Fundamentos de un laboratorio de investigaciones básicas

Laboratorio Central de Líquido Cefalorraquídeo (LABCEL)

Antecedentes

- El 14 de abril de 2004 se funda el Laboratorio Central de Líquido Cefalorraquídeo (LABCEL) adjunto a la Facultad de Ciencias Médicas Miguel Enríquez.

Misión de centro

Es un laboratorio de referencia nacional para el estudio del líquido cefalorraquídeo desde el punto de vista de investigación básica, docente y clínico.

Visión del centro

- Realiza proyectos de investigación para los Programas Nacionales, Ramales, Territoriales e Institucionales.
- Imparte docencia de pre y post grado nacional e internacional y
- Brinda servicios científico-técnicos de alto valor agregado

Funciones de centro

- Investigaciones básicas, de desarrollo y de innovación tecnológica
- Docencia de pre y post grado para estudiantes nacionales y extranjeros, dentro y fuera del Sistema Nacional de Salud.

Clasificada por el CITMA como una ECTI

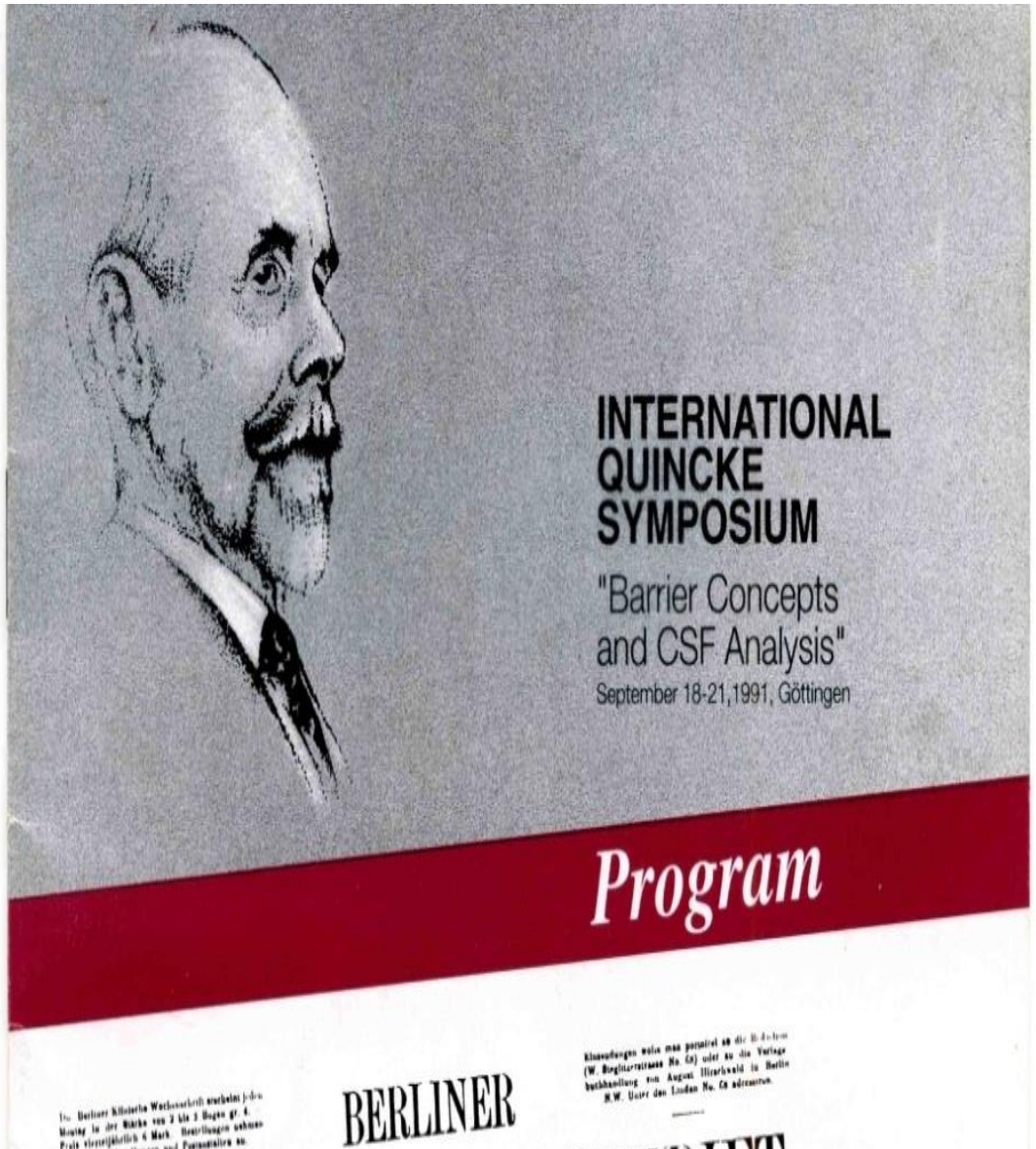
Entidad de Ciencia, Tecnología e Innovación

- Centros de Investigación (institutos)
- Centros de Servicios Científicos y Tecnológicos(LABIOFAM)
- Unidades de Desarrollo e Innovación (LABCEL, CNGM)

Génesis de LABCEL

Visita de Klaus Felgenhauer y Ernest Alan Meyer al Laboratorio de Inmunología del Hospital Pediátrico San Miguel del Padrón 1991

International Quincke Symposium Göettingen, Alemania 1991



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Trabajo conjunto 1994-1996

Visitas de trabajo 1996-2000

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Intrathecal Synthesis of Immunoglobulins in Eosinophilic Meningoencephalitis Due to *Angiostrongylus cantonensis*

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Eosinophilic meningoencephalitis due to the nematode *Angiostrongylus cantonensis*, which is endemic to Cuba, occurs in children and is due to accidental contact with soil snails. The course is less often fatal than in adult patients in southeastern Asia. Cerebrospinal fluid (CSF) and serum samples from 24 pediatric patients were analyzed and evaluated in CSF/serum quotient diagrams (Reiber graphs) to characterize the neuroimmunological response and the blood-CSF barrier dysfunction that occur in the course of the disease. At the time of the first diagnostic lumbar puncture, together with eosinophilic pleocytosis (1,920 ± 400 cells/μl), intermediate blood-CSF barrier dysfunction (i.e., an increased CSF/serum albumin quotient) with no intrathecal immunoglobulin G (IgG), IgA, and IgM class response was observed in all cases. Seven days later, at the time of early clinical recovery, the blood-CSF barrier dysfunction was normalized in 75% of the patients, but meanwhile, intrathecal immunoglobulin synthesis emerged in all cases, as either a two-class response (IgG and IgA in 85% of the patients) or a three-class response (IgG, IgA, and IgM; 30%). The fraction of eosinophilic cells (40%) remained large despite a decreasing total cell count. The neuroimmunological pattern of this inflammatory response to the parasite and its toxins is discussed with regard to the CSF patterns of other infectious diseases caused by bacteria or viruses.

Eosinophilic meningitis or meningoencephalitis induced by the nematode *Angiostrongylus cantonensis* is a disease with a poor prognosis commonly seen in southeastern Asia (6, 16), where fatal and chronic cases frequently occur. It was first recorded in Cuba in 1981 (1) and later in Puerto Rico (2). Most of the cases reported involved children with clinical manifestations different from those of adults (3), with less severe complications. In the majority, the anamnesis showed a history of accidental contact between soil snails and children living in rural and semirural areas. Infective third-stage larvae of the nematode develop in slugs and snails. Humans are infected due to ingestion of an infected intermediate host (1, 3, 6). In Cuba and other Caribbean countries, there is no tradition of eating raw snails, in contrast to the countries in southern Asia. This is why children are the primary victims of this disease in the Caribbean. The disease still continues to occur endemically in Cuba.

The clinical symptoms could confuse physicians because of the initial similarity to viral meningoencephalitis. The presence of eosinophilia in blood and cerebrospinal fluid (CSF) alerts the medical staff to suspect this disease. The best confirmation of the diagnosis is detection of *A. cantonensis* larvae surrounded by a cluster of eosinophilic cells in CSF (3). The pathological intrathecal synthesis of, e.g., immunoglobulin G (IgG) besides a change in the blood-derived fraction due to a blood-CSF barrier dysfunction.

The intrathecal immune response patterns and consequences for blood-CSF barrier function caused by parasites have not been described previously and deserve attention for diagnostic and theoretical, pathophysiological reasons.

MATERIALS AND METHODS

Patients. This prospective study included 24 pediatric patients (18 males and 6 females aged 3 to 14 years; mean age, 7.2 years) with acute meningoencephalitis who underwent lumbar puncture on suspicion of CNS infection. Informed consent for the lumbar puncture was given by the parents. The incubation period was 15 days. The clinical symptoms in all of the cases indicated meningoencephalitis. The most common symptom was fever (92%), followed by vomiting and headache. Detailed descriptions of the clinical symptoms and course of the disease were given in references 3 and 4. All of the cases in this study involved peripheral leukocytosis and eosinophilia (above 10%). The CSF cell differentiation showed 8 to 42% lymphocytes and 30 to 90% eosinophils. The frequency of worm detection in the lumbar CSF by an enrichment method previously described (2) was 30%.

The control group ($n = 15$) contained pediatric patients punctured after febrile convulsions to exclude an inflammatory process.

Samples. Serum and CSF were obtained simultaneously immediately after admission to the clinic during the acute phase, and a second puncture was done routinely 7 days later, at the time of clinical recovery.

Visitas de Trabajo 2000-2002

Presentación completa en esta dirección electrónica