Tuberculosis in Children EVOLUTION, EPIDEMIOLOGY, TREATMENT, PREVENTION

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Pretace

The first purpose of this book is to help doctors to recognise, treat and prevent tuberculosis in children. The second is to help teachers to look at tuberculosis as a general disease with a long natural history, which can, nevertheless, be controlled and treated.

The method used is to relate diagnosis and prevention to natural history as that is modified by age and nutrition of the host and changes in the environment. Disease can then be understood as a relationship between host and invading organism.

Tuberculosis begins with primary infection and will only be eradicated when infection ceases. Yet, although effective treatment and protective vaccination have been available for 30 years, tuberculosis remains, over much of the world, a major cause of death and a major problem in child health.

Each working doctor must help to change this situation and be able to answer the following questions about any child in his care:

- 1. Is this child infected with tuberculosis?
- 2. Is this illness caused by tuberculosis?
- 3. If so, how do I deal with it?
- 4. Where did the infection come from?
- 5. Have other adults or children been infected?
- 6. If the child is not infected, how can the risk of future infection be reduced?
 - 7. What is the prevalence of infection in the community?
 - 8. What can I do to prevent infection in other children?
- 9. Are there any local customs, beliefs, conditions of life which impede the acceptance of thoughtfully organised care?

If practical answers to these questions can be found then the book will have been worthwhile.

The need to understand the evolution and timetable of tuberculosis as a conceptual basis for preventive and therapeutic work also determines the arrangement of the material. The early chapters deal with the concept of primary infection; the nature and use of the phenomenon of tuberculin sensitivity; the practical use of BCG as a protective mechanism and the application of epidemiology to control and for prevention. Later chapters then describe the clinical manifestations and treatment of tuberculosis in children from birth to adolescence.

Mycobacteria other than *M. tuberculosis* and *bovis* are increasingly recognised as causes of illnesses in children and must be differentiated from 'true' tuberculosis. These organisms and the illnesses they produce are considered in Chapter 19.

Mycobacterial infections of animals can have important associations with human infections and are described briefly in Chapter 20.

An author must not leave his meaning or his convictions in doubt, so the ways in which technical terms have been used and the author's basic beliefs conclude the introductory section.

The book is founded upon experience with and study of tuberculosis in children in England between 1935 and 1970, and opportunities to observe children with tuberculosis in South East Asia during visits from 1966–1979. A detailed account of the early experience, shared with colleagues, was published in 1963 (Miller et al, 1963) in a volume now out of print. Later experience outside the United Kingdom has emphasised how the manifestations of tuberculous infection and the problems of control and therapy are affected by the local circumstances of the communities in which they occur.

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viii TUBERCULOSIS IN CHILDREN

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Terminology

Primary focus:

A primary focus is the cellular and structural change occurring in response to the presence and multiplication of Mycobacteria tuberculosis at the site of first implantation into the tissues of the host; the usual site is in a lung.

Primary adenitis: (Regional lymphadenitis)

The changes in the lymph nodes draining the primary focus. The first affected node is usually the most enlarged (primary node). Others along the line of lymph flow are smaller (satellite nodes). The primary node accurately reflects the anatomical site of the primary focus.

Primary complex:

The primary focus and associated tuberculous lymphadenitis.

'Fever of onset':

A phrase used by Wallgren to describe the fever sometimes observed in the host during the period of the development of the primary complex and the onset of tuberculin sensitivity.

Tuberculin:

A protein extract obtained from tubercle bacilli. A specific tuberculin can be extracted from each type of mycobacterium. An individual sensitised to one type of tuberculin may also be sensitive, but to a lower degree, to other types.

Tuberculin conversion:

The change in tissue reaction to tuberculin which results from primary infection with M. tuberculosis (or other mycobacteria).

Tuberculin skin sensitivity:

The delayed inflammatory reaction produced in the skin by the injection of tuberculoprotein in a person infected with a mycobacterium.

XIV TUBERCULOSIS IN CHILDREN

Tuberculin reversion:

Loss of skin sensitivity in a person previously known to possess the characteristic.

Primary exogenous reinfection:

The occurrence of a second primary infection with the development of a complex in a person who has undergone tuberculin reversion after the first infection.

Exogenous reinfection: (Suprainfection)

The production of a tuberculous lesion by a new infection, from another source, in a person who has had a primary infection and has remained tuberculin sensitive. The term exogenous suprainfection is also used to describe the same process.

Endogenous exacerbation:

The reactivation of an old and apparently healed tuberculous lesion derived from the primary focus, the associated adenitis or the haematogenous seedings thereof. The term endogenous reinfection is sometimes used to describe this process.

Erythema nodosum:

A painful red nodular rash typically seen on the shins: caused by several antigens in different parts of the world. When tuberculous it usually denotes a high degree of sensitivity to tuberculin and occurs at the time of primary infection.

Phlyctenular conjunctivitis:

A sensitivity phenomenon characterised by the appearance, at the sclero-corneal junction of one or both eyes, of tiny grey spots each at the apex of a leash of vessels. The eruption occurs with, or any time after, tuberculin sensitization and is often recurrent.

Segmental lesion:

The involvement of a pulmonary segment, lobe or whole lung secondary to bronchial erosion with the aspiration of material from the affected nodes. The term is descriptive of the extent of pulmonary involvement but does not denote a lesion with a particular histological appearance. The term includes the type of consolidation termed epituberculosis.

Epituberculosis:

See Segmental Lesion above; essentially a non-specific iobar consolidation, secondary to bronchial erosion, which clears spontaneously.

Obstructive hyperinflation: (sometimes called obstructive emphysema)

The hyper-inflation of a pulmonary segment, lobe or whole lung resulting from incomplete bronchial obstruction having a ball-valve effect allowing air to enter but not leave the affected area.

Round shadow: (Synonyms: Round Focus, 'Coin' Shadow, Nummular Shadow)

Any round solid shadow seen on radiological examination of the lung fields. The lesion, if tuberculous, may be caused in a variety of ways and the term 'round shadow' does not presuppose any single aetiology or histological appearance.

Miliary tuberculosis:

The generalised dissemination of tubercle bacilli and tubercle formation throughout the body. The term can be used in two senses, one clinical and the other pathological.

Chronic pulmonary tuberculosis: (Synonyms: bronchogenic reinfection, adult-type, post-primary pulmonary tuberculosis)

There has always been difficulty in finding an acceptable term to describe the slow but usually progressive pulmonary lesions which often arise years after primary infection and characterise the disease in adults. Objections can be raised to each of the synonyms; bronchogenic implies a particular route of spread; reinfection a particular origin; adult a particular age group; post—primary is often used for lesions appearing shortly after the primary infection; here the term chronic pulmonary tuberculosis is preferred. Whenever possible it should be amplified by further description e.g. exacerbation of primary focus or exacerbation in segmental lesion.

Bone and joint tuberculosis:

Includes all synovial and osseous lesions. These are always metastatic from a primary complex elsewhere in the body.

Serous meningitis:

A lymphocytic reaction in the cerebrospinal fluid with fever and headache, occurring occasionally in the course of primary infection and resolving spontaneously.

Tuberculous meningitis:

A progressive meningitis caused by the liberation of tubercle bacilli (and caseous material) into the c.s.f. following the rupture of a caseous lesion in brain tissue or on the meninges.

XVI TUBERCULOSIS IN CHILDREN

Froin's syndrome:

Occurs in spinal block preventing the circulation of c.s.f. Lumbar fluid below the obstruction becomes yellow and has a high protein content.

Tuberculoma:

A caseous tuberculous lesion in the substance of the brain or spinal cord. If large it may produce the symptoms and signs of a space-occupying lesion.

Tuberculide:

Any tuberculous lesion in the skin, other than a primary focus or erythema nodosum, whether or not bacilli can be demonstrated. Usually some further descriptive term is added e.g. papulo-necrotic or verrucose.

Tuberculous encephalopathy:

An illness with signs of encephalitis with loss of consciousness. Gross oedema of brain.

Post-primary tuberculosis:

This term is used by some writers to mean the group of local or haematogenous complications which occur in the first few months after primary infection. Others use it to describe later lesions, such as pulmonary round shadows not clinically related to the primary infection. In this book it is used only to describe the uncommon occurrence of a pleural effusion or erythema nodosum long after a primary infection and difficult to fit into the natural history of primary infection.

KEYS TO CONTROL AND ERADICATION

The key to the elimination of tuberculosis is the prevention of primary infection at any age by the progressive reduction of infective respiratory disease and the treatment of natural primary infection whenever detected.

The key to prevention is to understand the natural history of tuberculosis from the time of primary infection, the local epidemiology in any community and the correct use of BCG in that particular community.

The key to diagnosis is to appreciate the conditions and clinical situations in which tuberculosis is encountered. Suspicion is more than half way to diagnosis; once suspected diagnosis is rarely difficult.

The key to treatment is to accept the conditions required for successful chemotherapy, to treat as soon as possible for a sufficient length of time, with an effective regimen.

The eradication of tuberculosis from any community is possible given sufficient staff with the will and wherewithal to work, the cooperation of the people and the foresight to plan two generations ahead.

Contents

Terminology Keys to control and eradication	xiii xvii
PART I BASIC FACTS	
1. The evolution of primary infection with Mycobacterium tuberculosis	3
Mycobacterium tuberculosis and the human host/The concept of primary infection/The development of the primary complex/The mechanisms, timetal and risks of complications of untreated primary pulmonary infection/Effect of nutrition and intercurrent infection/The relationship of primary infection to chronic pulmonary tuberculosis/Regional differences in tuberculosis in childs	age,
2. Tuberculin sensitivity and the tuberculin test	18
Origins/Tuberculin sensitivity/Standardisation of tuberculin/Test dosage/Techniques of tuberculin testing/Comparison of tests/Geographical variation in tuberculin sensitivity and the efficiency of the tuberculin test	
3. The epidemiology of tuberculosis	37
The epidemiological situation/Measurement of tuberculosis in a population/I contemporary situation	î he
4. BCG vaccination	55
Rationale of vaccination/BCG vaccine/Techniques of vaccination/Events afte vaccination/Tuberculin sensitivity after vaccination/Revaccination/Complicat of vaccination/Accidents during vaccination/Progressive BCG infection/The efficacy of BCG vaccination/Studies of BCG vaccination/Tuberculosis after Evaccination/BCG and leprosy	ions
5. Control of tuberculosis	73
The doctor's responsibility/National tuberculosis programmes/Contact tracing/The doctor's personal responsibilities	
PART II CLINICAL MANIFESTATIONS	
6. The recognition, diagnosis and treatment of tuberculosis in children	85
$Introduction/Presentation/Symptom\ complexes\ of\ tuberculosis/Tuberculosis\ nutrition/Diagnosis/Treatment$	and

X TUBERCULOSIS IN CHILDREN

	. Intrathoracic tuberculosis and miliary spread	105
C	Pulmonary primary infection/Bronchial complications/Cardiac omplications/Chronic pulmonary tuberculosis/Progressive pulmonary uberculosis/Miliary tuberculosis	
8.	Oral and alimentary tuberculosis	137
te	Primary tuberculosis of the mouth/Visible primary tuberculosis of the onsil/Tuberculosis of middle ear and mastoid/Tuberculosis of the esophagus/Abdominal tuberculosis	
9.	Tuberculosis of the skin	144
ir	Classification (with synonyms)/Diagnosis of skin tuberculosis/Primary skin nfection/Haematogenous lesions/Injection abscesses/Erythema nodosum	
10.	. Tuberculosis of eye and conjunctiva	156
P	rimary infection of conjunctiva/Phlyctenular conjunctivitis/Choroidal uberculosis	
11.	Superficial tuberculous lymphadenitis	161
	linical presentation/Treatment	
12.	Tuberculosis of the central nervous system	168
II ei	ntroduction/Tuberculous meningitis/Tuberculomata/Tuberculous ncephalopathy/Tuberculous arachnoiditis and radiculitis/Paraplegia of uberculous origin	
13.	Tuberculosis of bones and joints	186
G	deneral considerations/Tuberculosis of the spine/The hip/The knee/Other b and joints/Multiple cystic tuberculosis of bone (Jungling's disease)	ones
14.	Tuberculosis of genital tract and breast	209
	Male genital tract/Female genital tract/Tuberculosis of the breast	
15.	Tuberculosis of the renal tract	212
E	volution of lesions/Clinical aspects/Differential diagnosis/Treatment	
16.	Tuberculosis of liver, spleen, haemopoietic system and adrenals	215
T	he liver/The spleen/The blood/The adrenals	
17.	Congenital tuberculosis and infection early in infant life	220
M sy	dechanisms and epidemiology/Pathology/Condition of the mother/Clinical and prognosis/Protection of the newborn in	
18.	Tuberculosis in immigrant children in the United Kingdom	225
ın	ntroduction/The epidemiological situation/Tuberculous infection and morbin immigrants/Policy regarding assessment, treatment and prevention/Clinical insiderations	ditu

CONTENTS	X1
19. Non-tuberculous mycobacterial infections	232
Introduction/Classification of organisms/Distribution of organisms/Sensitivity chemotherapy/Clinical syndromes of infection in children/Sensitivity to mycobacteria other than M. tuber.vulosis	to to
20. Tuberculosis in animals	250
Introduction/M. bovis infection/Tuberculosis in animals other than cattle	
Epilogue	256
Appendices	257
Introduction	
Appendix 1 Examination of gastric contents for AFB Appendix 2 Laryngeal swabs	
Appendix 3 Staining of M. tuberculosis and other acid fast bacilli—	
Ziehl-Neelsen method	
Appendix 4 Examination of c.s.f. for AFB	
Appendix 5 Locally prepared food (Hyderabad Mix)	
References	263
Index	279

Basic facts

The evolution of primary infection with Mycobacterium tuberculosis

MYCOBACTERIUM TUBERCULOSIS AND THE HUMAN HOST

The disease known as tuberculosis is the reaction of the tissues of the human host to the presence and multiplication of Mycobacteria tuberculosis or bovis. Therefore in describing the clinical states arising from the presence of organisms we are analysing a relationship which is the outcome on the one hand of the capacity of the host to contain and eliminate the organisms (resistance) and, on the other, of the organisms to multiply (pathogenicity, virulence).

Human disease is spread from persons with infective respiratory lesions by coughing so that organisms are inhaled, swallowed or come into contact with the skin or mucous membranes of susceptible persons. Infection with M. bovis is spread largely by infected unboiled cows milk. (Ch. 20)

Understanding of the fundamental importance of the first or primary infection came slowly over the past century and the concept of the primary infection evolved from the studies of Parrot (1876), Gohn (1912), Ranke (1917), Rich and McCordock (1929), Wallgren (1938a & b, 1948) and others who studied tuberculosis before chemotherapy was available. Yet even now the significance of the primary infection for the continuance of tuberculous infection from one generation to the next is not sufficiently recognised.

THE CONCEPT OF PRIMARY INFECTION

Wherever and whenever tubercle bacilli first lodge and begin to multiply in human tissues the invasion begins a series of events related in time.

Some bacilli remain at the site of entry and some are carried swiftly in the lymph flow to the nearest nodes. Multiplication then occurs in both places and the body responds so that 'tubercles' develop. The lesions at the point of entry and those in the node

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