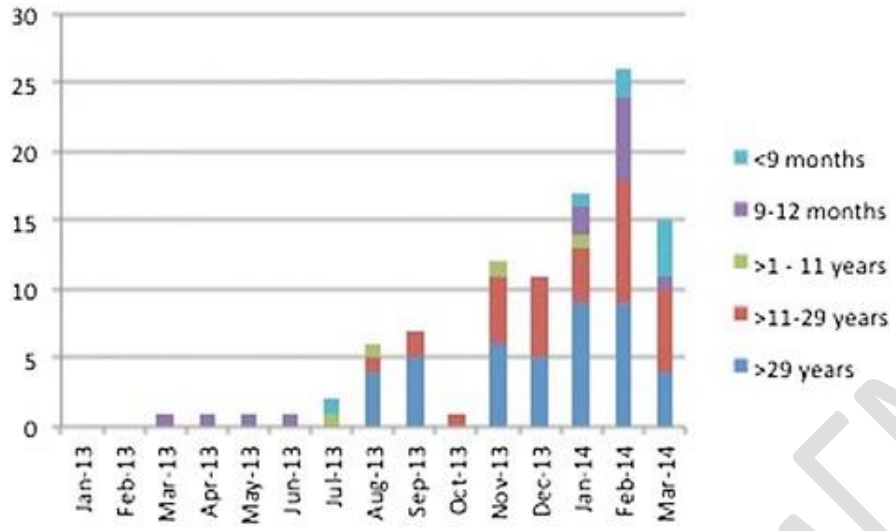

Figure1 — The distribution of suspected measles cases from January 2013 to March 2014, by age group

Conclusion

As mentioned above in the graph infants are the first cases of this outbreak.

Immunization prevents suffering, complications and death caused by measles. The measles vaccine is safe, effective and inexpensive and is one of the most cost effective public health interventions available for preventing deaths. In Sri Lanka measles vaccine is offered to the children through the Expanded Programme of Immunization (EPI) at 9 months of age as the live attenuated measles vaccine and at the age of 3 years as the live attenuated (weakened) measles-rubella (MR) vaccine. Surveillance of Measles is carried out with special investigation forms following the routine notification procedure as for other vaccine preventable diseases.

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PREVALENCE OF *HELICOBACTER PYLORI* IN BENIGN GASTRIC ULCERS IN SRI LANKA AND OTHER COUNTRIES

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Introduction

Peptic ulcers are lesions that develop in the lining of the stomach, they're usually formed as a result of inflammation caused by the bacteria *H. pylori*, as well as from erosion from stomach acids. Evidence suggests that the *H. pylori* remains attached to the cell surface, and does not penetrate into the epithelial layer itself. The

organism is, however, still capable of exerting a damaging effect on the cells. The organism releases cytotoxins into the cells on which it is attached [1].

Production of duodenal ulcers is generally thought to be related to injury to the mucosa, induced by HCl. The adaptive response to an excess of acid in the duodenum is metaplasia. Gastric metaplasia occurs in the duodenum. These areas are now susceptible to infection with *H. pylori*. The infection with *H. pylori* induces further damage to the intestinal mucosa and leads to ulceration. There is a very low yield of organisms from duodenal biopsies [2]. However, >90 % of duodenal ulcer patients also have *H. pylori* associated gastritis. Inflammatory mediators which are released in the stomach, in the response to gastritis, are washed into the duodenum, where they initiate a local response and mucosal damage.

A number of diagnostic tests are available, grouped into two categories: invasive biopsy-based methods, non-invasive methods. Various methods have different sensitivities and specificities.

Some methods are good screening tests whereas others are employed to assess the eradication following treatment.

1. Biopsy based method Culture on selective media; considered as the 'gold standard' method which gives indisputable identification of the organism[3].

2. Rapid Urease test; bedside' test in which a change in pH causes color change in medium is detected by a suitable indicator e.g. Phenol Red. pH is raised due to ammonia production.

3. Gastric biopsy PCR; Very sensitive technique, false positives can occur due to contamination. Non-invasive methods: Serology; detection of antibodies to *H. pylori* useful in epidemiological studies but not suitable for eradication confirmation, indicates past as well as current infection.

4. ICT (Immunochromatography).

5. ELISA.

6. Urea breath test: patient is fed C13 or C14 labelled urea. Urease activity generates labelled CO_2 which is expired and detected by scintillography after collection of expired air in a respiratory chamber.

7. Stool culture; can be technically difficult but can provide antibiotic sensitivity information. Stool EIA; again, prone to technical sampling errors [4].

8. 'Non-invasive' PCR; allows amplification of bacterial DNA to allow identification in Dental plaque, Saliva sample, Stool sample.

Goal

To study about the Prevalence of *Helicobacter pylori* in benign gastric ulcers. To know the causes, statistic and prevention.

Materials and Methods of research

The analysis and generalization of modern medical scientific literature on this topic.

Research results and discussion

Helicobacter pylori is a spiral bacterium with several flagella at one end. First recognized as a cause of G.I.T diseases and successfully cultured in 1984. Originally classified as *Campylobacter pylori*. It is a microaerophilic organism, with a potent urease activity. Barry Marshall and Robin Warren; they proved to the medical community that the bacterium *Helicobacter pylori* (H. pylori) is the cause of most peptic ulcers. Microaerophilic organism is one which can readily grow in minimal O2 environments. Urease activity is an enzyme which is capable of hydrolyzing urea to form ammonia. The resultant ammonia causes an increase in the immediate surrounding environment's pH. This urease activity then affords the organism protection from the potentially damaging gastric acid. Ammonia generated by the activity is directly toxic to mucosal cells. To colonize the stomach H^+. pylori must survive the acidic pH of the gastric. Urease production and motility are essential for this step. *H. pylori* uses its flagella to reach gastric niche. Flagella will allow the *H. pylori*

to make hole into the mucus and close to the stomachs epithelial cell layer. Urease enzyme secreted by the bacterium hydrolyzes urea into carbon dioxide and ammonia thereby neutralizing the acidity in the bacterial micro environment, permitting H. pylori to survive in an acidic environment. $(NH_2)2CO + H_2O$ urease $CO_2 + 2NH_3$. *H. pylori* can bind tightly to epithelial cells by multiple bacterial-surface components. It is thought that around 50 % of the world's population is infected with this organism. Fortunately, most individuals infected show no symptoms or adverse effects of the infection. Outcome of the infection seems to be dependent on multiple factors ~ both host and bacterial.

Several studies have been carried out in Sri Lanka to assess *H. pylori* infection to elucidate [5]. Its role in gastritis using different detection techniques. In Sri Lanka, varying prevalence rates of *H. pylori* infection have been reported in different studies using different *H. pylori* testing methods ranging from 2.9 to 70.1 %. Generally, infection with *H. pylori* is long standing. Individuals being infected for several decades. Epidemiological studies have indicated that the vast majority of cases of gastritis are accompanied by infection with *H. pylori*. In the U.K and U.S.A the infection is most common in adults, with the incidence with age; around 5–20 % below age of 20 years. Around 30–60 % over the age of 50 years. In the African and Asian continents rates of infection are generally much higher, between 60–90 % of the population. In Africa and Asia, the individuals generally become infected at an earlier age. Generally, the poorer the population the higher the incidence, and the younger the age of onset. The likelihood of new infection is greatly reduced due to improved hygiene and the development of effective new antibiotics [6].

Conclusion

H. pylori prevalence with all test methods used were consistently low, indicating truly low prevalence of the disease in the study population. Low prevalence of serum anti- *H. pylori* IgG levels suggest low exposure rate of the study sample to the *H. pylori* infection. Duodenal ulcers do not occur without gastric metaplasia until the metaplasia is reversed, the patient is vulnerable to re-infection. No clear relationship exists between H. pylori infection and smoking or alcohol intakes. There are no clear specific dietary links. Vegetarians are equally susceptible to infection.

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