Malabsorption Syndromes

Symposium held at the Second World Congress of Gastroenterology, Munich, May 19th, 1962

Edited by H. Schön, Erlangen

1963
S. Karger Basel (Switzerland) New York

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Chairman: A. C. FRAZER, Birmingham Secretary: H. SCHÖN, Erlangen

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With 19 figures and 14 tables

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BASEL (Switzerland) S. KARGER NEW YORK

Distributed in North America by Hafner Publishing Company, Inc., New York, N. Y.

2nd World Congress of Gastroenterology, Munich 1962

Praeses: N. HENNING (Erlangen); Curator: E. SCHMID (Erlangen).

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Additional publication:

Malabsorption Syndromes. Symposium held at the Second World Congress of Gastroenterology, Munich 1962. Editor: H. SCHÖN (Erlangen).

Verlag S. Karger AG, Arnold-Böcklin-Straße 25, Basel (Switzerland)

Printed in Switzerland by Buchdruckerei Friedrich Reinhardt AG, Basel
Clichés: Aberegg-Steiner, Bern

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Agastric Dystrophy

G. Berg, Erlangen

Gastric resection represents an important cause of disturbances in digestion and absorption. In some cases, even in spite of comprehensive and careful therapeutic measures, the clinical picture of dystrophy develops like that resulting from malnutrition. Henning (1) proposed the term "agastric dystrophy" for these cases because it contains cause and effect. This pathological aspect can also be classified under the general term of malabsorption syndrome in which many different causes lead to the same effect. Following the classification of Frazer (2), intraluminary, intracellular and extracellular causes of malabsorption are demonstrable, so that agastric dystrophy may serve as a model example of a malabsorption syndrome.

Among the manifold symptoms first diarrhea, weight loss and edema stand out, usually occurring after a latent period of several months up to one year (Lambling) (3). A thorough examination however, reveals still more signs of dystrophy. The subcutaneous fat tissue is lacking, and the muscles are atrophic. The skin is thin, smooth, and shiny. Pinpoint hemorrhages, as well as a positive Rumpel-Leede, are suggestive of capillary damage. The very important pruritus is remarkable, demonstrated even by scratch marks. Furthermore, there are transverse fissures in the corners of the mouth, there is glossitis, gingivitis and atrophy of the tongue. Signs of endocrine disorders are loss of hair, a decreased growth of the beard, the development of feminine pubic hair distribution in males, small testicles and impotence. The 17-ketosteroid excretion in the urine is decreased.

X-ray examination in such cases mostly reveals a precipitous emptying, in our experience the total passage period is shortened. Histological examination of the gastric mucous membrane reveals an atrophic gastritis, with some signs of metaplasia of the goblet cells; biopsy of the small intestine often shows inflammatory changes as well as atrophy of the mucous membrane (1). The activity of the

pancreatic enzymes is decreased (4). The digestion of fats, measured by microscopic stool examination after SCHMIDT's test diet, is diminished. Calorimetry of the stools shows high values (5). The absorption of iron and fat (I^{131}-labelled Triolein) is decreased (6). Usually anemia is present. Vitamin deficiency and electrolyte balance disorders are observed. There is hypoproteinemia due to a hypo-albuminemia, to be considered partly as the expression of an exudative enteropathy. In some cases we found an increased excretion of I^{131}-labelled polyvinylpyrrolidone as a sign of an albumin excretion disorder. The color change of the scalp hair, first observed by us, in such cases is to be considered as a consequence of the protein deficiency (1). Pathological liver function tests and liver biopsies reveal liver parenchymal damage, in a remarkably high percentage (7).

Among the causes leading to these more or less clear disorders of digestion and absorption, gastric resection according to Billroth II takes first place. In many cases a rapid emptying of the stomach due to the particular anatomical conditions can be observed. The contact of food with the mucous membrane and the digestive enzymes is short. The absence of the antrum and the functional exclusion of the upper duodenum lead to a diminution of the pancreatic function and to an insufficient mixing of the food with bile. Due to the rapid passage, the food at the time of digestion is already in the lower sections of the bowels, where no more absorption worth mentioning takes place. Another cause of the disturbed absorption are the morphologically demonstrable changes of the gastrointestinal tract. The atrophic gastritis (8, 9) of the resected stomach is a cause of constant loss of albumin. This loss of protein in the presence of insufficient food utilization can finally no longer be compensated, leading to hypo-albuminemia and protein deficiency. We consider the pathological changes of the small intestine to be the substrate of malabsorption, as we could prove mainly with I^{131}-labelled Triolein.

Summing up the causes of malabsorption in agastric dystrophy the anatomical conditions after gastric resection, the functional exclusion of the lower part of the duodenum, the rapid passage, the morphological changes of the mucous membrane of the gastrointestinal tract and sometimes also parenchymal liver damage are demonstrable.

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Discussion

O. J. TEN THIJE (Groningen): You mentioned the partial atrophy of the small intestinal villi as a cause of this agastric dystrophy. Are you sure that this represents a cause and not a consequence?

R. AMMANN (Zürich): The examination of the mucous membrane of the small intestine by use of the Crosby capsule in 18 non-selected patients with partial gastric resection showed no correlation between morphological changes of the mucous membrane (inflammatory infiltrations, fusions of the villi, atrophy of the mucous membrane) and the clinical symptomatology. 3 of the 18 patients revealed clinically slight to moderate steatorrhea, 2 of these patients showed mucous membrane changes, in a third patient the histological findings were normal. Of the 15 patients without steatorrhea 8 revealed morphological changes of the mucous membrane of the same kind. It seems that the mucous membrane changes in the jejunum occur relatively frequently after gastric resection, and probably are, like postoperative gastritis, a coincidental finding. The histological changes of the mucous membrane are also distinctly patchy and less definite than in idiopathic spure. The pre-existence of a latent non-tropical sprue appears to be rather unlikely in our cases because of past history, clinical and morphological findings.

G. BERG (Erlangen): Final conclusions about the frequency and significance of morphological findings of the small intestine may only be possible in the presence of a larger examination material. In our material the frequent pathological findings in patients with malabsorption syndromes were striking.

Malabsorption Syndromes, Symp. 2nd World Congr. Gastroenterol., Munich 1962, pp. 4–7 (Karger, Basel/New York 1963)

The Epidemiology of Tropical Sprue

A Preliminary Report of an Epidemic of Tropical Sprue in North and South Arcot Districts, Madras State, S. India, 1961–1962

S. J. Baker, V. I. Mathan and I. Joseph, Vellore, South India

Tropical Sprue is a name which means different things to different people. It may perhaps best be defined as a primary malabsorption syndrome occurring among people resident in the tropics. As such it is usually associated with steatorrhea, and other evidence of small intestinal disfunction such as defective absorption of glucose, xylose, vitamin B_{12}, folic acid and other nutrients, together with radiological changes in the small intestine. It is important to realise that tropical sprue so defined is probably a syndrome of multiple aetiology, and not a single disease entity. In some parts of the tropics primary malabsorption may be due to gluten induced enteropathy but in South India the local population do not eat wheat or wheat products and therefore this possibility can be excluded. At least two other groups may be distinguished—what we have called "endemic sprue" which occurs sporadically and which has been present in South India for many years, and is of unknown aetiology. The other group may be defined as "epidemic sprue" which may well be caused by an infective agent. Whether or not epidemic and endemic sprue are the same or different diseases has yet to be demonstrated.

Epidemics of tropical sprue have occurred in India in the past. Stefanini (1948) reported one among Italian prisoners of war and Walters (1947) and Avery (1948) in British and Indian troups in North India and Burma during the 2nd world war. In Madras State, South India, there have been at least 5 epidemics of chronic diarrhoea of unknown aetiology occurring in the past 30 years. There is evidence to suggest that these epidemics resembled tropical sprue but they were not recognized as such at the time of their occurrence.

We have recently had the opportunity to study a large epidemic of tropical sprue which occurred in North and South Arcot districts

of Madras State beginning in September 1960. The region affected is South West of Madras City and covers an area of approximately 4000 square miles being about 40 miles from east to west and 100 miles from north to south. The epidemic area is bordered on the west by a range of hills, but on the northern, southern and eastern borders there are no particular geographical features limiting the spread of the epidemic.

The area receives maximum rain from the north east monsoon beginning in September and lasting to early December. In 1960 the rainfall during this period was exceptionally high. The tanks filled and the rivers became swollen and there was a large amount of surface water lying on the ground.

There are no big towns in the area, the vast majority of people are agricultural labourers living in small scattered villages ranging in population from 200 to 5000. The main crops grown are rice, sugar cane, several varities of grains, groundnuts and some vegetables. The majority of families live in mud huts with thatched roofs, ventilation is poor, the floor is of mud and in the rainy season is usually damp. There is no separate kitchen, the cooking being done in one corner of the house and food is not protected from flies or rats. The surroundings of the houses are frequently dirty and there is no attempt at sanitation.

The staple articles of diet are rice and ragi. Dhal may be used once a day, meat or fish very occasionally and milk and milk products hardly ever. Cooking oil is made from locally grown ground-nuts, either made in the house, or purchased from the local store.

A typical village in this region is that of Padiri where cases of diarrhoea first appeared in early October 1960. Fresh cases continued to occur throughout November and December of 1960 and during January and February of 1961. Among the total population of 361, there were 144 individuals affected and 29 deaths. The time of onset among individuals in one house usually varied; rarely a number of people developed symptoms at the same time, but more usually there was a period varying from about 5 days to 2 months between the time of onset of different cases. It is noteworthy that some houses were exceptionally heavily affected while others were completely free. The highest number of cases occurred among the under 10 age group with another peak in the 30 to 40 age group. However when the incidence is analysed on a percentage basis the incidence increased with increasing age.

An analysis of 921 cases and several thousand unaffected individuals in the same area shows that there is no relation between wealth, diet, food habits, and the presence or absence of intestinal parasites and the incidence of the disease.

The aetiology of tropical sprue is as yet unknown. There are however 3 main theories; (1) that it is caused by a food toxin; (2) that it is a dietary deficiency, and (3) that it is an infective agent. These studies have shown no evidence that the disease is caused by a food toxin or dietary deficiency, since there was no difference between the food eaten by the people who got the disease and those who did not get it, likewise there was no apparent difference between the diet eaten by the affected and unaffected individuals—both were equally poorly nourished. The very wide spread nature of this epidemic; the rapid rate in which it spread over the effected area; the fact that 10% of patients complained of fever at the onset of diarrhoea; and the nature of the spread within the village, all suggest that it may be caused by an infective agent. Cultures of stools specimens and rectal swabs have failed to reveal the presence of any bacterial pathogens. It is therefore thought that this epidemic was caused by an infective agent possibly an entero-virus. Further work is in progress on this problem.

ACKNOWLEDGEMENTS

This study was made possible by a generous financial grant from the Wellcome Trust. We also wish to thank the Director of Medical Services and the Director of Public Health, Madras State, for their help.

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Discussion

J. S. LOGAN (Belfast): The speaker should be congratulated on drawing attention to the occurrence of epidemics of tropical sprue. A very severe epidemic of sprue occurred among Indian troops in the Imphal Plain in the course of the operations in 1944. A remarkable feature was that vegetarian troops (who had not received their usual supplement of milk and milk products) were predominant-

ly affected. Indian troops who had received a little goat meat each week seemed to be protected and were able to remain on duty. The nutritional state of the population involved, therefore, seems important.

A. C. FRAZER (Birmingham): a) What was the fat intake of the patients studied in the sprue epidemic? If it was very low, this might affect the incidence of steatorrhoea. – b) Was there any difference in incidence of sprue in the different socio-economic groups?

K. LACHENMAIR (Munich): Did your surviving patients later on show symptoms of a permanent intestinal damage?

S. J. BAKER (Vellore) to J. S. LOGAN: In this epidemic both vegetarians and non-vegetarians were affected. Vegetarians have lower reserves of vitamin B 12 and are therefore more likely to become B 12 deficient more quickly than non-vegetarians. We were not however able to show any difference between the incidence among different people according to their diet. In fact in some cases those living on a poorer diet had a lower incidence of the disease.

Reply to A. C. FRAZER: (a) The incidence of steatorrhoea will undoubtedly vary with the fat intake at the time the test is undertaken. In our field study fat balances were carried out with the patients on their ordinary diet which probably contained at most 20–30 grams of fat. Therefore the observed incidence of steatorrhoea is probably rather lower than the true incidence.

(b) There were a number of differences in the incidence among different socio-economic groups. All these have not yet been analysed but at least in some villages the poorer community, the Harijans, who live on an extremely poor diet, had a lower incidence of the disease.

Reply to K. LACHENMAIR: The majority of patients who survived appeared to have recovered completely but there is a small proportion who at the present time are still suffering from the disease. Since there is wide spread evidence of intestinal damage in the general population even among those who do not have sprue (e.g. in biopsy findings) it is impossible to assess the late effects of this epidemic on intestinal function.

Malabsorption Syndromes, Symp. 2nd World Congr. Gastroenterol., Munich 1962, pp. 8–10 (Karger, Basel/New York 1963)

Temperate Sprue

W. T. Cooke, E. V. Cox, C. T. Jones and M. J. Meynell, Birmingham

The diagnosis of idiopathic steatorrhoea has rested upon the demonstration of steatorrhoea, haematological abnormalities and changes on radiological examination of the small intestine in the absence of any demonstrable pathological abnormality (Cooke, Peeney and Hawkins, 1953). This diagnosis may cover more than one aetiological disorder and recently, changes have been demonstrated in the jejunum of many patients with idiopathic steatorrhoea.

In a preliminary report on our findings concerning the jejunal biopsies in such patients, Fone, Cooke, Meynell, Harris and Cox (1960) concluded that there were at least two groups of patients amongst those previously considered as suffering from idiopathic steatorrhoea. An extension of these observations offers further support for this hypothesis. The main group of 48 patients we have termed adult coeliac disease since approximately 50% have a history dating back to childhood and a characteristic jejunal biopsy when untreated with a gluten free diet. The biopsy is characterised by prolongation of the crypts, little evidence of the normal change from basophilia to acidophilic staining, abnormal surface epithelial cells and abnormal brush borders, the whole giving a flattened appearance with absent villi though the thickness of the mucosa is not necessarily decreased. Review of the clinical features emphasised certain points usually attributed to idiopathic steatorrhoea. There is usually a long history of variable degrees of ill health, of recurrent or continuous loose stools, a mild anaemia, rarely severe megaloblastic anaemia (only 2 examples), disturbances of calcium and bone metabolism are not uncommon and there is a history of childhood intestinal disturbance in 50% patients.

In the second group of 12 patients, the jejunal biopsies (the majority taken prior to treatment), appeared to be entirely different from those of adult coeliac disease. Villi are present, often larger than normal, sometimes swollen and sometimes appearing almost normal but

with none of the flattened appearance of the coeliacs. The clinical features of this group were strikingly different in that the history was short, the longest being six months, they all had severe megaloblastic anaemia and rapidly remitted with a small dose of folic acid or citrovorum factor.

Though there are thus clinical and histological reasons for regarding these patients as belonging to two different groups, we wish to present further evidence that supports this hypothesis based on laboratory findings. In considering the significance of these findings, it should be realised that folic acid, glucose, iron and xylose are preferentially absorbed from the upper jejunum whilst vitamin B_{12} is predominantly absorbed from the lower ileum. Fat is absorbed from the whole length of the intestinal tract which is able to absorb large quantities of dietary fat and also suffer short lengths to be put out of action without causing steatorrhoea. These substances cover the commonly used absorption tests and therefore it might be expected that any one area affected by different pathological processes gave essentially similar absorptive tests. This was not so. In the following slides there were shown the main findings and significant differences between the two groups—*haematology*, *glucose tolerance*, *folic acid excretion and folic acid absorption tests*, gastric HCl, $Co^{58}B_{12}$ absorption and fat excretion.

Therefore, we believe that on clinical, histological and other laboratory findings we are justified in considering these two groups as being of different aetiology, even though we do not know the aetiology of either. If so, the problem of nomenclature must be considered. We believe that it is wrong to continue to apply the diagnosis of idiopathic steatorrhoea to all these patients indiscriminately and it is important for the development of our knowledge that this broad category should be broken down into further clinical entities. Therefore, we have applied the term adult coeliac disease to the group with flat biopsies and for the purposes of this communication have called the small group with the short clinical history and a folic acid deficient megaloblastic anaemia, "temperate" sprue believing that they bear a closer relationship to tropical sprue than to coeliac disease. We recognise that there are many other terms which might be preferable.

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Discussion

I. W. DELAMORE (Edinburgh): In a series of approximately 100 cases we, in Edinburgh, have been unable to find any correlation between mucosal appearances and absorptive defects; both kinds of change having been found in undoubted coeliac disease. I wonder if Dr. COOKE has any explanation for this finding?

A. C. FRAZER (Birmingham): In our experience the differences described by Dr. COOKE occur successively in our patients with gluten-induced enteropathy. I would like to raise one particular point about the slides Dr. COOKE has just shown us. It seemed to me that the nuclei of the group in which folic acid deficiency was said to be a major feature looked remarkably normal. In folic acid deficiency, changes in the small intestinal cells are commonly observed. I should be glad if Dr. COOKE would comment on this point.

W. T. COOKE (Birmingham): (1) I am suprised for in the paper by GIRDWOOD, DELAMORE and WILLIAMS (1961) there appeared to be at least three patients comparable to these.

(2) Dr. FRAZER has perhaps missed the point. These laboratory and biopsy findings are not encountered in untreated coeliac disease, nor does severe megaloblastic anaemia occur in a treated coeliac at a time when the laboratory findings and biopsy findings have improved and are comparable to the observations we are reporting. Folic acid deficiency in the experimental animal and the changes in certain human diseases is produced by the administration of antifolic metabolites. Neither of these conditions are comparable with the type of folic acid deficiency arising "spontaneously" in the patients I have described. The nuclei are not completely normal and in smears it can be shown that many of the nuclei are much longer than normal and in no way comparable to those seen in the surface epithelium of untreated adult coeliac disease.

Malabsorption Syndromes, Symp. 2nd World Congr. Gastroenterol., Munich 1962, pp. 11–16 (Karger, Basel/New York 1963)

An Evaluation of Secondary Malabsorption Syndromes of Intestinal Origin

H. H. SCUDAMORE, Rochester, Minnesota

A malabsorption syndrome may occur in any disorder that impairs absorption from the small intestine. The term is better used to designate a category of similar diseases rather than a single entity such as nontropical sprue.

A classification based on the site and mechanism of absorptive defects has been given previously (SCUDAMORE, 1961). The origin may be intestinal, gastric, pancreatic, or hepatobiliary. The malabsorption syndromes of intestinal origin may be *primary*, as in nontropical sprue, idiopathic steatorrhea, tropical sprue, and celiac disease, or *secondary*, when there is some other specific condition involving the small bowel or its mesentery.

Most of the recognized causes of secondary malabsorption syndromes of intestinal origin are listed in Table I. Usually a combination

Table I

Causes of Secondary Malabsorption Syndromes of Intestinal Origin

Regional enteritis
Amyloidosis
Scleroderma (systemic sclerosis)
Radiation (actinic) enteritis
Whipple's disease
Tuberculosis
Lymphoma, carcinoma, carcinoid
Surgical resections, short circuits
Diverticula, blind loops, strictures
Internal fistulas (gastrojejunocolic)
Parasites (Giardia, Diphyllobothrium, hookworm)
Acute enteritis (viral, bacterial)
Diabetes mellitus with neuropathy
Mesenteric vascular insufficiency
Intestinal abnormalities (e.g., pneumatosis cystoides intestinalis)
Drugs (neomycin, phenindione)

of factors are responsible for a given malabsorption syndrome. The details of pathophysiology have been recorded elsewhere (BOOTH, 1960; BADENOCH, 1960; FISCHER, 1961; SCUDAMORE, 1961).

Differential Diagnosis

When a patient presents evidence of a malabsorptive state, a primary intestinal malabsorption syndrome should be considered first. However, the disorder should be differentiated from secondary syndromes of intestinal origin and from malabsorption syndromes of pancreatic, gastric, or hepatobiliary origin. The history, physical findings, and laboratory studies will suggest evidence of impaired absorption and permit some distinctions to be made. Radioactive vitamin B_{12} absorption studies will allow differentiation among sprue, pernicious anemia, and "stasis" syndromes (SCUDAMORE, 1961).

Roentgenologic examination of the small intestine following a barium meal may establish the diagnosis (LAWS AND PITMAN, 1960). A "deficiency pattern" is characteristically found in nontropical sprue, but it may be observed also in regional enteritis, amyloidosis, scleroderma, Whipple's disease, lymphoma, and diabetes mellitus with neuropathy. Distinctive roentgenologic changes may be found in these conditions as well as in cases of bowel resection, diverticulosis, blind loops, strictures, fistulas, and actinic enteritis.

In some instances, intubation biopsy of small intestinal mucosa or abdominal exploration with biopsy of bowel or mesenteric lymph nodes may be necessary to confirm the diagnosis.

Intubation Biopsy in Diagnosis

The importance of intubation biopsy in diagnosis and research has been suggested by several authors (BOLT et al., 1958; SHINER, 1959; CULVER et al., 1959; GIRDWOOD et al., 1961). The histologic changes of the mucosa in nontropical sprue and related disorders are well known, but some questions exist as to the specificity, reversibility, and significance of the observed changes.

Peroral intubation mucosal biopsy with the Crosby or Carey capsule (GREEN et al., 1961) was performed in a variety of malabsorptive disorders. The mucosa was classified as normal or, arbitrarily, as