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Letter to the Editor

The Involvement of the Central Nervous System in Multiple Myeloma must be Demonstrated through Appropriate Studies

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Letter to the Editor

We reviewed the article by Melchiori *et al.* on a 67-year-old woman with IgA lambda multiple myeloma, which initially manifested as osteolytic bone lesions and cytogenetic abnormalities, was treated with bortezomib, lenalidomide, steroids, and autologous stem cell transplantation, and relapsed with lesions in the lungs, pleural mediastinum, and presumably the central nervous system (CNS) [1]. Despite expansion and maximization of therapy, the myeloma progressed and the patient died [1]. The study is interesting, but some ambiguities need to be clarified.

The first point is that the diagnosis of CNS involvement was based on suspicion, but was only supported by MRI without other objective findings [1]. Neither cerebrospinal fluid (CSF) examination nor brain biopsy was performed. To confirm intracerebral or meningeal involvement in myeloma, it would have been crucial to document the deposition of paraprotein or plasmablastic plasma cells in the brain or meninges. It was also not reported whether fluorodeoxyglucose positron emission tomography (FDG-PET) also included the brain and whether or not it showed cerebral hypermetabolism [1]. Only venous sinus thrombosis (VST) and hypophysitis were suspected. However, the pituitary gland was described as normal on MRI [1]. An MRI with contrast agent does not sufficiently rule out immunological or infectious encephalitis.

The second point is that the suspicion of VST was not confirmed by appropriate imaging tests [1]. To confirm VST, it would have been essential to perform computed tomography venography (CTV) or magnetic resonance venography (MRV) with contrast medium. One argument against VST is that the patient clearly did not show any symptoms of VST. VST usually manifests itself in headaches, seizures, and motor and sensory disturbances [2].

The third point is that no autopsy was performed [1]. In order to confirm or rule out suspected CNS involvement, it would have been crucial to at least examine the patient's brain through an autopsy [3]. To assess whether the patient actually suffered from hypophysitis, histological and immunohistological examinations of the pituitary gland and hypothalamus would have been useful.

The fourth point is that the suspected VST was attributed to myeloma, but the side effects of the therapy used for the malignancy were not sufficiently ruled out [1]. Since there is evidence of an increased prevalence of venous thrombosis, including VST, with the combination of lenalidomide and steroids [4], it cannot be ruled out that the suspected VST was due to a side effect of the therapy used rather than the myeloma.

The fifth point is that there is no indication of what the "endocrine laboratory" comprised [1]. Of particular interest is whether the pituitary hormones (growth hormone, thyroid-stimulating hormone, adrenocorticotropic hormone, follicle-stimulating hormone, luteotropic hormone, prolactin) were elevated or decreased or were within normal ranges.

The sixth point is that the patient did not undergo a thorough cardiological examination [1]. Since the patient complained of exertional dyspnea and chest pain and had elevated troponin levels, and since myeloma can affect the heart [5], it would have been crucial to rule out Takotsubo syndrome, myocarditis, pericarditis, endocarditis, heart valve dysfunction, and myeloma involvement. We should know whether the echocardiography showed reduced systolic or diastolic function or pulmonary hypertension.

The seventh point is that the time course was not clearly stated [1]. Of particular importance is how long the patient received initial treatment and after what period of time the relapse developed. We should also know whether the initial treatment was tolerated with or without major side effects.

In summary, suspected CNS involvement in multiple myeloma must be confirmed by CSF examination, FDG-PET, biopsy, or autopsy of the brain.

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