Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

Kaapstad, 14 September 1963 Deel 37 No. 37 Volume 37 Cape Town, 14 September 1963

VAN DIE REDAKSIE : EDITORIAL

TERINGBESTRYDING IN SUID-AFRIKA

Ten spyte van die groot vordering wat daar gedurende die afgelope aantal jare op haas elke gebied gemaak is ten opsigte van die bestryding van infektiewe siektes in die algemeen, bly die toestand wat tuberkulose betref nog baie onbevredigend.

Gedurende die eerste helfte van hierdie eeu het ons 'n algehele revolusie beleef wat betref die soort benadering tot die probleem van tuberkulose. Daar is, byvoorbeeld, die faktore van vermeerderde aandag aan maatskaplike toestande soos voeding, behuising, ens., die gebruik van tuberkulien, binnehuidse BCG, massa-röntgenografie, en antituberkulosemiddels, en tog styg die aanmeldingsyfer nog steeds.

Hoe ernstig die posisie is, kan uit die volgende gegewens afgelei word: In 1962 was daar in Suid-Afrika 63,450 nuwe aanmeldings—'n toename van ongeveer 5,000 bo dié van die vorige jaar. Dit beteken dat daar meer as 170 nuwe gevalle elke dag aangemeld word. Veertig pasiënte sterf elke dag (meer as 15,000 jaarliks) aan tuberkulose.

Hierdie verhoogde aanmelding van tuberkulose vind plaas uit al die seksies van die gemeenskap, van die mees bevoorregte tot die mees primitiewe. Die feit dat daar in 1962 5,000 meer nuwe gevalle as in 1961 aangemeld is, beteken natuurlik nie noodwendig dat daar soveel meer persone siek geword het nie, aangesien die verbeterde metodes van opsporing van gevalle tot hierdie resultaat kon bydra. Hoe dit ook al sy, die feit van die saak bly dat die posisie ernstig is. Die hoop wat bestaan het dat tering deur die nuwe chemoterapeutiese middels uitgewis sal word, is verydel. Die hele vraagstuk van teringbestryding moet dus nog soos in die verlede as 'n nasionale probleem aangepak word en aandag moet aan alle aspekte daarvan gegee word. Dit is wel waar dat die beskikbare aantal beddens van 1946 tot 1961 vermeerder het van ongeveer 3,000 na ongeveer 29,000, maar dit moet vir almal duidelik wees dat as daar elke jaar 63,000 nuwe gevalle aangemeld word, daar baie meer gedoen moet word.

In die lig van die voorgaande is dit dus van die allergrootste belang om erkenning en ondersteuning te verleen aan pogings en bewegings wat hulle ten doel stel om hierdie siekte te bestry. Dit is dus van pas dat ons hier kortliks verwys na die monumentele bydrae wat die Suid-Afrikaanse Nasionale Teringvereniging (SANTA) op hierdie gebied gelewer het en nog lewer.

SANTA bestaan nou alreeds 16 jaar. Dit is 'n vrywillige vereniging wat bestaan uit toegewyde lede wie se doel en strewe dit is om hierdie ernstige siekte te beveg en dit, indien moontlik, met wortel en tak uit te roei. SANTA het alreeds 34 sentrums in Suid-Afrika gestig met ongeveer 7,000 bedde daarin beskikbaar. Meer as 40,000 pasiënte is al in hierdie sentrums teen 'n lae koste behandel. SANTA het ook 120 takke, die meeste waarvan bedien word deur vrywillige werkers. Deur hierdie takke word behoeftige tuberkulose pasiënte en hulle gesinne gehelp met kos, klere, huurgeld, advies, rehabilitasie, ens.

Op die gebied van publieke opvoeding het SANTA ook groot dienste gelewer. Alle moontlike middels word gebruik in hierdie opvoedkundige kampanje, insluitende kennisgewings, pamflette, films en lesings wat gerig word tot alle dele van die gemeenskap. SANTA stel hom verder ten doel om alle nuwe gevalle so vroeg as moontlik te probeer opspoor. In Johannesburg het SANTA byvoorbeeld twee mobiele röntgeneenhede, en verder moedig die vereniging mense aan om röntgenondersoeke te laat doen deur die Staatsgesondheidsdiens of die plaaslike owerhede.

Vir die toekoms het SANTA groot planne en ondernemings in gedagte. Hierdie planne sluit in die propagandering van grootskaalse BCG-inenting, die voorsiening van beskutte arbeidsgeleenthede vir chroniese teringlyers en die daarstelling van beurse vir werkers op die gebied van teringbestryding.

Dit moet dus duidelik wees dat SANTA 'n uiters belangrike skakel vorm die hele masjinerie wat gerig is op teringbestryding. Aangesien SANTA egter 'n vrywillige vereniging is, het dit fondse nodig om die werk te kan verrig wat in die vooruitsig gestel word. Die vereniging het dan ook besluit om hierdie jaar (vir die eerste keer sedert 1952) weer op nasionale skaal 'n poging aan te wend om fondse in te samel om teringbestryding op die beste en mees produktiewe wyse moontlik aan te pak. As mediese professie kan ons hierdie lofwaardige pogings nie sterk genoeg ondersteun nie.

GLAUCOMA

Glaucoma is presently one of the commonest causes of blindness in civilized communities. In England and Wales it is responsible for 14% of the blind population.

The disease is defined as a composite congeries of pathological conditions which have the common feature that their clinical manifestations are to a greater or lesser extent dominated by an increase in the intraocular pressure and its consequences,[1,2] and is classified into three clinical types — congenital, secondary and primary. Congenital glaucoma includes cases presenting from infancy up to early adult life, often associated with developmental abnormalities of the anterior chamber angle. Secondary glaucoma occurs as a complication of some other ocular disease, e.g. iridocyclitis or intraocular tumour. The primary adult glaucomas, however, are the largest and most important group.

In the normal eye, aqueous humour, secreted by the ciliary body into the posterior chamber, maintains a constant flow through the pupil into the anterior chamber, from there by way of the trabecular tissue at the angle

of the anterior chamber into Schlemm's canal, and thence to the extraocular venous system. In primary glaucoma affecting the adult, there is an obstruction to this flow. The obstruction may operate in one of two ways — either as closed-angle glaucoma or as open-angle glaucoma.

Closed-angle glaucoma affects the hypermetropic eye with a shallow anterior chamber in which the lens is relatively large and bulky and the anterior chamber becomes narrow and crowded. The lens continues to grow throughout life, and by the age of 50 years it may act as a stop in the pupillary aperture. The flow of aqueous humour through the pupil is impeded, a positive pressure builds up behind the iris, which balloons forwards, and the peripheral part of the iris is forced against the trabecular meshwork in front of the canal of Schlemm. Outflow of aqueous becomes impossible and intraocular pressure rises rapidly, producing an attack of acute closed-angle glaucoma. The entire eye becomes oedematous, owing to obstruction of venous outflow. Oedema of the iris results in paralysis of the sphincter and dilator muscles, and the pupil is fixed and semi-dilated. Corneal oedema causes blurring of vision, and the patient may see coloured haloes around lights; a most important symptom and one that is almost pathognomonic of this disease. Females are more frequently affected than males. Repeated attacks may result in adhesions between the peripheral iris and the corneo-trabecular meshwork, so that flow becomes reduced and the intraocular pressure is permanently raised, resulting in chronic closed-angle glaucoma. An acute attack can be artificially produced in persons with shallow anterior chambers and narrow angles by dilating the pupil;[3] the iris is thrown into folds, which may be forced against the trabecular meshwork, and the aqueous outflow is impeded. Hence the danger of using mydriatics, e.g. atropine, in the presence of a shallow anterior chamber and in patients with closed-angle glaucoma.

In primary open-angle glaucoma (glaucoma simplex) aqueous outflow is gradually reduced, despite a deep anterior chamber and open angles that appear clinically normal. The mechanism here is ill-understood, but is at present believed to be due to either sclerosis or degeneration of the trabecular meshwork, so obstructing aqueous outflow.[4] It is possible that the obstruction is caused in some cases, especially in Negroes, by episcleral pathology.[5] The disease has an equal sex incidence, and heredity plays a considerable part. Abnormalities of aqueous humour dynamics are commoner in relatives[6] and siblings[7] of known patients with open-angle glaucoma, than in the population generally.

Open-angle glaucoma is characterized by an absence of symptoms, particularly in the early stages, the only abnormality being an increased lability of intraocular pressure in its diurnal variation. This gradually gives way to permanently raised intraocular pressure and a reduction of aqueous outflow as measured by tonography. Visual deterioration comes much later (15 - 20 year after the onset of raised intraocular pressure)[8] and manifests itself as a gradual and characteristic constriction of the visual field, leaving central vision intact until the end stages of the disease; this constitutes a serious pitfall in diagnosis. *Pari passu* with field loss, pathological cupping and later atrophy of the optic disc are found. Finally, the whole field of vision is engulfed and the eye becomes blind. The disease is almost always bilateral and at a different stage in each eye.

Pathological cupping of the optic disc with constriction of the visual field results from chronic ocular hypertension and ischaemia of the optic-nerve fibres, and is common to the late stages of both types of primary glaucoma. When seen late the two types present a similar clinical picture and can be differentiated only by a careful history-taking and an examination of the anterior chamber angle with a gonioscope. In rare instances both types of glaucoma may co-exist; this is called mixed glaucoma.[9]

Treatment is directed at increasing aqueous outflow and reducing aqueous production, either by medication or by operation. Optic-nerve damage is irreversible, but successful treatment will arrest further damage. Therefore it becomes important to diagnose the disease early and commence treatment before pathological cupping of the disc occurs.

In closed-angle glaucoma early diagnosis presents little difficulty. There are the prodromal symptoms of blurring of vision and coloured haloes around lights, and an acute attack occurs early in the course of the disease, except in those unusual cases in which the disease runs a chronic course, when diagnosis becomes more difficult. If the diagnosis is in doubt an acute attack can be provoked by dilating the pupil (provocative test). Treatment should always be by operation, because a miotic, although relieving the acute attack, does not remove the root cause (pupillary block), and does not prevent recurrences.[10] Peripheral iridectomy establishes free communication between the posterior and anterior chambers, and in properly selected cases is adequate treatment;[11] in others a filtration operation is required.

Early diagnosis of open-angle glaucoma is more difficult and depends on demonstrating raised intraocular pressure (which may be intermittent) and reduced aqueous outflow. Sporadic diagnoses may be made by an ophthalmologist's inspired suspicion, but the belief is growing that early detection can only be effectively achieved by screening all persons over the age of 45 years; tonometry is at present thought to be the best screening test available.

Mass screening measurements of intraocular pressure have been carried out in the USA,[12] South Africa[13] and Germany,[14] among other countries. The measurement is made with a Schiotz tonometer on random samples of the population. Accepting the upper limit of normal as 21 mm.Hg on the 1957 Friedenwald scale,[15] between three and four per cent of those over 45 years of age in the population tested were found to have raised intraocular pressure and were subsequently subjected to tests designed to measure the aqueous outflow (tonography) and/or to provoke an abnormal rise of the intraocular pressure, e.g. the water-drinking test (water-loading). Definite early open-angle glaucoma was found in approximately two per cent of the screened population over 45 years of age. Those affected do not necessarily exhibit cupping of the optic disc or visual-field loss at this stage.

Sufficient work has been done to permit the generalization that approximately two per cent of the population over 45 years of age has unsuspected early open-angle glaucoma. This represents a minimum figure, because only

those persons with a raised intraocular pressure at the time of testing are discovered by the screening test. Effective treatment at this stage will probably arrest the glaucomatous process; the evidence for this is sufficient to justify action based on this concept. Early diagnosis in all cases is essential, and we believe this can best be achieved, in the context of South African medical practice, if tonometry becomes a routine part of every general practitioner's physical examination of individuals over the age of 45 years. Tonometry is a simple and safe procedure, and the instrument used is inexpensive. Patients with raised intraocular pressure should be referred to an ophthalmologist for further assessment and, if necessary, treatment. Thus the all-too-common sight of patients presenting for the first time with one eye totally blind from open-angle glaucoma and the other affected with advanced glaucoma, can become a memory of the past.

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