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Case report

Intracranial multiple myeloma may imitate subdural hemorrhage: How to overcome diagnostic limitations and avoid errors in treatment☆

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ABSTRACT

Background: Although the diagnosis of subdural hematoma is usually straightforward, occasionally it may be erroneous, leading to mistakes in the treatment. For example, leptomeningeal malignancies, even in the absence of bleeding, may clinically and radiologically mimic subdural hemorrhage.
Objective: To stress the importance of not only intuitive thinking but also in analytic thinking in appropriate and accurate treatment strategies.
Methods and Illustrative case: In this report, the clinical and radiological pitfalls in differentiating malignant leptomeningeal infiltration and subdural hematomas are discussed. A sample case of an intracranial extra-osseous manifestation of a multiple myeloma that is atypical with regard to its location and clinical presentation is presented for illustration.
Conclusions: The variability of intracranial presentation and the wide spectrum of leptomeningeal malignancies necessitate careful preoperative evaluation of the patient's individual history as well as radiological images to avoid misdiagnosis. A clinician who has become familiar with the pitfalls in the differential diagnosis between leptomeningeal infiltrations and subdural hematoma will act more analytically to solve the patient's problems properly and avoid potential complications for the patient.

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1. Introduction

Subdural hematomas are frequently considered as neurosurgical emergency cases [1]. Although the diagnosis of subdural hematoma is usually straightforward, occasionally it may be erroneous, leading to flawed treatment. For example, leptomeningeal malignancies, even in the absence of bleeding, may clinically and radiologically mimic subdural hemorrhage of any stage, such as acute, subacute or chronic [2–5]. The misinterpretation of morphological-neuroimaging features can lead to inappropriate surgical decision-making and consequently to a suboptimal neurosurgical and oncological approach. In cases of acute subdural hematoma, urgent surgery is the treatment of choice, whereas in cases of leptomeningeal neoplastic infiltration with subdural tumor progression in patients with extensive cancer, the same strategy may worsen the course of the disease [6]. Moreover, urgent implementation of the oncological therapy may thus be delayed. Furthermore, in the surgery of chronic subdural hematoma, one or two burr holes and the drainage of blood may be adequate, whereas, in cases of tumoral lesions the aim would either be a large craniotomy, intra-operative biopsy and frozen section examination of the biopsy specimen and gross total removal of the tumor or a biopsy alone, depending on the underlying pathology.

Intracranial manifestation of a multiple myeloma is very rare and represents less than 1% of all intracranial tumors [7,8]. Because of the aggressiveness and invasiveness of myeloma cells, intracranial involvement may be dural, leptomeningeal or intraparenchymal, or manifest in skull bone, as well as in cranial nerve lesions [2–5,8–11]. Leptomeningeal and dura mater involvement in multiple myeloma is estimated to be even more rare [3,12,13]. Extra-osseous neoplastic hemispheric intracranial lesions may be confused with a subdural hematoma, especially in a computed tomography (CT) image, in which the hypercellularity of the lesion may appear as a hyperdense, crescent-shape, extra-axial collection and thus imitate an acute hemorrhage [2,14].

In this report, clinical and radiological pitfalls in differentiating malignant leptomeningeal infiltration and subdural hematomas are discussed. A sample case of an intracranial extra-osseous manifestation of a multiple myeloma that is atypical with regard to its location and clinical presentation is presented for illustration.

2. Illustrative case

A 62-year old woman was diagnosed with a multiple myeloma IgG, type Lambda, Durie–Salmon, Stage II, four years ago. Initially, the manifestations of the disease were multiple osteolytic lesions in the vertebrae. In the course of the disease, an extra-osseous progression appeared and one year later switched to the stage III. After a chemotherapy regime, including lenalidomide, pomalidomide, and finally autologous stem cell transplantation, partial remission of the disease was achieved. The further course of the disease was interpreted as stable. One year later there was a relapse of the disease, and consequently a multimodal oncological approach with re-induction therapy including lenalidomide, bendamustin, pomalidomide and skeletal radiation was implemented.

Six months later, the patient was admitted into hospital because of a general weakness, somnolence, and a novel moderate, left-sided hemiparesis. Glasgow Coma Scale (GCS) score of the patient was 11 (eye opening to speech 3, verbal response with inappropriate words 3, best motor response; localizing pain, 5).

A CT scan showed a right-sided, extra-axial, hemispherical, hyperdense lesion with a secondary mass effect displacing the gray-white matter interface medially, and midline shift (Fig. 1), which was interpreted as an acute subdural hematoma. Additionally, a preoperative laboratory examination showed pancytopenia with a low thrombocytes level. Because of the clinical symptoms and the radiological significant mass effect of the extra-axial lesion, the decision to perform an emergency evacuation was made.

Intraoperative features of intracranial presentation of multiple myeloma. Intraoperatively, unexpectedly, instead of a hematoma, tumor tissue was found. A frozen section was possible during emergency surgery, and was made but the final results of examination were not waited, because the main aim of the operation was to remove the mass lesion, decompress the cerebral tissue and avoid herniation. The initial craniotomy had to be enlarged with the aim of complete tumor resection. The tumor mass appeared smooth, lobulated and well vascularized. Invasion of the calvarium appeared as an irregular erosive, osteolytic destruction. The infiltrated dura was hypervascularised, thickened and inseparable from an infiltrated arachnoidea. Consequently, a subdural space did not exist, being broadly filled by an infiltrative tumor process. Microscopically, the intradural mass, infiltrating the arachnoidea and pia, complicated the plane dissection, separation and removal of the tumor without causing damage to the superficial cortical neurovascular layer. Even minimal manipulations resulted in bleeding from the fragile pathological neoplastic neo-vessels of the pia and cortical superficial microvessels leading to cortical micro-contusions. Despite this, the lesion could be removed near gross totally.

Histopathological examination proved the lesion to be an intracranial manifestation of multiple myeloma (Fig. 2). Microscopically, there were fibrovascular septae throughout the tumor mass, variable tumor-cells, cells with eccentrically shaped large nucleoli, a high mitotic rate and additionally, apoptotic cells. Immunohistochemically, the tumor cells showed a markedly positive CD 138 profile. There was no reaction using the CD20 antibody. Sporadic kappa–light chains and a significantly high level of positive lambda chains were seen. The proliferation rate (Ki67) was 80–90%.

Postoperatively, the immediate clinical condition of the patient was satisfactory. She recovered complete consciousness. Postoperatively, GCS score of the patient was 15 (eye opening; spontaneous 4, verbal response; orientated 5, best motor response; obeying commands 6, apart from hemiparesis). The left sided hemiparesis improved markedly. Oncological therapy and radiotherapy of the cranium was indicated because of achieving subtotal tumor resection, and begun immediately after the wound healed. Unfortunately, the radiotherapy had to be interrupted because of impairment to the wound healing. Simultaneously, the patient developed a

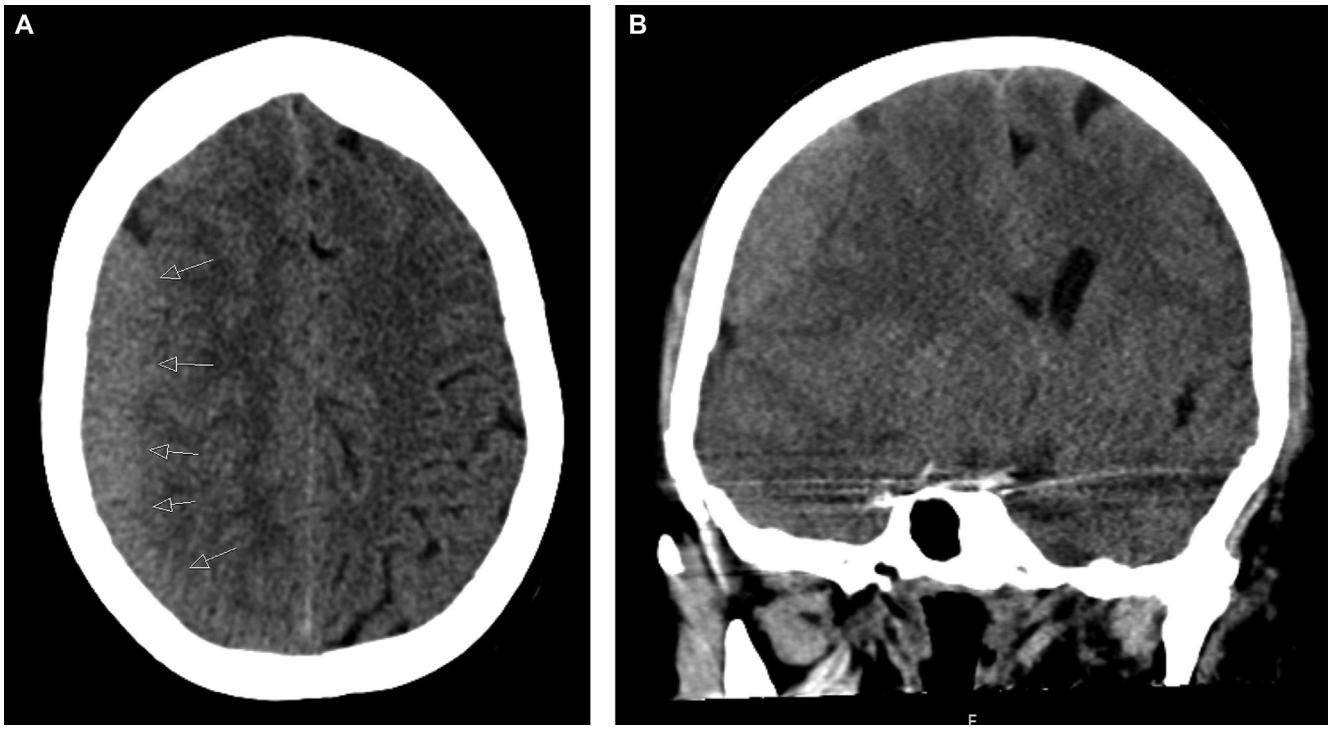

Fig. 1 – (A) Pre-operative axial, non-contrast CT (soft tissue window) demonstrates a right-sided fronto-parietal, slightly hyperdense subdural collection of plasma cell myeloma, initially thought to be an acute subdural hematoma. (B) Coronal view showing the same subdural lesion with secondary mass effect. A midline shift is also seen.

cytomegalovirus (CMV) induced pneumonia as a result of a suppressed immune system. Although intensive, antiviral therapy and supportive care were implemented, the patient's condition worsened rapidly. Two months after neurosurgical intervention the patient died.

3. Discussion

3.1. *Clinical differential features between intracranial multiple myeloma and subdural hematoma*

A careful and detailed evaluation of the patient's history, the course of disease and its dynamics, is crucial and mandatory for accurate image interpretation and appropriate treatment strategy. Clinical history, symptoms and signs may be misleading in the differential diagnosis between subdural hematoma and leptomeningeal malignant lesions. Leptomeningeal malignant infiltrations are mainly seen in advanced stages of the cancers of the prostate, breast and lung, in sarcomas and in hematologic malignancies such as multiple myeloma and lymphomas [2,5,15–25] (Table 1). Previous to intracranial involvement and its clinical manifestation, there is usually a prolonged history over several years of the primary malignant lesion solely. Nevertheless, a sporadic primary intracranial involvement as a first manifestation without peripheral manifestation of the tumor lesion has been reported in the literature [26].

In the case of intracranial metastatic involvement, the spread of tumor cells is possible by many paths, including the hematogenous route, breaking through the blood-brain-barrier, or directly from adjacent tissues, or finally, via cerebrospinal fluid space, by diffuse circulating myeloma cells [14]. However, there may be no history of malignancy and leptomeningeal infiltration during the initial presentation of the disease [26]. Furthermore, if the patient develops some focal neurological deficits and there is a history of falls or head trauma, a speculative and inaccurate diagnosis of intracranial bleeding can easily be made. Finally, a patient with diagnosed intracranial leptomeningeal involvement may simultaneously develop a subdural hemorrhage independently after a traumatic episode [27]. Moreover, this group of cancer patients seems to be more predisposed to develop of subdural hemorrhage because of the fragility of the neo-vasculature of the infiltrated leptomeningeal layer. Additional factors leading to increased tendency of bleeding in the presence of multiple myeloma, such as interference of myeloma-produced antibodies against clotting factors, or amyloid damage of the endothelium and platelet dysfunction certainly may play a significant role in the imbalance of hematological homeostasis [39]. However, the definite mechanism of hematological malignancies associated with intracranial hemorrhage has not been clearly defined and the exact mechanisms of these coincidence are still under investigation [27].

In the preoperative stage of clinical decision-making, the dynamics of developing clinical symptoms should be carefully evaluated. Clinical findings of acute SDH can vary from none to severe neurological deficits including coma. Most patients with acute SDH rapidly develop consciousness deterioration and are low on the Glasgow Coma Scale on admission.

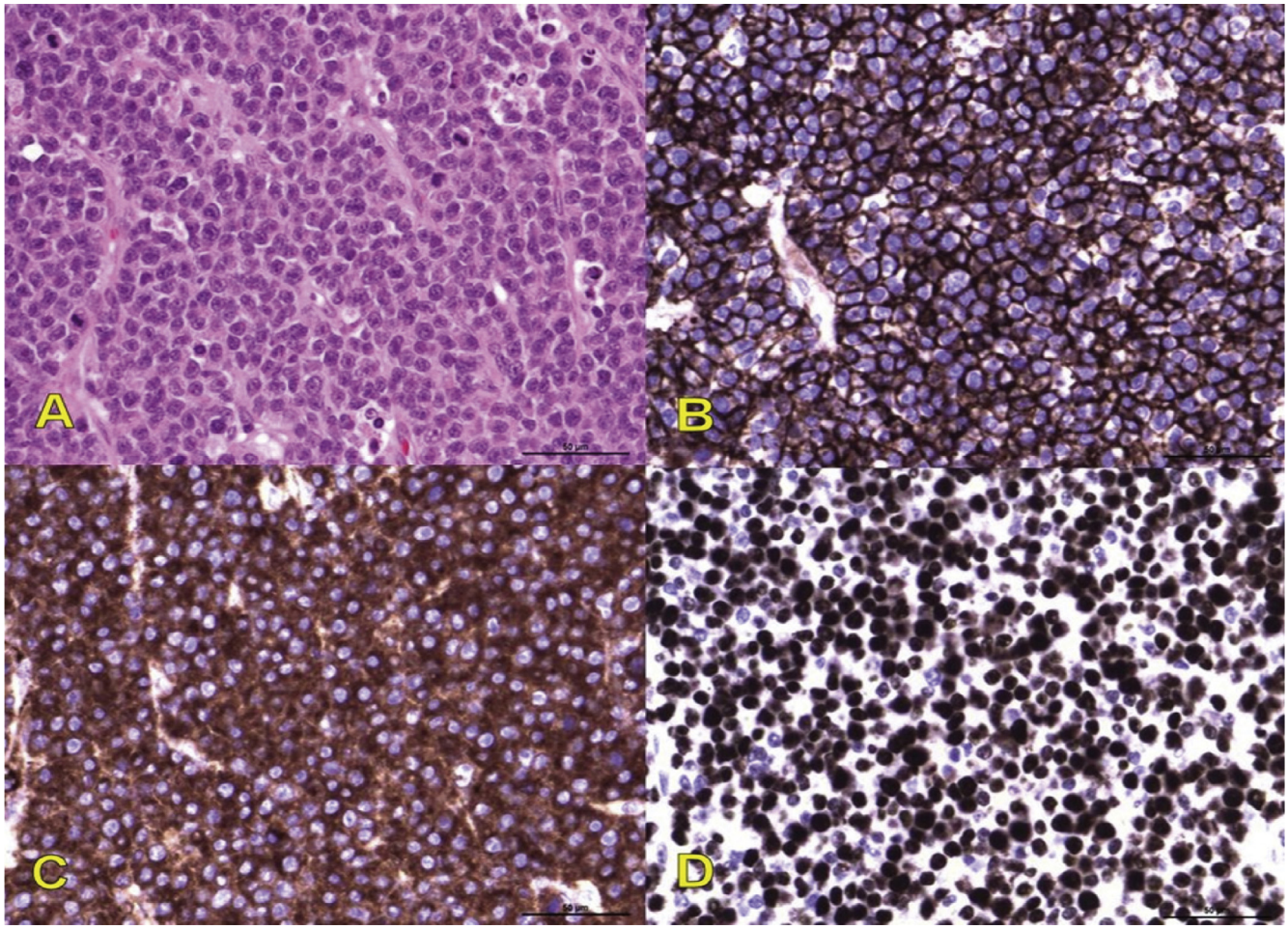

Fig. 2 – Histopathological (A), Immunohistochemical analysis (B, C, D) and features of the tumor. (A) HE; tumor mass with large neoplastic cells with large oval and eccentric lied nuclei divided by delicate fibrovascular septae (B) CD 138 is diffusely expressed in the cell membrane of the tumor cells, (C) Lambda-light chain is prominently expressed, (D) The proliferation rate (Ki67), is estimated to be approximately 80–90%.

Nevertheless, delayed deterioration, especially in elderly anticoagulated patients is common [28]. Acute subdural hemorrhage with or without leptomeningeal neoplastic involvement develops rapidly in most cases and through constantly increasing mass effect leads to life-threatening brain herniation, clinically manifesting as progressive neurological deficits with anisocoria and unconsciousness [29]. In these cases, the classical emergency neurosurgical approach aims at the volumetric reduction of the hematoma and thus a decrease in intracranial pressure as the treatment of choice. Additionally, clinical diagnosis may be complicated by nonspecific neurological and vegetative symptoms, such as

Table 1 – A literature review regarding errors in treatment associated with the differential diagnosis between subdural hematoma and leptomeningeal tumoral lesions.

Author	Year	Pathology	Errors
Oertel [19]	2003	Spindle cell sarcoma	Twist drill craniostomy
Schluterman [5]	2004	Multiple myeloma	Preoperative misdiagnosis, possible mistake in surgery
Evliyaoglu [25]	2006	Non-Hodgkin's lymphoma	Inadequate bony exposure at the beginning of surgery
Tsang [20]	2006	Multiple myeloma	Bur hole, possible delay in CT and RT
Yoon [18]	2008	Non-Hodgkin's lymphoma	Bur hole in the beginning of surgery
Cheng [21]	2009	Metastatic adenocarcinoma of the prostate	Inadequate bony exposure at surgery
Ramnarayan [24]	2013	Non-Hodgkin's lymphoma	Two operations, no biopsy in the first one
Mathon [22]	2013	Burkitt lymphoma	Surgical technique: Bur hole
Boukas [15]	2015	Metastatic adenocarcinoma of the prostate	Repeated bur hole drainage, biopsy in the second surgery
Nzokou [17]	2015	Metastatic adenocarcinoma of the prostate	Surgical technique: Bur hole, no frozen section

headache, vomiting, nausea and lethargy, which may indicate an increased intracranial pressure related to rapidly increasing mass effects in acute subdural hematomas. However, these symptoms are often observed in leptomeningeal carcinomatosis without a mass effect [30]. Moreover, metabolic side effects of multiple myeloma even without intracranial manifestation may lead to neurological deterioration; attendant hypercalcemia may produce lethargy, weakness or confusion. Hyperviscosity of blood intensifies headache and fatigue, leads to visual disturbance and causes retinopathy [31]. Summarizing, there are no absolute pathognomonic, characteristic clinical symptoms neither for a SDH, nor for intracranial and leptomeningeal myeloma involvement, which are sufficient and infallible at the stage of clinical diagnostic decision making.

From a biological and hematooncological point of view, the multiple myeloma represents a malignant proliferation of plasma cells derived from a single clone and belongs to a group of B-cell lymphoproliferative diseases [31,32]. Multiple myeloma is a neoplasm of a single clone of well-differentiated plasma cells. It is characterized by a proliferation of plasma cells in the bone marrow, predominantly involving the ribs, sternum, spine, skull and proximal extremities, and the infiltration of adjacent tissues by mature and immature plasma cells [33]. In contrast, a plasmocytoma is a solitary neoplasm of monoclonal plasma cells. In the course of tumoral activity and biological behavior, a plasmocytoma may progress to its disseminated, malignant form, namely multiple myeloma [11]. Although the initial predilection is involvement of spinal vertebrae, osseous lesions in the skull base, nose, and paranasal sinuses are reported [26,28]. The latter is thought to be a strong positive predictor for progression from solitary plasmocytoma to multiple myeloma.

The intracranial presentation of a multiple myeloma has a wide variety of imaging manifestations, ranging from lesions with an extra-axial, focal or diffuse meningeal enhancement to solid or diffuse intra-axial, intraparenchymal nodules or masses mimicking other intracranial tumors [7,28,34]. Intracranial involvement and unusual locations for example, skull base involvement with myelomatous infiltration and secondary hypoglossal nerve palsy has been described previously [9,11,28,35–37]. Other unusual locations, such as the involvement of the orbit, cavernous sinus, Meckel's cave, sella turcica, petrous bone or clivus have also been reported [26,28,38,39]. Some rare presentations, such as myelomatous meningitis with diffuse meningeal thickening, may exist with intraparenchymal lesions [10].

Intracranial progression of tumor masses can be a result of the invasion of tumor cells arising from the calvaria or the skull base [14]. Since the dura mater is relatively avascular, dural involvement is believed to be a consequence of direct spread of the involved bone lesion. On the other hand, primary dural involvement is extremely rare [3,12,13]. Leptomeningeal spread can occur hematogenously as focal, multifocal or diffuse [14]. Roddie and co-workers studied myelomatous involvement of the dura mater in patients with multiple myeloma [3]. In samples taken in autopsy, in patients with leptomeningeal involvement, they found circulating myeloma cells which diffusely infiltrated arachnoid veins, consequently leading to the destruction and occlusion of arachnoid trabeculae. Through this path, further spread into the cerebrospinal fluid may be possible.

3.2. *Radiological differential features of intracranial multiple myeloma and subdural hematoma*

Apart from the patient's history and clinical examinations, radiological studies may also be misleading. If clinical findings lead to a diagnosis of an intracranial hematoma, then the routinely preferred radiological examination is usually a non-contrast CT scan solely [32]. Even in patients with a history of cancer, additional neuro-imaging modalities are usually not ordered in urgent cases with a subdural lesion diagnosed by the initial CT scan. On the other hand, the preferred radiological examination in cases with an intracranial tumoral lesion is magnetic resonance imaging (MRI) with and without contrast medium administration. Leptomeningeal tumor infiltrations can be seen as hyperdense lesions in CT scans, even without any obvious bleeding [5,14].

Radiologically, intracranial multiple myeloma can present in various forms; from a solid and well-limited lesion to diffuse infiltrating mass lesions [14,28,40]. From a radiological point of view, the CT depicts lesions that involve cortical bone, and appear as well defined lytic, destructive and erosive lesions without a sclerotic border, involving the diploe and cortical bone. The extra-axial mass generally respects the border of brain tissue. MRI shows extra-osseous, intracranial involvement. Typically, the lesions appear isodense to hyperdense on CT, isointense to hyperintense on T1-weighted MRI and markedly hypointense on T2-weighted MRI. Notably, hyperdensity on CT modality as well as a hyperintensity on T1-weighted MRI suggest a high cellularity and a low nucleocytoplasmic ratio. Because of dense vascularity, after administration of contrast media the lesion is markedly enhanced. The multiple myeloma shows increased diffusion-weighted images [23]. If surgery for an intracranial multiple myeloma is intended it is important to perform both non-contrast CT and MRI with and without contrast medium administration preoperatively, the former to delineate osseous involvement and the latter to demonstrate the soft tissue, myelomatous involvement of meninges or brain parenchyma.

3.3. *How to provide individual treatment*

The treatment strategies for multiple myeloma in patients with systemic manifestation are clearly defined [31]. In contrast, there is a lack of guidelines for the treatment of progressed stages of multiple myeloma with intracranial involvement.

From the hemato-oncological point of view, multiple myeloma in its phase of disseminated malignancy of plasma cells represents the final stage of the disease progression [26,39]. The stage of the disease involving dural and leptomeningeal compartments correlates with a very poor prognosis and should be considered a distinct complication of myeloma disease [3]. This is of critical importance in preoperative treatment strategy decision-making. As in the case presented here, the extremely poor prognosis of patients with myelomatous involvement of the meninges is estimated to be on average approximately eight weeks from the onset of

neurological symptoms to death [3]. Based on the above facts, in our opinion, it is essential that neurosurgical intervention and neurosurgical treatment regimens in the extensive stage of multiple myeloma with intracranial involvement should be individually indicated. In cases of suspected subdural hematomas in patients with diagnosed multiple myeloma, where possible, a preoperative MRI scan should be performed to differentiate the tumor mass from the hematoma. In emergency cases with a computer tomogram diagnosed life-threatening mass effect of intracranial lesion, an emergency craniotomy with aim of reducing the mass effect and evacuating the lesion should be performed. On the other hand, for patients with intracranial involvement, who are neurologically stable, apart from detailed focused on intracranial and neuroaxis MRI findings, an immunohistochemical, morphological, cytogenetic profile of the myeloma should be evaluated and systemic co-morbidities taken into account, with aim of implementing an individual therapy. Various multimodal treatments options for intracranial myelomatous involvement have been proposed, including cranial radiotherapy, intrathecal chemotherapy and systemic chemotherapy, but in general responses are partial and short-lived [3].

4. Conclusion

The consequences of erroneous diagnosis can be fatal. Unnecessary surgery may worsen the prognosis of patients with a leptomeningeal malignant lesion without any intracranial bleeding, during the final stage of the disease. The variability of intracranial presentation and the wide spectrum of leptomeningeal malignancies, varying from focal to diffuse, from extra-axial to intra-axial, from cranial osseous to meningeal and intraparenchymal, necessitate careful preoperative evaluation of the patient's individual history as well as of radiological images to avoid misdiagnosis. A neurosurgeon or a trauma surgeon should be very cautious in the diagnosis of subdural hematoma in patients with extensive cancer disease in order to avoid unexpected surgical and prognostic complications. A clinician who has become familiar with the pitfalls in the differential diagnosis between leptomeningeal infiltrations and subdural hematoma will employ analytical thought processes to provide appropriate treatment.

Conflicts of interest

None declared.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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