Doerr · Seifert · Uehlinger

Spezielle pathologische Anatomie

Band 8

Tropical Pathology

By Herbert Spencer

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and

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Editorial Preface

General pathology works nomothetically, whereas specific pathology works idiographically. Modern pathological anatomy on the other hand seeks to rise above the level of merely gathering data and to proceed to a practical knowledge of defined pathological aspects and patterns. JEAN MARTIN CHARCOT'S conception of the entité morbide as the abstract basis of nosologically independent phenomena is paradigmatically corroborated in an especially impressive way by the study of tropical diseases. And so the idea suggested itself that we should present the main pathological patterns from the field of tropical pathology with particular reference to their interconnection. We soon came to the firm conclusion that such a compilation could be successfully undertaken only by a pathologist from the English-speaking world. The classic practical knowledge of our British colleagues, which has matured in world-wide experience, would be bound to find its ideal verification here.

And so we turned for help to our friend Prof. WALTER PAGEL (Dr. med., Dr. h.c. Basle, Dr. h.c. Heidelberg) in London. He immediately referred us to Prof. HERBERT SPENCER. That we managed to enlist the help of Professor SPENCER is entirely due to Professor PAGEL's kind offices. We owe the fact that the undertaking has been completed in relatively so short a time to Professor SPENCER's exceptional knowledge both of his subject and of the field-workers themselves. With extraordinary skill he overcame all organisational difficulties and with a masterly touch he made an excellent selection from the whole field of the pathological anatomy of tropical diseases. Our gratitude to him is as lasting as it is deep. However, we also have to thank all our other colleagues, who unhesitatingly complied with the proposed order and contributed decisively in this way to the success of the undertaking.

As always, our thanks go to Dr. HEINZ GÖTZE (Dr. phil., Dr. med. h.c., Dr. h.c.) and his Springer publishers for their co-operation to our plans and for the excellent quality of this volume, as also of their others.

W. DOERR
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Heidelberg-Hamburg-Zürich, August 1, 1973

Preface

Diseases in the tropics are in many respects identical with those found in temperate and arctic regions and their pathology is similar. Certain diseases, however, are confined to tropical regions either because the causative agents or their vectors can only survive in a tropical environment or because the necessary conditions for their propagation are nowadays mostly found in the underdeveloped tropical countries. Many of the infective diseases formerly endemic throughout the world less than a century ago are now confined almost entirely to underdeveloped and mainly tropical regions. In the ensuing account only those conditions which to-day are found mostly in the tropics will be considered. No attempt has been made to describe diseases such as tuberculosis, rheumatic disease, lobar pneumonia, measles etc. which are so widespread and claim so many lives in many tropical regions but which nevertheless still occur in temperate countries and are fully described in textbooks of pathology. Such diseases, however, may nevertheless be greatly modified by racial, dietary, and environmental factors and often present in a more severe and aggravated form in tropical countries. Likewise only those tumours which occur mainly in the tropics are considered individually and the reader is referred to textbooks on pathology for details of other neoplasms common to both temperate and tropical regions.

Whereas in former times the truly tropical diseases were almost entirely confined to the tropics, nowadays following mass emigration and the greatly improved facilities for rapid travel such diseases are seen with increasing frequency in temperate and highly developed countries, and thus a knowledge of tropical diseases becomes ever more important.

This book would not have been possible without the great help provided by numerous pathologists, parasitologists and clinicians throughout the world who so generously provided illustrations or material and whose names appear in the legends to the illustrations. The Editor especially wishes to thank the Commandant of the Royal Army Medical College, London, for allowing him to photograph many of the College parasitological specimens, to Dr. A. J. DUGGAN, Curator of the Wellcome Museum of Medical Science for access to the Museum specimens, and also Professor G. S. NELSON and Miss V. C. C. WILSON of the London School of Hygiene and Tropical Medicine for their helpful suggestions in the preparation of the chapters on helminth, amoebic and other protozoal diseases. Finally, he wishes to thank both Mr. A. E. CLARK and Mr. A. L. PACHE for their unstinted help in the preparation of many of the photographs and photomicrographs, Miss SUSAN WEBB for the preparation of much of the manuscript, and his wife for her great forebearance and continual encouragement and support.

Summer 1973 H. SPENCER

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Chapter 1

Intestinal Bacterial Infections

H. Spencer

Contents

Most of the diseases to be described in this chapter may occur throughout the world, but have largely disappeared in many temperate regions and now occur mostly in the tropics and subtropics. The diseases flourish wherever sanitary conditions are bad and water supplies are polluted. As sanitation and the quality of the water supplies improved throughout Western Europe, North America and Australasia during the latter half of the 19th Century the incidence of the intestinal bacterial infections in these areas dropped dramatically. Cholera, except for occasional sporadic cases introduced by travellers returning from endemic areas, has now almost disappeared from Western Europe, North America and Australasia where formerly it was endemic.

Typhoid fever though still endemic on a reduced scale around the shores of the Mediterranean and in Eastern Europe, has almost disappeared from Western Europe, North America and Australasia. In the latter areas occasional small, mainly water-borne epidemics occur following accidental contamination of water supplies, and sporadic cases have followed its introduction by returning travellers and immigrants from endemic regions.

Bacillary dysentery is still endemic throughout the world, though on a very much diminished scale than formerly in Western Europe, North America and Australasia where it is now mainly confined to small outbreaks in institutions for the mentally sick and subnormal, infants' schools and occasionally in children's wards in hospitals. In common with all intestinal bacterial infections it is a disease which flourishes among armies in the field and in conditions of famine occasioned by the mass movement of populations due to war and civil unrest. It is probably the most common of the intestinal bacterial infections in adults in tropical regions and is responsible for much morbidity and many deaths in the very young and the elderly. Together with other intestinal bacterial pathogens, including pathogenic strains of *E. coli*, it is probably responsible for more deaths among the neonatal and infant population in tropical underdeveloped regions than any other

agent. Often intestinal infection follows in the wake of malnutrition and famine.

In addition to these three principal intestinal bacterial infections, all of which are now mostly confined to the tropics, many other Salmonella infections are more common in underdeveloped, tropical regions where sanitation is bad and ignorance of the most elementary principles of hygiene is rife. Under such conditions all intestinal infections flourish and claim a particularly high mortality among the infant and child population.

Typhoid Fever

Synonym: Enteric Fever

Typhoid fever, the most serious and lethal of the many Salmonella infections, is of great antiquity and was known to Hippocrates. In this account the changes occurring in typhoid fever will be described but they also apply to the less severe Paratyphoid A, B and C infections. All four infections are referred to as the Enteric fevers, but typhoid fever is reserved for the infection caused by *Salmonella typhi*.

Typhoid fever has claimed multitudes of lives in past centuries and it was not until the introduction of the antibiotic chloramphenicol in 1948 that an effective drug became available for its treatment.

Until the close of the last century typhoid fever was endemic throughout most of the world but with the introduction of water-borne sanitation, firstly in the large cities of Western Europe and North America and more gradually elsewhere, coupled with the more general availability of clean water supplies, the incidence and importance of typhoid fever has dwindled to near vanishing point except for imported cases in many developed countries. It still remains, however, a serious disease and public health problem is less developed countries where the supply of clean water does not receive the same priority as in Western Europe, North America and Australasia. Today typhoid fever is still endemic throughout Africa, Asia, the Americas with the exception of the U.S.A. and Canada, and is still present though on a much more reduced scale than formerly in Southern and Eastern Europe.

The typhoid groups of fevers are now known to be alimentary infections as was foreshadowed by WILLIAM BUDD (1873), and are spread mainly through infected water supplies and to a lesser extent by faecally contaminated food, fruit and vegetables. Most of the major epidemics of typhoid fever can be traced ultimately to sewage contaminated water. Although a human intestinal carrier state may persist and can cause the spread of the disease, infected water is the usual source of infection. A further source is provided by infected shellfish gathered from sewage polluted seawater beds. The shellfish which include oysters, mussels etc. filter out and concentrate typhoid bacilli from the polluted water.

In most countries where the disease is still endemic, sporadic cases occur throughout the year but seasonal rises occur towards the end of the hot season. Furthermore, small focal areas of very high endemicity often occur within a country. In regions where schistosomiasis is endemic, chronic urinary typhoid carriers occur much more frequently and may provide a major source of continuing infection especially in rural areas. The problem is made more difficult by the

frequent absence of any clinically recognisable disease in such patients (FARID, 1970).

In common with many intestinal infections, typhoid fever can prove a scourge to Armies on active service unless prophylactic measures are taken coupled with the most stringent control of water supplies and the enforcement of sanitary discipline. The breakdown of public health control when mass and uncontrolled movements of populations occur favour the outbreak of large epidemics of the disease.

The causative bacterium *Salmonella (Eberthella) typhi* was discovered by EBERTH (1880). It is a gram-negative, motile bacillus with at least three antigenic components, the somatic, flagellar and Vi antigens. In common with many intestinal pathogens it is non-lactose fermenting and grows well in bile salt containing media.

Clinical

Typhoid fever may mimic many diseases in its early stages and may present in many unusual ways. In a classical untreated case the stage of onset (invasion) is characterised by general malaise, headache which may sometimes simulate meningitis, epistaxis, nausea, constipation, sore throat and a daily step-ladder rise in temperature often coupled with bradycardia. By the end of the first week the septicaemic phase of the disease is established with a high swinging temperature, and in a minority of cases a characteristic exanthem appears on the abdomen in the form of Rose spots. In severe untreated cases the patients become delirious and sink into a stupor. The tongue is coated, the spleen and liver enlarge and the abdominal reflexes disappear. At this stage a persistent cough due to pharyngitis may be much in evidence and bronchopneumonia may develop and the patients may die about the 10th to the 12th day from overwhelming septicaemia and endotoxic shock. At the end of the second week the stage of intestinal ulceration with its complications begins. The abdomen becomes distended and tympanitic and a pea-soup type of diarrhoea may gradually develop though in many cases constipation may continue. Towards the end of the third week the sudden complications of intestinal haemorrhage and perforation may develop. In uncomplicated cases the convalescent stage begins in the fourth week, the temperature falling by lysis. Relapse, however, after the patient has become afebrile occurs in about 10% of cases. During the course of the illness cardiac irregularities not infrequently develop including pulsus alternans and auricular fibrillation.

The course of the disease has now, however, been radically altered since the introduction and use of chloramphenicol and ampicillin which abort the illness and reduce the febrile period. Unless a sufficiently long course of treatment is given, relapse and drug insensitivity are likely to occur. Chloramphenicol resistant strains of *S. typhi* due to transferable R. factor have occurred in Mexico and some have been successfully treated with co-trimoxazole.

Ambulant cases of the disease are seen in which the patient first presents with intestinal perforation, and as already stated an afebrile illness may characterise typhoid fever in association with schistosomiasis.

In the early stages of the illness the enteric group of diseases have to be distinguished from the many other causes of a pyrexia of unknown origin (PUO).

Pathology

The lesions in established typhoid fever are well known and described. As GOODPASTURE (1937) stated, however, little is still known about how *S. typhi* enter the body tissues. Several recent experimental investigations have shed light on the manner in which other Salmonellae penetrate the intestinal mucosa and a similar mechanism probably applies to *S. typhi* invasion in man. Whether or not typhoid fever follows the ingestion of *S. typhi* depends upon a number of variable factors including the number of ingested organisms, the state of the gastric acidity, the motility of the bowel, the virulence of the strain of *S. typhi* and the general state of health of the individual (HORNICK and WOODWARD, 1966). It is now generally agreed that *S. typhi* in common with other Salmonellae enter the body through the small intestine epithelium and do not gain access as was at one time thought through the tonsil and pharynx. KENT *et al.* (1966) showed that in guinea pigs a reduction of the intestinal motility by opium facilitated the entry of *S. typhimurium* through the intestinal mucosa and the organisms were demonstrable in the liver and spleen within 24 hrs of ingestion. TAKEUCHI (1967) in a series of electron microscopic studies showed that *S. typhimurium* invade the mucosal epithelial cells by invaginating the free surface. The microvilli at the point of invasion swelled and fused together as the bacterium moved through the brush border. The bacteria were also capable of disrupting the surface junctional complexes uniting epithelial cells and of penetrating the epithelial barrier by passing through the intercellular spaces. Many of the bacteria contained within phagosomal sacs inside the epithelial cell were probably destroyed by lyzozomal action but others succeeded in reaching the lamina propria. From there they were carried both by lymphatics and the portal vein to the mesenteric lymph glands and liver and soon reached the blood stream. In the case of *S. typhi* following the dissemination of the bacteria throughout the body and their continued selective growth in reticulo-endothelial cells, further multiplication in the bowel is restricted to the sites of lymphoid aggregations in the ileum and proximal large intestine.

S. typhi excites a characteristic mononuclear cell type of cellular response wherever it multiplies. The characteristic cells closely resemble at first sight a plasma cell and also large macrophage cells (Fig. 1.2). The former cell type though about the same size as a plasma cell has a much denser chromatin nuclear mass which does not show a characteristic clock face pattern, and the cytoplasm is more eosinophilic staining than the cytoplasm of a plasma cell. The macrophage cells also have a denser and more compact nucleus than most tissue macrophages and the cytoplasm is filled with nuclear debris, red blood cells and typhoid bacilli. Many of the macrophages die and the resultant necrotic area contains much karyorrhectic nuclear debris (Fig. 1.3). It is probable that both the cell types found in typhoid lesions are closely related and are types of macrophage cells. This view receives further confirmation as GOODPASTURE found bacilli *(S. typhi)* in the cytoplasm of both types of cells.

The Peyers patches become swollen and pink by the end of the first week but their surface mucosa remains intact but rugose. At the same time the mesenteric lymph glands are enlarged, soft and red. By the end of the second week the rugose mucosa starts to ulcerate resulting in greenish-brown, bile tinged, ragged ulcers

Fig. 1.1. (A) Photograph showing terminal portion of ileum with ulcerated Peyer's patches, the ileocecal valve, appendix and caecum. In the latter there are small circular ulcers involving lymphoid tissue. (B) Specimen showing ulcerated Peyer's patches in ileum. × 2/3rds natural size

restricted to the Peyers patches (Fig. 1.1). Ulceration is partly a consequence of actual necrosis due to bacterial damage and partly to thrombosis of mural blood vessels. At this stage the mesenteric lymph glands are more swollen and contain areas of necrosis and haemorrhage in their substance. The latter may occasionally result in large retroperitoneal haemorrhages. By the third week the floor of the ulcers extend to the muscle coat and in 2% of untreated cases perforation occurs or erosion of blood vessels results in massive intestinal haemorrhage. As the intes-

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