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Case Study

Neurological Challenges in Burkitt Lymphoma: A Case Report of Vincristine-Induced Neuropathy

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A B S T R A C T

Non-Hodgkin lymphoma-related immune-mediated neuropathies are uncommon and might be challenging to distinguish from neuropathies caused by other aetiologies. We present the medical and pathological data of a 16-year-old patient who had Burkitt-like cancer. Motor axonal polyneuropathy was suspected due to the neuropathy characteristics, which developed during chemotherapy induction with a cumulative dosage of 0.4 mg vincristine. Our theory is that the lymphoma-induced immunological mechanisms caused disruption to the peripheral nervous system (PNS), making it more susceptible to vincristine's harmful effects.

Keywords: Burkitt Lymphoma (BL), Non-Hodgkin Lymphoma (NHL), Motor Axonal Polyneuropathy, Vincristine (VCR), Peripheral Nervous System (PNS)

Introduction

The aggressive form of non-Hodgkin B-cell lymphoma is known as Burkitt lymphoma (BL). Epstein–Barr virus (EBV), human immunodeficiency virus (HIV), and chromosomal translocations that lead to the overexpression of the oncogene *C-MYC* are all linked to the illness. The World Health Organization (WHO) divides BL into three clinical categories: immunodeficiency-related, sporadic, and endemic. Malaria and EBV are associated with the endemic type. HIV and organ transplantation are both connected to the immunodeficiency-related variation. The disease's prognosis is great in children with intensive chemotherapy treatment, but dismal in adults.[1] BL with 11q aberration is recognised by the WHO as a new provisional entity in their classification of lymphoid neoplasms for 2016. This tumour lacks *MYC* reorganisation but looks like BL morphologically, phenotypically, and through gene expression profiling to a significant degree.[2] In wealthier nations, the overall cure percentage for BL is over 90%, whereas it is lower in low-income nations. Adults are less likely to get BL, and their prognosis is poorer.[1]

Case Report

A 16-year-old male from Rajkot in Gujarat was diagnosed with BL (non-Hodgkin lymphoma (NHL) in the small intestine) and admitted to the hospital, with symptoms including nausea and vomiting, loss of appetite, and quadriparesis. He was on a mixed diet and had no significant family or personal history. He had no social history of alcohol consumption or smoking.

Biopsy from enhancing omental nodules was morphologically adjudged as a malignant small round cell tumour, and subsequently diagnosed as NHL. PET-CT scan showed a 166 mm long segment involving the mid-to-distal jejunum with luminal narrowing. A perilesional small bowel mesenteric lymph node measuring 20*15 mm was also observed.

The maximum single wall thickness on the abdomen and pelvis CT scan was 36 mm, and the APP length of involvement was 23 cm. A few subserosal mesenteric deposits were observed extending along the superior mesenteric arteries, with the largest loops (APP 51 mm) measuring 24*23*53 mm.

During hospitalisation, the patient was treated with one cycle of injection rituximab (410 mg), injection etoposide (50 mg), injection doxorubicin (10 mg), injection vincristine (0.4 mg), and injection cyclophosphamide (750 mg), with other nutrients. For a speedy recovery, the patient was kept under constant observation.

The given three-chemo-cycle patient had toxicity of vincristine. During evaluation based on the Balis grade, the patient was found to be severe (grade 3), and it was found to be motor axonal polyneuropathy. Hence, the patient was diagnosed with vincristine-induced neuropathy. Other laboratory investigation showed: haemoglobin: 7 gm/dL (13–17gm/dL), RBC: 2.47*10^6 /cmm (4.5–5.5*10^6 /cmm), serum sodium: 127 mEq/L (135–145 mEq/L), serum total protein: 3.90 gm/dL (6–8 gm/dL), serum albumin: 1.90 gm/dL (3.5–5 gm/dL), and serum creatinine: 0.29 mg/dL (0.9–1.4 mg/dL).

The patient was treated with human albumin (20 mg), injection ceftriaxone (2 gm), injection chlorpheniramine (4 mg), and folic acid with other nutrients. For a speedy recovery, the patient was kept under constant observation.

Discussion

It is exceedingly uncommon for BL, a severe form of B-cell non-Hodgkin lymphoma, to develop in the nose and paranasal sinuses. It may exhibit a variety of symptoms, which might result in a delayed or incorrect diagnosis. While early detection and therapy can improve prognosis, BL is deadly if left untreated.[3]

EBV is linked to BL. Uncertainty surrounds the precise mechanism behind EBV and B-cell cancer. A latent EBV protein expressed in endemic BL is called EBNA1. The *EBNA3A-C* genes are expressed as a result of *EBNA2* gene loss. Since tumour cells produced from such cell lines are resistant to apoptosis, *EBNA2* may give the cancerous cells a survival advantage. BL is endemic in places like Brazil, Papua New Guinea, and equatorial Africa, where malaria is holoendemic. Studies revealed a connection between elevated *Plasmodium falciparum* antibody titres and endemic BL. HIV is linked with immunodeficiency-associated BL.[4]

The SEER (Surveillance, Epidemiology and End Results) database showed that between 2001 and 2006, there were 1,759 new cases of BL/ leukaemia per year in the United States, affecting people of all ages. The majority of these occurrences affected people between the ages of 20 and 64 years. About 0.4% of the total lymphoid malignancies were caused by this condition.[5]

Chemotherapy is typically the first course of therapy for BL. The GMALL-B-ALL/ NHL 2002 protocol and the altered Magrath regimen (R-CODOX-M/ IVAC) are only a couple of these regimens.[6] As with many tumours, the timing of the diagnosis affects how the chemotherapy works. Burkitt cancer is a fast-growing tumour, and as a result, it reacts more quickly than slower-growing tumours. A condition known as "tumour lysis syndrome" might develop as a result of the patient's fast response to treatment. Throughout the procedure, it is crucial to keep an eye on the patients and provide them with enough water. Given that BL has a strong potential for spreading to the CNS (lymphomatous meningitis), intrathecal treatment with methotrexate, ARA-C, and/ or prednisolone is administered in addition to systemic chemotherapy.[7] Chemotherapy includes cytarabine, ifosfamide, vincristine, methotrexate, cyclophosphamide, doxorubicin, etoposide, and rituximab. Immunotherapy, stem cell transplants, bone marrow transplants, tumour removal surgery, and radiation are other therapies for BL.[8]

Acute lymphoblastic leukaemia, lymphomas, rhabdomyosarcoma, neuroblastoma, and nephroblastoma are just a few of the paediatric malignancies that are commonly treated with VCR, which is an essential drug in combination chemotherapy regimens. Vincristine-induced peripheral neuropathy (VIPN), which results in peripheral and primarily symmetric sensory-motor neuropathy, is the primary adverse effect of VCR. VIPN can limit dose, prolong treatment cycles, and even force chemotherapy to be stopped.[9] Neuropathic pain, tingling and numbness in the feet and hands, weakness in the muscles, areflexia, and changed gait are a few of the clinical signs of VIPN. Additionally, it may harm autonomic nerves and impair vision and hearing.[10]

Conclusion

This case highlights the difficulty in forming differential diagnoses of acute quadriplegia in NHL patients receiving treatment. Although the precise pathophysiological causes in this instance could not be identified, the most probable diagnosis, according to the clinical presentation and the results of the bilateral upper limb and lower limb nerve conduction study, was motor axonal polyneuropathy. It is intriguing to consider whether a weakened immune response in the setting of BL and chemotherapy may have contributed to the peripheral nervous system's heightened immunological vulnerability. However, it is possible that a distally enhanced "dying-back" pattern of loss of axons at autopsy reflects the toxic effects of VCR, even at low doses, overlaid on peripheral nerves.

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