tate cancer with a huge meningeal metastatic tumor may occur.[5] We herein report on a patient with bilateral optic neuropathy and hemorrhagic central retinal vein occlusion, bilateral hearing loss and lateral gaze paresis, sensorimotor neuropathy, diffuse osteosclerosis of the skeleton and skull bones, and cervical lymphadenopathy due to metastatic prostate cancer. We address the multiple mechanisms leading to the progressive and permanent bilateral blindness, hearing loss, lateral gaze palsy, and limb numbness.

CASE REPORT

A 59-year-old man presented with subacute onset of progressive loss of bilateral vision for 2 weeks. The visual disturbance first occurred in the left eye in late October 1999, and several days later, in the right eye. He also complained of numbness of both hands and all toes for 2 months since September 1999. He noticed excessive body weight loss from 59 kg in February 1999 to 48 kg in November 1999. He had pain in the bilateral shoulders and right knee for 5 months. Increasing pain in the neck and bilateral hip joints developed a few days prior to admission. Urinary frequency, voiding hesitancy, and terminal dripping were noted for several months. There was neither hematuria nor dysuria. He had had hypertension for 2 years. He denied having headache, vomiting, or fever in the past few months. Physical and neurological examinations at admission revealed that his blood pressure was 230 mmHg systolic, and 130 mmHg diastolic. The body temperature was 36.5 °C, heart rate was 72 beats/minute, and respiratory rate was 16/minute. He was oriented to time, place, and person. He had very poor vision with only light perception in both eyes. Marcus-Gunn pupils were present, being more prominent on the left side. Ophthalmoscopic examinations showed bilateral papilledema with peripapillary nerve fiber layer hemorrhages and venous congestion. The macula appeared edematous. Bilateral abducens palsies with bidirectional nystagmus was striking when he was performing conjugate eye deviation to either side. His hearing acuity was remarkably impaired in both ears. His neck was supple. There was an enlarged cervical lymph node, 1.5 cm in diameter, in the right cervical area. Virchow's node was not palpable. There was mild kinetic tremor of both hands in an outstretched position and on action. The muscle power was generally graded 4 of 5. Muscle tone was normal, and there was no fasciculation. There was no limb or truncal ataxia. Tendon jerks were diminished in the distal parts of all limbs. Babinski sign was absent. Pinprick and thermal sensations were impaired in both hands and feet in a stocking-and-glove distribution. Joint and vibration sensations were relatively preserved. Physical examinations of the chest and abdomen were unremarkable. There was a knocking pain over the thoracolumbar vertebrae and sacrum. Patrick sign was positive on both sides. A digital rectal examination (DRE) showed an enlarged prostate gland with a smooth surface and a firm consistency. A hemogram showed hemoglobin of 8.7 gm%, hematocrit of 27.8%, and a white blood cell count of 5030 mm^3 (segmented form 89.8%, lymphocytes 7.0%, monocytes 1.6%, basophils 1.0%, and eosinophils 0.6%). Urine Bence-Jones protein was negative, and serum protein electrophoresis was unremarkable. The Westegren erythrocyte sedimentation rate was elevated to 80 mm/h. Blood C-reactive protein was 6.74 mg/dl (normal, < 0.8 mg/dl). Serum alkaline phosphatase was remarkably elevated at 1621 U/L, and uric acid was 9.1 mg/dl. The serum level of prostate specific antigen measured 228.5 ng/mL. Plasma glucose was 94 mg/dl, serum albumin was 3.3 g/dl, and total protein was 6.5 g/dl. Serum protein electrophoresis remained normal. The liver function (SGOT 36 U/L, GPT 19 U/L) and renal function tests (BUN 22 mg/dl, creatinine 0.6 mg/dl) were normal. The blood calcium level was 7.7 mg/dl, phosphorus 3.5 mg/dl, potassium 3.7 meq/L, sodium 140 meq/L, chloride 110 meq/L. Parathyroid hormone PTH-C was 1.0 ng/mL. Antinuclear antibody (ANA) was positive at a 160-fold dilution (speckled type). The flash visual evoked potential (VEP) showed an absence of all responses bilaterally. Nerve conduction studies showed sensorimotor neuropathy with more-prominent slowing in the sensory nerve conduction velocities (SNCVs) of the left median and left ulnar nerves (Table 1). The slowing of SNCVs was more or less asymmetric in the upper