which has developed several mechanisms necessary for its survival and replication in the acidic environment of the stomach. H. pylori synthesizes urease, an enzyme catalyzing the hydrolysis of urea to ammonia, which alkalizes low stomach pH and produces a neutral environment around the bacteria (*Newell et al.*, 2007).

It has been shown that urease activity depends on the PH around H. pylori, and it is increased in a lower pH which opens urea channels in the bacterial cell membrane. Urease has a toxic effect on gastric epithelial cells, and it is a potent immunogen which stimulates a strong immune response. Apart from urease and its product, ammonia, several other bacterial products are involved in the gastric tissue damage. Glycosulfatase, a proteolytic enzyme, degrades mucin and phospholipases A2