A 24-year-old male presented with back pain of one-month duration followed by sudden onset paraplegia with retention of urine. Neurological examination showed grade 0 muscle power in both lower limbs with loss of deep tendon reflexes and complete loss of sensation below T1, spinal segment. Complete blood count showed hemoglobin of 6.4 g/dl, TLC of 52,800/mm³ with 82% blasts and platelet count of 26,000/mm³. Bone marrow examination confirmed AML—M4. MRI spine showed soft tissue mass extending from T4-T9 with cord compression. He developed febrile neutropenia on day 14 of chemotherapy and expired.

Case 3: A 24-year-old male presented with back pain followed by weakness of both the lower limbs. Neurological examination revealed grade 3 muscle power with brisk deep tendon reflexes and bilateral extensor planter reflex. There was hypoaesthesia below T5 dermatome. Hemogram and spine radiogram was normal. MRI spine revealed epidural soft tissue mass extending from T4 to T6 with cord compression. Biopsy of spinal mass showed infiltration with blasts. Bone marrow revealed the diagnosis of AML—M4. He had complete neurological recovery following induction chemotherapy and radiotherapy.

Although the prevalence of GS is rare, factors such as certain chromosomal abnormalities t (8; 21), inv-16 and FAB subtype M2, M4 and M5 are associated with higher incidence of GS². All three of our patients were of AML-M4. Commonest site of cord involvement is thoracic spine. Spinal involvement in almost all of the cases reported has been of extradural in origin.³ Without the clue of circulating myeloblasts, the diagnosis of GS is usually delayed or not considered. Therapy of GS is not well defined because of rarity of cases. Therapeutic options available are chemotherapy, local irradiation and surgical decompression. GS are usually associated with poor prognosis even in otherwise favorable subgroup of AML patients.

Granulocytic Sarcoma of Spine: An Unusual Initial Presentation of Acute Myeloid Leukemia

Sir,

Reported incidence of Granulocytic Sarcoma (GS) in AML is 2.9 –3.1%.

1. However, granulocytic sarcoma of spine presenting with neurological symptoms as initial presentations of acute myeloid leukemia is rare.²³ We would like to report three cases of GS who presented with neurological manifestations and managed with combination of chemotherapy and local radiotherapy.

Case 1: A 24-year-old male presented with a history of weakness of all four limbs and retention of urine. Three weeks later he became anemic requiring blood transfusion and referred to our center. Examination revealed moderate pallor and sternal tenderness. Neurological examination showed the muscle power of grade 4 in all the limbs with extensor plantar reflex and no sensory deficit. His hemoglobin was 8 g/dl; total leucocyte count (TLC) of 17,000/mm³ with 16 % blasts. Bone marrow examination confirmed the diagnosis of AML – M4. Magnetic resonance imaging (MRI) of spine showed extensive extradural deposits at multiple levels (C4-C6, T2-T4, T8-T10, L1-L5 and sacral roots). He received local radiation and induction chemotherapy. Post-chemotherapy bone marrow was in remission and managed with neurological manifestations and managed with combination of chemotherapy and local radiotherapy.

Case 2: An 18 years old male presented with low back pain of one-month duration followed by sudden onset paraplegia with retention of urine. Neurological examination showed grade 0 muscle power in both lower limbs with loss of deep tendon reflexes and complete loss of sensation below T1, spinal segment. Complete blood count showed hemoglobin of 6.4 g/dl, TLC of 52,800/mm³ with 82% blasts and platelet count of 26,000/mm³. Bone marrow examination confirmed AML—M4. MRI spine showed soft tissue mass extending from T4-T9 with cord compression. He developed febrile neutropenia on day 14 of chemotherapy and expired.

References


reference to an epidemic of Chikungunya in Tamilnadu, particularly in Madras Metropolitan area in 1964. A large study of the disease was conducted by my colleagues and myself in Govt. Stanley Hospital in collaboration with the Virus Research Lab of ICMR at Pune. 242 subjects of the disease were studied in detail: In 86 subjects, there was lab confirmation by isolation of the virus from acute phase sera or rising titre of HI antibodies or both. In 35 serology was negative. In 121 subjects the diagnosis was clinical.

Besides the clinical profile of the current epidemic described in the review article, our study had shown that the disease could produce serious and sometimes fatal results.

Hyperpyrexia occurred in 2 cases, one of which ended fatally. Haemorrhagic manifestations occurred in 11.6% of laboratory proven cases and in 2.6% of the lab negative cases. None were fatal. Neurological complications occurred in 5 cases: Encephalitis with External Ophthalmoplegia, Polyneuropathy of the Guillaine Barre type in one case, transient Dysarthria in another case were observed.

Electrocardiographic changes suggestive of Myocarditis (excluding other causes like electrolyte abnormalities, hyperpyrexia etc) were seen in 3 out of 18 lab proven cases and 2 out of 37 lab negative cases.

The study had shown the possibility of the disease presenting with serious life threatening complications - besides the transient morbidity due to articular disease.

In a parallel study by the ICMR Virology unit from Pune and ourselves - a serological survey of asymptomatic subjects - 38.4% showed HI antibodies to Chikunguniya virus - indicating that a much larger population were infected, though had not developed the clinical disease.

Studies of haemagglutination inhibition tests on paired sera of febrile subjects showed 60.7% to positive for Chikunguniya. An interesting finding was that 6.9% of the sera showed positivity to Group B Barboviruses and in 4.8% to both Chikunguniya and Group B Viruses. This may be expected as both the viruses have a common vector for transmission.

This letter may supplement the observation of the authors of the review article, in which this study of the 1964 epidemic in Madras could have found a place.

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Sir,

In Andhra Pradesh, we are witnessing an unprecedented rush of cases of fever with acute severe polyarthritis and we are treating all of them as Chikungunya. Some of the cases have been serologically confirmed.

Now Chikungunya has become so common in this area (coastal Andhra Pradesh) not a single family has been spared. I would like to make the following comments based on my own experience of treating >150 cases of Chikungunya fever over the past 6 months.

The onset of illness is so acute that the patient is disabled within hours with severe crippling polyarthritis and high grade fever with rash. Most of the patients don’t respond to routine analgesics or NSAIDs. In this situation, I see no reason why steroids should not be given, particularly as immune complexes are said to be involved in the pathophysiology of Chikungunya arthritis. In fact, dramatic improvement is seen with steroids, especially with high dose IV Hydrocortisone. The dosage can be quickly tapered off and substituted with oral steroids.

Many patients are having recurrent acute arthritis. Most probably the virus triggers an autoimmune type of reaction with pts presenting symptoms mimicking acute rheumatic fever and rheumatoid arthritis.

Uric acid levels tend to be high in all these patients. We have given them a trial of Allopurinol with variable results.

Blood Widal test and VDRL are positive, 48 hrs after the acute symptoms, but quickly become negative after 1 week.

Use of Hydroxychloroquine in these patients has not been giving good results.

I would like to get views from all our colleagues in South India, treating Chikungunya fever. We find that it is more rampant than reported and as correctly mentioned by the authors, the epidemic is continuing unabated in Andhra Pradesh, both in the urban and the rural areas.

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