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**Fatal Rabies Encephalomyelitis Despite Chick Embryo Vaccine Prophylaxis**

S.S. Sheth  
Varsha Gharpure  
Kalyani Nair  
U.B. Nadkarni  
C.T. Deshmukh  
A.P. Desai  
M.K. Jain  
M.D. Shah

Rabies encephalitis was one of the earliest known diseases, and because it is uni-

formly fatal, prevention of virus invasion is of great importance. With the introduction of the first rabies vaccine in 1885, post-exposure prophylaxis assumed a new dimension. The initial vaccines were all purified neural vaccines, but a high rate of adverse reactions led to the introduction of the duck and chick embryo vaccines and finally the human diploid cell vaccine in 1974. Concomitant administration of rabies immune globulin is necessary for complete protection(1,2), and up to 1985, no case of fatal rabies encephalitis following this combination, prophylaxis has been reported.

**Case Report**

A 12-year-old boy was brought with complaints of high grade fever with bodyache for 8 days. There was progressive difficulty in walking, and episodes of diplopia and delirium. He had been bitten on the right forearm, and scratched on the right buttock and left leg by a dog 25 days ago. The same dog had bitten several others but its fate was unknown. The wound had been cleaned with hydrogen peroxide and povidone iodine, and treated with silver sulfadiazine cream. He had also received chick embryo rabies vaccine on days 0, 3, 7, and 14. There was no history of seizures, focal deficits, aerophobia or hydrophobia.

Examination revealed stable vital parameters. All the wounds had healed with

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*From the Departments of Pediatrics and Pathology, Seth G.S. Medical College and K.E.M. Hospital, Parel, Bombay 400 012.
Reprint requests: Dr. M.K. Jain, Honorary Professor of Pediatrics, Seth G.S. Medical College and K.E.M. Hospital, Parel, Bombay 400 012.
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scar formation. The child was drowsy but easily arousable, and showed intermittent abnormal behavior with rambling and incoherent talk. The cranial nerves and pupillary reaction were normal. There was hypotonia with Grade II-III power in all limbs, and fasciculations were noticed in the hamstring and calf muscles. There were occasional jerky involuntary movements. The deep tendon jerks were depressed and plantars were equivocal. There was truncal ataxia and severe finger nose incoordination. The sensory system was normal but there was neck stiffness. The other systems were normal.

The hemogram was normal. CSF examination showed proteins of 55 mg/dl, with 110 RBC, 20 polymorphs and 80 lymphocytes per cu mm. Sugar and chlorides were normal. EEG revealed dysmodulated background activity at 3-5 Hz, with isolated paroxysms of sharp waves and large amplitude slow waves bilaterally—suggesting predominant white matter affection. Magnetic resonance imaging done subsequently showed patchy areas of altered signal intensity deep in the right parietal lobe involving the caudate and lentiform nuclei (Figs. 1a & b).

A tentative diagnosis of post-vaccination encephalomyelitis was made, and the child started on high dose corticosteroid therapy. He was also given mannitol, intravenous fluids and general supportive therapy. Anti-rabies human immunoglobulin (20 U/kg) was also administered.

Inspite of these measures, there was gradual deterioration, with loss of external ocular movements, unconsciousness, retention of urine and central respiration. The downhill course continued, and the child finally expired 9 days after admission. However, hydrophobia, aerophobia or spasms were not documented.

Fig. 1. Magnetic Resonance Image showing altered signal intensity in the right caudate and lentiform nuclei (A) and left hippocampal gyrus (B).
Post mortem revealed Negri bodies in the brain. Saliva which had been sent earlier was reported positive by the Rapid Rabies Immunodiagnostic Test (RRIT).

Discussion

This case is being reported because it presented with diagnostic as well as therapeutic problems. The child had received immediate attention after the bite, including prompt post-exposure prophylaxis by way of chick embryo rabies vaccination in the correct schedule. Therefore, when the child was brought, post-vaccination encephalomyelitis was suspected first. The child deteriorated, and though the clinical picture did not fit with that of classical rabies, when the salivary test and MRI were available, paralytic rabies due to failure of the vaccine was diagnosed.

The therapeutic implications of this case are open to discussion. Obviously, the post-exposure prophylaxis that the child received was inadequate to protect him but adequate to modify the course of the disease. There are many studies describing the failure of the sample vaccine(3), but there are an increasing number of reports of failure of the chick embryo(4) as well as human diploid cell(1,5) vaccines. Minor antigenic differences between the infecting and vaccinating strains could account for the variable degrees of protection(6). However, the authors have postulated that these patients may have had an underlying immune deficiency. Nevertheless, it is necessary to reevaluate the recommended dose, schedule, and route of administration of the various prophylactic measures available.

The efficacy of prophylactic measures depends on their accessibility to the virus, which is probably reduced once the virus is already within nervous tissue(7). It would therefore be of great value to administer rabies immune globulin both intravenously and by infiltration around the wound, as early as possible in all suspected cases. It is important to note that too large a dose would probably suppress the host's antibody response to the subsequently given doses of the vaccine. The recommended dose is 20 U/kg(8). Simultaneously, administration of the vaccine must be started. The Human Diploid Cell Vaccine (HDCV) is preferred to the Chick Embryo Vaccine owing to its greater rate of protection(9). Again, it should be pointed out that HDCV alone is not adequate(1,7), and rabies immune globulin must be given simultaneously for better immunization(2). However, the high cost and limited availability of this combination would be the limiting factors for its widespread use.

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REFERENCES

4. Progress in Rabies Control. Proceedings


Successful Treatment of Hepatic Hemangiomas with Corticosteroids

J.A. Padalkar
V.S. Bapat
M.A. Phadke
F. Ujjainwala

Corticosteroids are successfully employed to treat a patient with disseminated hemangiomatosis (cutaneous with large hepatic hemangiomas) which produce high output congestive heart failure. Children with this malformation may present with congestive cardiac failure and may have very high mortality if untreated. The present report presents a patient with this disease in whom therapy with corticosteroids was successfully employed.

Case Report

A 3-month-old female child was brought with the complaints of cough, fever, breathlessness, poor feeding for 7 days. There was a history of multiple hemangiomas present all over the body since birth.

On examination her pulse was 180/minute and respiratory rate was 62/minute with marked distress. The peripheral pulses were bounding. The cardiac examination showed Grade III systolic murmur at left upper sternal border. The liver was palpable 3 cm below costal margin and the upper border was present in the 6th intercostal space. There was no icterus. Investigations revealed normal liver function tests, X-ray showed a diffuse enlarged heart with increased pulmonary vascularity. The electrocardiogram was normal. Ultrasonography of abdomen revealed echolucent vascular deformities in posterior part of the liver. Close liver biopsy was not performed because of high vascularity of the tumor(1).

Computerized tomography scan (CT Scan) of abdomen showed entire right lobe

From the Department of Pediatrics, B.J. Medical College and Sassoon General Hospitals, Pune 411 001.

Reprint requests: Dr. Jyotsna A. Padalkar, Lecturer in Pediatrics, Department of Pediatrics, B.J. Medical College, Pune 411 001.

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