Sensory complaints of our patient preceded the diagnosis of cancer and neuropathy by 2 months. Nerve conduction studies showed a greater extent of asymmetrical involvements of the sensory conductions than of motor conductions. Neuropathy of this sort is most commonly encountered in patients with diabetes mellitus and autoimmune-mediated vasculitic neuropathy. However, our patient did not have diabetes mellitus based on normal blood glucose tests. Other causes of asymmetrical neuropathy or mononeuropathy multiplex are immune-mediated vasculitis and entrapment injury of nerves. A dozen causes are known to induce vasculitic neuropathy. A few them are polyarteritis nodosa, systemic lupus erythematosus, rheumatoid arthritis, Churg-Strauss syndrome, and Wegerner granulomatosis. None of these are likely to be the cause of the neuropathy in our patient. Entrapment of the nerves at the ulnar notches and carpal tunnels could have occurred in our patient with prominent osteosclerotic bone change. However, proportionate involvement of the motor and sensory components of nerves is more typical in entrapment neuropathy. Our patient showed more-prominent involvement of sensory fibers than motor fibers, and that led us to consider another possible association with a paraneoplastic syndrome. Sensory neuropathy in association with systemic cancer is a well-known paraneoplastic syndrome. Circulating autoantibodies may cross-react with myelin-associated glycoprotein (MAG) in patients with paraneoplastic sensorimotor neuropathy. Despite lacking direct evidence from a nerve biopsy or measurements of circulating autoantibodies, a more-prominent slowing of the sensory conductions than motor conductions in some nerves (Table 1) on one hand, and a lack of any other better explanation for the causes of neuropathy in this specific patient on the other hand, led us to consider the possibility of a paraneoplastic neuropathy. Moreover, slowing of the SNCVs, and delayed distal motor latencies and sensory latencies of the median and ulnar nerves were more or less asymmetric. However, the discrepancy in SNCV of the bilateral sural nerves was less significant. We suggest that electrophysiological studies of small-fiber sensory system, such as thermal perception thresholds[12] may be more valuable than nerve conduction studies in elaborating sensory neuropathy as the latter measures only the fastest conducting fibers of nerves.

Markedly elevated serum alkaline phosphatase with a low serum level of calcium, normal parathyroid hormone, and x-rays of the skeleton showing a diffuse increase in osteoblastic bone formation suggest the diagnosis of either Paget's disease, metastatic lymphoma, or metastatic prostate cancer. A previous report[13] showed that radiographically malignant or sarcomatous degeneration in Paget disease may show osteolytic lesions with sclerotic or thickened trabeculae in the bones. A more common osteolytic metastatic lesion of the spine may manifest clinically as spinal cord compression syndromes.[14] Osteosclerosis of the skeleton may also occur in plasma cell dyscrasia with polyneuropathy and POEMS syndrome.[15] This was not the case in our patient.

Finally, multiple cranial neuropathy with leptomeningeal-enhanced lesions may also occur in conditions other than leptomeningeal carcinomas. A biopsy-proven case of a limited form of Wegener granulomatosis,[16] and an autopsy-proven case of primary multifocal leptomeningeal gliomatosis have been reported.[17] All of these settings must be included in the differrential diagnosis. In conclusion, bilateral multiple cranial neuropathies with optic nerve involvement and CRVO, progressive hearing loss, bilateral abducens palsies, and sensorimotor neuropathy (possibly paraneoplastic) are rare in prostate cancer with leptomeningeal metastasis. The diffuse osteosclerosis of the bones resembles Paget's disease. Advanced prostate cancer with leptomeningeal carcinomatosis must therefore be considered in a middle-aged or elderly man with symptoms of prostatism, bone pain, limb numbness, signs of increased intracranial pressure without lateralized neurological deficits, progressive bilateral hearing loss, and subacute progressive blindness.

REFERENCES

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