Helicobacter pylori: the African enigma

Data on Helicobacter pylori infection in Africa are at odds in several aspects with those published in the west. Gastric H pylori infection is common, almost ubiquitous in Africa, but the pattern of infection, age of acquisition, environmental, dietary, and genetic influences are different from those in the west. These differences alter the pathological role and clinical relevance of the organism in Africa where, apart from gastritis, there is no established correlation between H pylori infection and upper gastrointestinal disease.

Epidemiology

The measurement of circulating antibodies to H pylori using an enzyme linked immunosorbant assay provides a simple, reliable, and non-invasive means of diagnosing H pylori infection, with a sensitivity of 95% and specificity of 85%. This technique is adaptable, can be used in large numbers of subjects, and has been used to map the epidemiology of H pylori infection worldwide. Random serological studies have shown that most of the population in Africa are infected by H pylori for most of their lives – 70–80% have antibodies to H pylori. Not only is infection common but it is acquired at an early age. Fifty five per cent of the population under age 10 years have immunoglobulin G (IgG) antibodies to H pylori in the Ivory Coast, while half of children aged under 5 have IgG antibodies to H pylori in northern Nigeria and the Gambia. These figures derived from serological studies have been confirmed by culture and histological examination of gastric mucosal biopsy specimens. Seventy to 97% of patients with dyspepsia are infected by H pylori, as are 80% of asymptomatic volunteers.

With such a high prevalence of infection, a high prevalence of those diseases said to be associated with H pylori infection would be expected.

Gastritis

Gastritis is very common throughout Africa and shows a strong correlation with H pylori infection. Indeed, as in the west, type B antral gastritis is a good indicator of H pylori infection. In endoscopic studies, histological gastritis is present in 80–100% of subjects with dyspepsia and was found in 88% of a group of 40 asymptomatic controls.

Gastric Ulceration

Gastric ulcer is a rare disease in Africa, occurring six to 30 times less commonly than duodenal ulcer. In northern Nigeria duodenal ulcer is uncommon, yet gastric ulcer is six times less common. In Ethiopia, Tsega found only 5 gastric ulcers in 1084 patients undergoing gastroscopy, compared with 154 duodenal ulcers. Yet gastritis, which is believed to be fundamental to the cause of gastric ulcer, is present in 90% of Africans, and H pylori, which is associated with gastric ulcer in the west, is present in over 80% of Africans.

Gastric Cancer

Because H pylori causes gastritis it has been suggested as a precursor of gastric cancer. Forman et al have documented an increased incidence of gastric cancer with increased prevalence of H pylori infection in China, and in a case control study in Wales an odds ratio of 2.8 has been calculated for the risk of gastric cancer in those infected by H pylori. Similar figures have also been recorded in the USA.

In Nigeria H pylori gastritis is common, yet gastric cancer is uncommon. In the absence of accurate population statistics the most useful indicator of tumour incidence is the proportional frequency of one tumour compared with that of all other tumours. Using this measure gastric cancer accounts for less than 2% of all malignant tumours in northern Nigeria and only 2–3% of malignancies in the Sudan, Uganda, and Zimbabwe.
Pathological role of H pylori

The mechanism by which H pylori exerts its pathological effect is unknown. Wyatt and Dixon suggest that it leads to ulceration only in the presence of gastric metaplasia in the duodenum,15 and this is increased in hyperacidity.16 Studies from Africa have shown lower basal and maximal acid outputs in asymptomatic controls17,18 compared with the UK.19 It is reasonable to suggest that there is less acid, less gastric metaplasia, less duodenal H pylori infection, and therefore fewer duodenal ulcers. There is certainly a need to document the prevalence of duodenal gastric metaplasia in Africa and to correlate this with the prevalence of duodenal ulcer. If there is a good correlation this will support the suggestion that it is a combination of gastric metaplasia in the duodenum and H pylori infection which is fundamental to the pathological role of H pylori in duodenal ulcer. But why do Africans infected by H pylori produce less gastric acid when western subjects with H pylori infection have been shown to have increased concentrations of gastric acid?20

A low prevalence of gastric metaplasia does not account for the low prevalence of gastric ulcer,21-23 which requires neither high acid output nor gastric metaplasia in the duodenum. Graham has suggested that H pylori infection acquired at a young age is less pathogenic than that acquired as an adult, a situation analogous to paralytic poliomyelitis which is more likely to occur if infection is acquired after infancy.24 With most infection in Africa acquired in early childhood, this may be important.

Different strains of H pylori may have different pathogenicity. It has been suggested that organisms showing a gastric IgA response to the 120 kDa protein are more likely to cause peptic ulcers.25 It would be of interest to map the strain type in Africa and to correlate this with the incidence of peptic ulcer. This will become practical if a systemic marker of the H pylori pathogenicity is identified.

Environmental and social factors may combine to protect patients from the harmful effects of H pylori. Of these, diet may play an important protective role. In India, areas of high and low incidence for duodenal ulcer have been identified and clearly correlated with diet.26 A pulse, lentil, okra, and millet diet is protective, while a refined rice diet is not.27 The staple diet in northern Nigeria is millet with large amounts of okra (personal observation) which may well, as in India, help to prevent duodenal ulcer. Other environmental and social factors are also different, for instance, few subjects smoke or take non-steroidal anti-inflammatory drugs in northern Nigeria.28

Gastric cancer is a tumour of old age. The life expectancy in Nigeria is only 55 and in the west gastric cancer is uncommon below this age. However, if an H pylori induced gastritis is an important initial step in some, it is the age at which the gastritis first occurs which may be important. H pylori infection and gastritis occurs 20–30 years earlier in Africa than in the west,29 but this has not led to an increase in gastric cancer in early middle age. The peak incidence for gastric cancer in South Africa is in the seventh decade.30 The incidence of gastric cancer is thought to be higher in patients with intestinal metaplasia,31,32 and it has been suggested that intestinal metaplasia is the result of long term gastric inflammation possibly secondary to H pylori infection.33 Of interest then is the low prevalence of intestinal metaplasia in Africa. In H pylori positive patients in the west, 17 to 30% show intestinal metaplasia in gastric biopsy specimens.34 In a series of 157 patients with non-ulcer dyspepsia and 40 asymptomatic controls in Nigeria, intestinal metaplasia was present in only 2% (personal observation) and an observation noted in reports from other African countries.11-13 It is likely that the environmental or genetic factors, or both, which cause the change from gastritis to intestinal metaplasia and subsequently dysplasia and cancer are not present.

Conclusion

Geographical variations in the incidence of peptic ulceration and gastric cancer have been apparent for many years, but in Africa these are not reflected by variations in the prevalence of H pylori infection. Indeed, with the exception of gastritis, the case for a pathological role for H pylori in Africa is unproved.

Above all else, the data from Africa underline the multifactorial nature of the cause of peptic ulcer and gastric cancer. H pylori exerts its influence in concert with other environmental, social, and genetic factors. The relative importance of these and how they interact in a given community or individual remain to be elucidated.

C HOLCOMBE

Department of Surgery, Charing Cross Hospital, Fulham Palace Road, London W6

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