CASE REPORT

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Unusual occurrence of avascular necrosis with bilateral involvement and ankylosing spondylitis, meningioma and Hodgkin lymphoma

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Abstract

This case report aimed to reveal the multidisciplinary aspect and discuss an association of disorders in a complex case with Hodgkin disease, meningioma, avascular necrosis with bilateral involvement and ankylosing spondylitis. We report the case of a 47-year-old patient, diagnosed with Hodgkin lymphoma at the age of 14. Two decades later, she was diagnosed with frontal chordoid meningioma, which was surgically removed, without radiotherapy. She was re-operated 10 years later for recurrence of meningioma. At the age of 39, she was diagnosed with bilateral avascular necrosis of the humeral head, femoral head and scaphoid bones. In the same year, she was also diagnosed with ankylosing spondylitis. We considered that the occurrence of rare chordoid meningioma, avascular necrosis with multiple locations and bilateral involvement in a patient with a malignancy during childhood and an autoimmune disease was unusual and there may be some common pathways.

Keywords: tumor, avascular necrosis, ankylosing spondylitis, chordoid meningioma.

Introduction

Hodgkin lymphoma (HL) is a lymphoproliferative malignancy affecting mostly teenagers and young adults, but it may also affect elder individuals. Classical HL is divided into several subtypes, of which the nodular sclerosis type prevails in affected young patients. The exact etiology is unknown, but many studies revealed that genetic factors play an important role [1, 2]. During recent years, different susceptibility genes have been identified [2–7].

HL is characterized by the presence of neoplastic cells in an inflammatory background. The association between polymorphisms in inflammation genes and the risk for HL was also reported [8]. The role of the major histocompatibility complex was confirmed by a strong association with human leukocyte antigen (HLA), data that provided new insights into the pathogenesis of the disease [6]. A review of genetic associations in classical HL revealed that *HLA* alleles represent the strongest risk and protective polymorphisms in the susceptibility for this type of lymphoma, emphasizing the role of the immune system in the etiology of the disease [7]. Some observations support the role of chronic immune stimulation in the etiology of the disease. There may be different risk patterns for the different subtypes, suggesting the possibility of several pathogenetic mechanisms [9].

Chordoid meningioma is a rare subtype of meningioma, with high risk of recurrence and aggressive growth. It occurs predominantly in young adults and has a predilection for the supratentorial location [10]. Gene dosage alterations in primary and recurrent chordoid meningiomas were found. It was also demonstrated that cell populations had similar genetic profiles, supporting the previously proposed theory of monoclonal origin for both initial and recurrent tumors [11]. Occurrence or recurrence of benign and malignant tumors has been associated with some *HLA* genes. These associations might suggest that the immune system is involved in antitumor surveillance, recognition and also in destruction of tumor cells [12].

Avascular necrosis leads to destruction of the involved joint. The etiology is multifactorial; there are idiopathic forms and secondary forms, which can be due to a variety of causes, including long-lasting corticotherapy. Recent research provided new insights into the pathogenesis of the disease [13–15]. Any bone can be involved, but the femoral head is more commonly affected. Scaphoid osteonecrosis is very rare and existing data are based on retrospective studies with a small number of patients. Different etiological factors were encountered [16]. Bilateral occurrence of avascular necrosis is rare. Only one case with bilateral involvement of the *tuberculum majus* was described [17].

Ankylosing spondylitis is a chronic inflammatory disorder with progressive evolution. Genetic factors play an important role in the etiology, the strongest genetic

association was found with HLA-B27, but other genes also influence the susceptibility to the disease. Twenty-five loci were found to represent risk variants for the disease [18] and some gene polymorphisms might be associated with progression in ankylosing spondylitis [19].

Inflammatory responses play different roles at different stages of tumor development. It is now known that chronic inflammation may precede tumor development, but it may also follow tumor development. Thus, solid malignancies generate an inflammatory response, which promotes a pro-tumorigenic microenvironment [20, 21]. Several auto-immune and chronic inflammatory diseases have been associated with an increased risk of malignant lymphomas. Even though many aspects are still uncertain, it was found that there is heterogeneity in risk and risk mediators among different inflammatory diseases [22].

Our aim was to reveal the multidisciplinary aspect and discuss an unusual association of disorders in a complex case with Hodgkin disease, meningioma, avascular necrosis with multiple and bilateral involvement and ankylosing spondylitis, trying to draw attention to a possible link between these disorders. Informed consent was given by the patient.

Case presentation

A 47-year-old woman, M.C., Caucasian, presented in the Medical Rehabilitation Clinical Hospital, Băile Felix, Bihor County, Romania, in February 2016, with seven months history of pain in the left crural and tibiotarsal region, rachialgia of low intensity, dizziness, vertigo, general tremor, 10 kg weight loss.

Her medical history was significant for HL during childhood, nodular sclerosis type, diagnosed at 14 years, treated with splenectomy, in the same year, without irradiation. After 21 years, at the age of 35 years, a frontal meningioma, grade II, was diagnosed and surgical resection without radiotherapy was applied. Four years later, she was diagnosed with bilateral avascular necrosis of the humeral head, bilateral femoral head, bilateral scaphoid bone, in the context of corticotherapy (Dexamethasone 16 mg/day), which was used for one month before the first surgery for meningioma. In the same year, she was also diagnosed with ankylosing spondylitis. At 45 years of age, after 10 years from the surgical intervention for frontal meningioma, she was diagnosed with meningioma in the external part of the right sphenoid wing and she was re-operated. Previous medication was Sulfasalazine 3000 mg/day, Methotrexate 15 mg/week and nonsteroidal anti-inflammatory drugs.

Upon admission in our Clinic, her physical examination revealed the following abnormal data: cheek erythema, limited bilateral shoulder and hip mobility in all axes of motion, positive Trendelenburg's sign on the left side, right leg shortened 1 cm, 10 m walking test – 10.01 s. Measurement of spine mobility showed its limitation in all axes (Schober index 10/10.5, Stibor index 56/60, Ott index 30/36, cirtometry index 95/96.5).

Laboratory tests at presentation revealed inflammatory syndrome, with 1-hour erythrocyte sedimentation rate (ESR) of 35 mm, 2-hour ESR of 70 mm, fibrinogen 607 mg/dL, C-reactive protein (CRP) 16 mg/L.

Liver function tests were not elevated. Serum protein, hemoglobin electrophoresis, immunoelectrophoresis and immunofixation showed no pathological findings. Rheumatoid factor, antinuclear antibodies, anti-double stranded deoxyribonucleic acid (dsDNA) antibodies, anticardiolipin (aCL) antibodies, anti-β2-glycoprotein I antibodies, and antineutrophil cytoplasmic antibodies (ANCA) test were negative.

Pathological examination of the specimens taken during the second neurosurgical intervention for the recurrent meningioma, of the right sphenoid wing, revealed areas with cells arranged in placards alternating with areas forming trabeculae arranged in a mucoid stroma and a scant lymphoplasmacytic infiltrate (Figures 1 and 2). In some fragments, meninges and brain parenchyma, without brain invasion, were identified. This aspect was also observed in a sphenoid bone fragment. Alcian Blue (AB)–Periodic Acid–Schiff (PAS) staining was positive in mucinous matrices (Figure 3). Cytokeratin AE1/AE3 was negative on tumor cells and excluded a metastatic carcinoma (Figure 4). S100 protein was diffusely positive on tumor cells. Tumor cells were positive for epithelial membrane antigen (EMA) and expressed a low proliferation (Ki67) index of only 3% (Figures 5–7).

All these aspects led to the diagnosis of chordoid meningioma, *World Health Organization* (WHO) grade II.

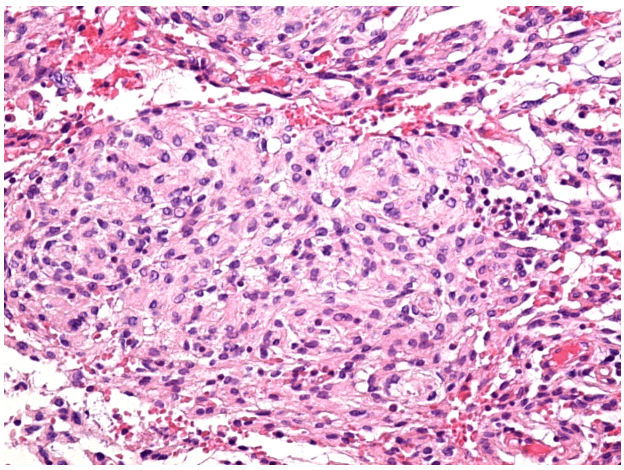

Figure 1 – ***Chordoid meningioma WHO grade II: proliferation of epithelioid cells arranged in trabeculae in a myxoid stroma (HE staining, ×200). WHO: World Health Organization; HE: Hematoxylin–Eosin.***

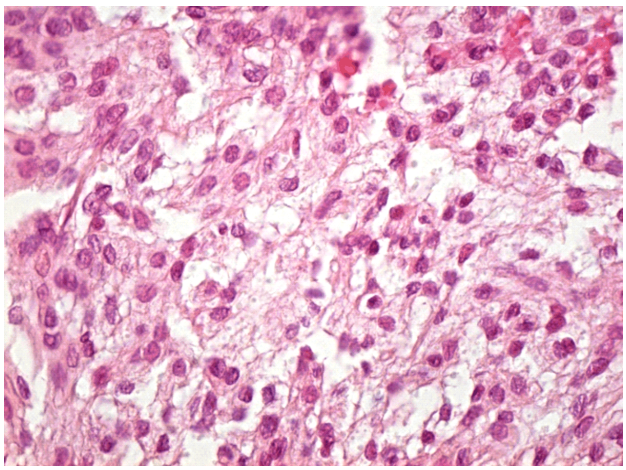

Figure 2 – ***Chordoid meningioma WHO grade II: epithelioid cells with nuclear pseudoinclusions and a scant lymphoplasmacytic infiltrate (HE staining, ×400).***

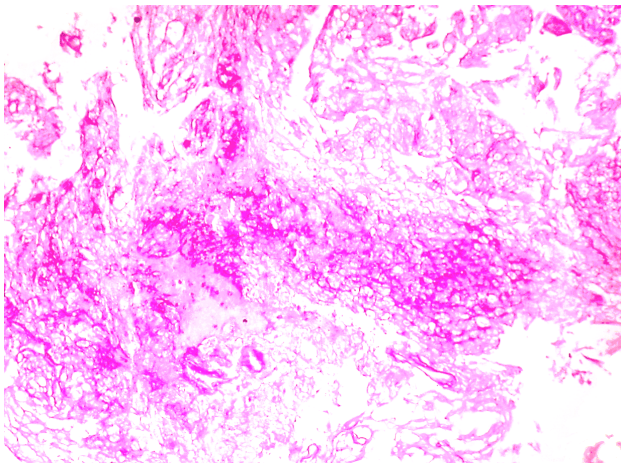

Figure 3 – ***Chordoid meningioma WHO grade II: the stroma is rich in PAS-positive acid mucins (AB–PAS staining, ×100). AB–PAS: Alcian Blue–Periodic Acid–Schiff.***

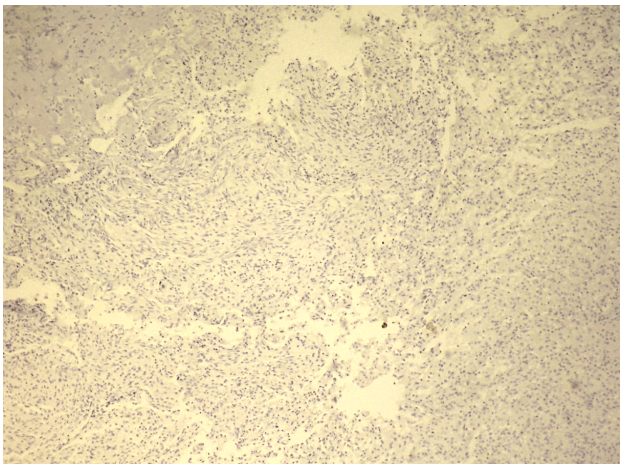

Figure 4 – ***Chordoid meningioma WHO grade II: cytokeratin AE1/AE3 was negative on tumor cells (Immunostaining with cytokeratin AE1/AE3 antibody, ×100).***

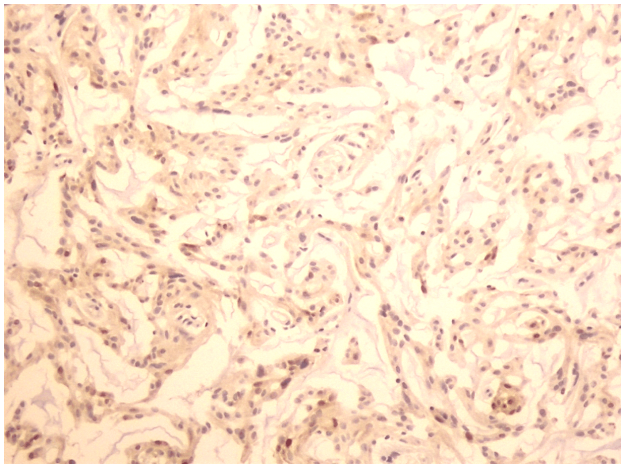

Figure 5 – ***Chordoid meningioma WHO grade II: S100 protein is diffusely positive on tumor cells (Immunostaining with anti-S100 antibody, ×100).***

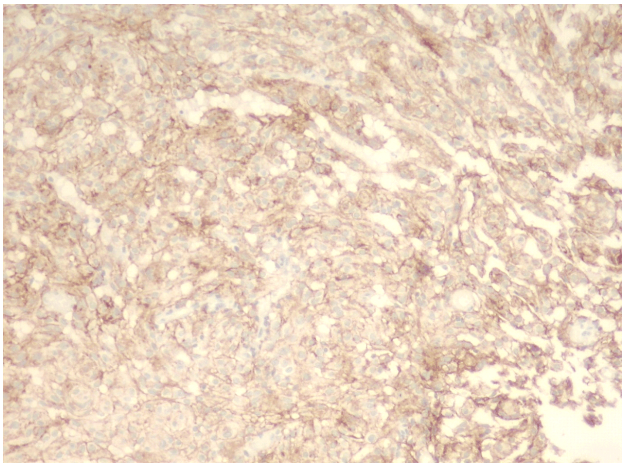

Figure 6 – ***Chordoid meningioma WHO grade II: tumor cells are positive for EMA (Immunostaining with anti-EMA antibody, ×100). EMA: Epithelial membrane antigen.***

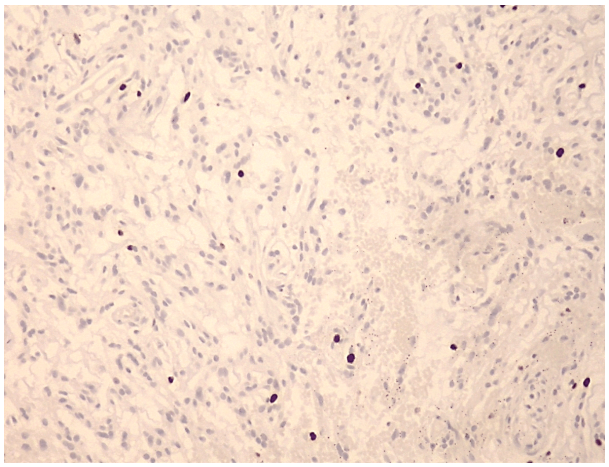

Figure 7 – ***Chordoid meningioma WHO grade II: tumor cells express a low (only 3%) Ki67 proliferation index (Immunostaining with anti-Ki67 antibody, ×100).***

Hip, shoulder and scaphoid radiographs revealed bilateral osteonecrosis.

Abdominal ultrasound, gastroscopy, computed tomography of the abdomen and chest showed no significant abnormalities.

By differential diagnosis were excluded other causes like malignancy, connective tissue diseases. Considering clinical examination and laboratory relevant data, she was diagnosed with ankylosing spondylitis, operated meningioma, bilateral avascular necrosis of the humeral head, femoral head and scaphoid.

At present, the patient is treated with Leflunomide 20 mg/day, follows a daily kinetic program and twice a year complex medical rehabilitation. After Leflunomide therapy was introduced, 2-hour ESR was 28 mm, fibrinogen decreased to 260 mg/L, and CRP to 0.46 mg/L. Three months after starting treatment, a good clinical response was noticed. Hip arthroplasty for hip osteonecrosis was proposed, but the patient refused. Annual control for a possible recurrence of meningioma was recommended.

Discussions

In this study, we describe a case of 47-year-old woman, with a medical history of HL and recurrent chordoid meningioma, which after about 20 years was diagnosed with ankylosing spondylitis.

The association between cancer and chronic inflammation has been reported in the literature, but a firm

causal relationship has not been demonstrated. It is known that inflammatory diseases increase the cancerous disease risk [23, 24].

Pathogenic mechanism underlying this association is based on the presence of inflammatory cells and mediators (chemokines, cytokines, prostaglandins) in tumor tissues, tissue remodeling and angiogenesis, similar to that seen in chronic inflammatory diseases. Cancer and inflammation are linked by intrinsic and extrinsic pathways. The intrinsic pathway is triggered by genetic activation: oncogenic mutation (mutations in the genes encoding *RAS*, *MYC* and *RET*), chromosomal rearrangements and the inactivation of tumor-suppressor proteins [von Hippel–Lindau (VHL) tumor suppressor, transforming growth factor-beta (TGF-β), phosphatase and tensin homologue (PTEN)]. In contrast, the extrinsic pathway is triggered by inflammatory or infectious conditions. Lastly, the two pathways converge causing the release of inflammatory mediators and the production of an inflammatory microenvironment in tumors [24].

In the present case report, HL was diagnosed during childhood at the age of 14 years. In most cases reported in the literature, the incidence of HL is high among adolescents aged 15 to 19 years, children aged 10 to 14 years, five to nine years, and 0 to four years [25]. In addition, after 21 years, this patient developed a frontal chordoid meningioma and ankylosing spondylitis.

Chordoid meningiomas are very rare and there are limited literature data about their attributes. Histological analysis of chordoid meningioma shows the presence of groups of vacuolated spindle or epithelioid cells, which are fixed in a mucoid stroma, but inflammatory cells may be also present [26–28]. Other tumors with similar histological aspects, such as chordoid glioma, chordoma, metastatic mucinous carcinoma, chordoid sarcoma, low-grade chondrosarcoma, etc., must be taken into consideration and excluded within differential diagnosis [26, 29].

In our case, investigations were initially directed towards an autoimmune disease and even to a chronic inflammatory disorder. The lack of proximal muscle weakness and normal values of serum muscle enzymes [creatine kinase (CK), lactate dehydrogenase (LDH)] excluded polymyositis. Normal values of alkaline phosphatase excluded Paget's disease, bone metastases and multiple myeloma. X-rays did not show signs of bone metastases or Paget's disease. Immunoelectrophoresis data excluded myeloma.

We have also discussed the possibility of an anti-phospholipid syndrome (APS) commonly associated with avascular necrosis, but our patient was negative for aCL and anti-β_2-glycoprotein I antibodies. Also, other autoimmune diseases (systemic lupus, mixed connective tissue disease, Sjögren's syndrome) were excluded. Thus, we have not registered immunological abnormalities, such as: rheumatoid factor, antinuclear antibodies, anti-dsDNA and anti-ribonucleoprotein/Smith (RNP/Sm) antibodies, anti-Ro [Sjögren's syndrome-related antigen A (SSA)] autoantibodies, ANCA, complement component 3 (C3) and C4.

Thyroid peroxidase antibodies and anti-thyroglobulin tests were within normal limits. The free thyroxine (T4) and thyroid-stimulating hormone (TSH) in the normal range excluded impairment of thyroid.

Based on clinical symptoms, laboratory tests and imagistic outcomes, the associated diagnosis was ankylosing spondylitis. Our findings coincide with other reports, which show that autoimmune or chronic inflammatory diseases can be associated with cancerous diseases [30, 31].

Further, the development of osteonecrosis with multiple locations, especially with bilateral involvement (humeral, femoral heads and scaphoid bones) was seen in our patient. We believe that osteonecrosis appeared both in the context of corticosteroid therapy, but also linked to chronic inflammation. Other possible risk factors for osteonecrosis are chronic dialysis, chemotherapy, hyperlipidemia, myxedema, pancreatitis, pregnancy and systemic diseases (lupus erythematosus, rheumatoid arthritis, Cushing's syndrome) [32]. Actually, in our case none of the above situations were noted.

Conclusions

Our case is characterized by several features: the onset of HL during childhood followed by the development of a frontal chordoid meningioma; diagnosis of avascular necrosis with multiple and bilateral involvement after about 25 years from the onset of HL and four years after meningioma; the onset of ankylosing spondylitis after the two tumoral diseases. This study stands out because of the multidisciplinary aspect of this case, but also in terms of discussing possible pathogenetic mechanisms that underlie these disorders.

Conflict of interests

The authors declare that they have no conflict of interests.

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