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Brown-Sequard Syndrome due to an Acute Cervical Spinal Cord Compression Fracture Associated with Asymptomatic C5 Vertebral Hemangioma

—Case Report and Review of the Literature

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Abstract

Background: Spinal hemangiomas are benign tumors that develop mostly in vertebral bodies but their behavior is uncertain. The thoracic spine is the most frequently affected segment by these lesions, followed by the lumbar and cervical levels. Although the vast majority are asymptomatic, aggressiveness in some cases can cause spinal pain, various neurological compromises or spinal instability. **Case Description:** We present a case in which a previously asymptomatic male patient suffered a craniocervical trauma that caused a severe compression fracture of C5, which manifested clinically and radiologically as a Brown-Sequard syndrome. Due to intraoperative behavior and surgical findings, the bone fragments and the tissue found within them were histologically analyzed, which confirmed the existence of a capillary hemangioma that considerably involved the vertebral body. The surgical treatment and neurological recovery were adequately evaluated with very good results. **Conclusions:** Vertebral hemangiomas are considered lesions with a well-defined etiology but an uncertain evolution. The clinical characteristics in the case presented are special due to the development of Brown-Sequard syndrome associated with a severe vertebral fracture that contained a previously undetected vertebral hemangioma that could have influenced the weakening of the fractured vertebral segment. Due to the increasing amount of information about the behavior of these hemangiomas, therapeutic decisions are diverse. Nevertheless, in the case of aggressive hemangiomas, definitive treatment protocols should focus on surgery and total excision of the lesions as a gold standard.

Keywords

Cervical, Hemangioma, Fracture, Brown-Sequard Syndrome

1. Introduction

Spinal hemangiomas are benign tumors that develop mostly in vertebral bodies. However, as these vascular lesions develop, simultaneous proliferation of epidural vessels or vessels in the spinal cord has been seen. The thoracic spine is the most frequently affected segment by these lesions, followed by the lumbar and cervical levels. Hemangiomas can be detected in approximately 10% to 12% of mostly asymptomatic adults [1]. Thus, asymptomatic evolution may result in important changes in vertebral bone structure leading to the development of vertebral fractures. Approximately 1% of these lesions become symptomatic and often cause localized vertebral pain [2]. Vertebral hemangiomas were first described by Virchow in 1867. Different authors, such as Perman in 1926, contributed descriptions of radiological characteristics. Later, Schmorl (1927), Topferin (1928), and Junghanns (1932) demonstrated the frequency of hemangiomas in autopsy materials, concluding that lesions of this kind were present in about 11% of all spines [3] [4].

Below, we present a case in which a previously asymptomatic male patient suffered a craniocervical trauma that caused a severe compression fracture of C5, which manifested clinically and radiologically as a Brown-Sequard syndrome. Due to intraoperative behavior and surgical findings, the bone fragments and tissue eliminate therein were histologically analyzed. We concluded that the suspicious material constituted a capillary hemangioma of the vertebral body that may have contributed to the development of such fracture.

2. Case Presentation

This is the case of a 65-year-old slim male who was known to suffer chronic hypertensive disease that was controlled with a daily dose of telmisartan 80 mg/ hydrochlorothiazide 12.5 mg (Micardis Plus, Boehringer Ingelheim, Germany). At the time, he had no other medical problems and was a nonsmoker. He was standing on a ladder installing Christmas lights on the roof of his house when he fell from a height of about 8.2 ft. The resulting direct frontal impact on the ground produced a transient loss of consciousness. Upon regaining consciousness, he complained of severe neck pain and cervical muscle spasms that made it unbearable for him to move his neck in any direction. His relatives noticed that movement of the left side of his body was severely limited, so they immediately took him to our Emergency Medical Service where the patient received initial medical care. He was immobilized on a vacuum mattress and his C-spine was stabilized with a Stifneck®. Upon arrival, he was alert and cooperative. ABCDE assessment, including log roll, revealed no pathologic findings. Once the patient's hemodynamic

status was confirmed as stable, a careful secondary assessment revealed severe neck pain that extended into both shoulders and cervical muscle rigidity in all directions.

A neurological examination according to ASIA criteria revealed that he could raise his shoulders symmetrically. Left biceps and triceps strength had diminished by 2/5. The rest of the left side of his body was symmetrically and flaccidly paralyzed. Strength on the right side of the body was 4/5. Deep tendon reflexes on the left side were absent, and on the right were 2/4. Bilateral Babinski signs were identified. He referred to not well defined paresthesia on the left side of the body. Sensory examination revealed that fine touch, vibration, two-point discrimination, and conscious proprioception were affected from C5 down; pain, temperature, and crude touch were absent on the right side of the body below C6.

Plain radiographs were of no value due to the obstruction caused by the shoulders. An emergency MRI, however, revealed a severe C5 burst fracture in which C4 had impacted over C5. The posterior left wall of C5 was literally expelled into the medullary canal where it compressed the left side of the spinal cord causing severe spinal cord edema that extended from C3 to C6. Axial MRI images at C5 confirmed severe compression of the left side of the spinal cord by a vertebral body fragment. The expelled bone fragment exhibited severe destruction of various intensities without a well-defined pathological pattern. This compression at C5 produced an important left-sided edema in both adjacent cranial and caudal segments, predominantly affecting the posterior white column and posterior horn (**Figure 1**).

The patient was immediately transferred to the operating room scheduled for an anterior cervical procedure to perform a partial resection of the inferior platform of C4 and a total C5 corpectomy. Some of the ejected C5 fragment was behind the C4 vertebral body, so extracting it before removing the inferior platform of C4 was considered too risky. A conventional anterior cervical spine approach, allowed us to confirm the presence of an important hematoma along the longus colli muscle fibers. As C5 was drilled, we noticed a profuse bleeding which was controlled with bipolar sealer electrocautery, topical hemostatic agents, intravenous etamsylate (Dicynone), and hypotensive anesthesia. A local biopsy was obtained because of the presence of dark and softly vascularized tissue between the osseous striae. As C5 resection progressed, bleeding could be controlled notably and the compressive fragment was successfully extracted without increasing injury to the spinal cord.

After ensuring spinal decompression, spinal alignment was performed manually under fluoroscopic control until appropriate lordosis was obtained. Next, a vertebral body replacement device (ADD Plus® and VBR®, Ulrich) was interposed and progressively expanded at the site of the C5 corpectomy. Once complete adhesion of the device to the inferior surface of C4 and superior surface of C6 was achieved, fixation of it was performed using cortical and expandable blocking screws (Ulrich Osmium™) (**Figure 2**).

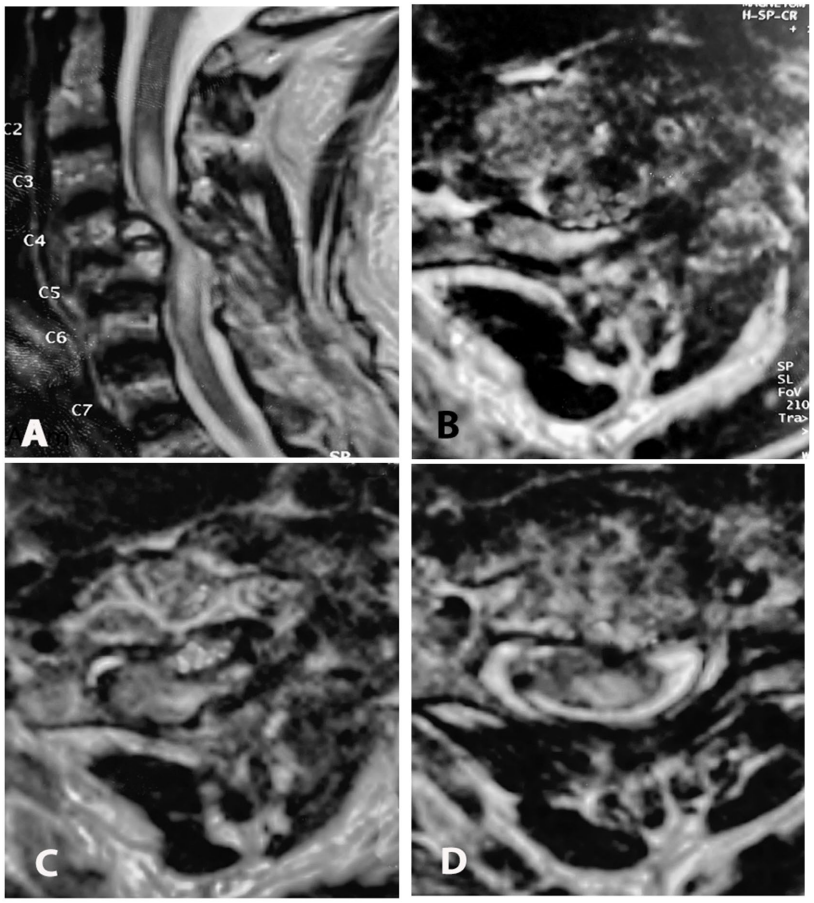

Figure 1. (A) Sagittal T2-weighted Magnetic Resonance Imaging (MRI) of the cervical spine demonstrating a severe compressive fracture of C5 with a posterior wall expelled bone fragment located behind C4 and compressing the spinal cord. Extensive left side edema of the spinal cord is evident. Note the increase in signal intensity of the expelled bone fragments. (B) Axial T2-weighted section of C5 confirms the left-sided spinal cord compression with severe edema that extends both (C) rostrally and (D) caudally in the spinal cord.

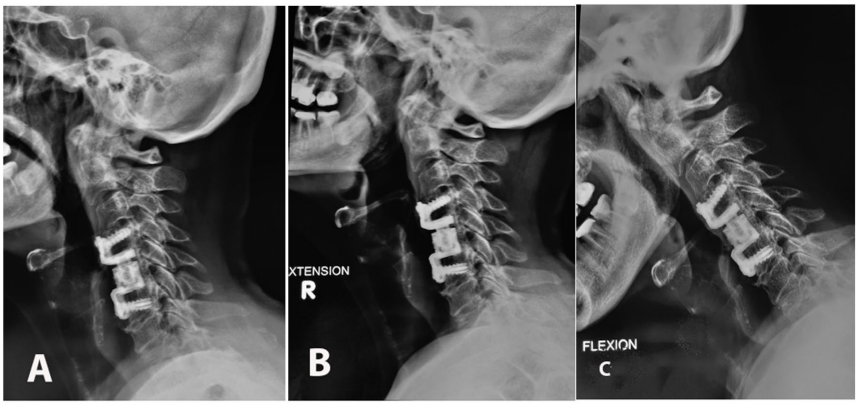

Figure 2. (A) Immediate postoperative lateral X-Ray films confirming an adequate position of the stabilizing device with expandable and bicortical anchorage of the screws. (B) and (C) Three months after the procedure, stability of the expansive cage is acceptable on dynamic films.

Postoperative surveillance was provided at the Intensive Care Unit, where the patient was kept under mechanical ventilation assistance for 72 hours with no adverse events. When he regained consciousness, 2/5 symmetrical arm and leg strenghtwas evident on the patient's left side. He initiated physical therapy and recovered progressively including sensitive alterations. At 3 months, he was able to carry out all his activities unassisted and at 6 months, he returned to his normal life.

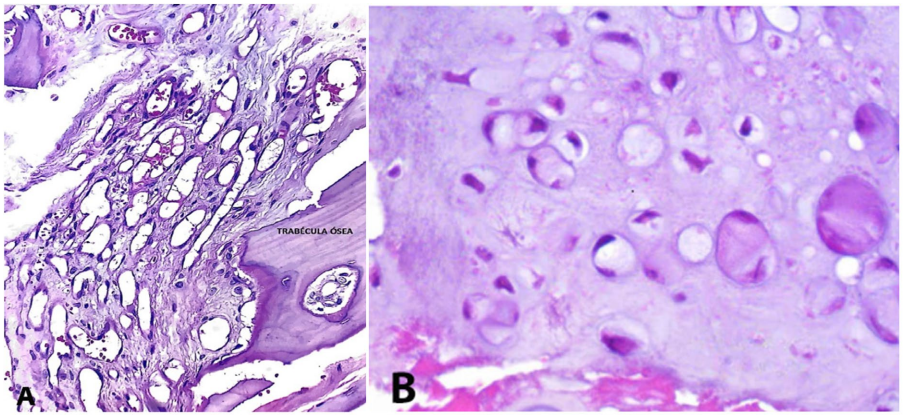

Figure 3. H&E Stain. (A) Bone thinned trabeculae are interspersed with fibrocartilage tissue containing medium caliber blood vessels proliferation. It is observed the presence of a thin wall and dilated lumen where the endothelium showed a thin layer of flattened cells. (B) Chondrocytes atypia but no mitosis, reactive bony beams of different size and shape were also present on the margins suggesting a vertebral capillary hemangioma.

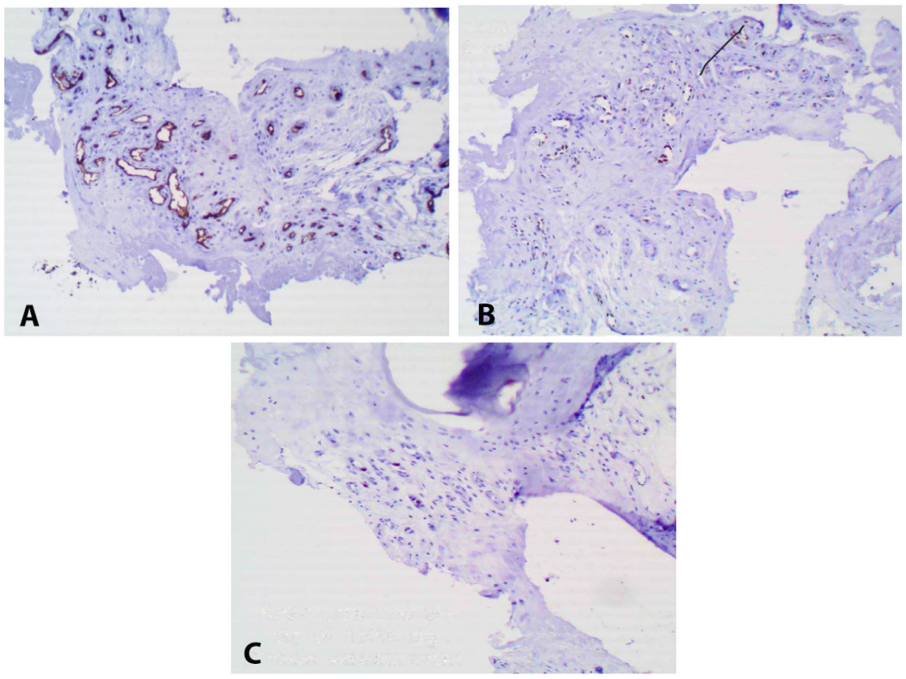

Figure 4. Immunohistochemical assay demonstrated (A) the presence of CD 34 lymphocytes, (B) ERG 3+ positivity and (C) Ki67 3+ positivity in 15% of vascular areas, confirming the vascular etiology of the tissue without malignancy.

Histological report of the bone fragments evidenced the presence of bony trabeculae interspersed with fibrocartilage tissue containing a proliferation of medium caliber blood vessels with thin walls and dilated lumen. The endothelium showed a thin layer of flattened cells with chondrocyte atypia but no mitosis. On its margins, there were reactive bony beams of various sizes and shapes suggesting a vertebral capillary hemangioma (**Figure 3**). The immunohistochemistry assay demonstrated the presence of CD 34 lymphocytes, with an ERG 3+ positivity and a Ki67 3+ positivity, thus confirming the vascular etiology of the tissue (**Figure 4**).

3. Discussion

Brown-Séquard Syndrome (BSS) was first described by Dr. Charles-Édouard Brown-Séquard in 1849, as an unusual and treatable spinal cord disease in which muscle weakness (or paralysis) on one side of the body coexisted with loss of sensation on the other side [5]. Some estimates indicate that 2% to 4% of people suffering traumatic Spinal Cord Injuries (SCIs) in the United States may present with Brown-Sequard Syndrome [6]. Traumatic injuries are by far the most common causes: gunshot wounds, stabbings, motor vehicle accidents, blunt trauma, or a fractured vertebra resulting from falls have all been described [7]. Other non-traumatic causes include vertebral disc herniation, spinal cord cysts (that may or not be associated with intramedullary tumors), cervical spondylosis, multiple sclerosis, cystic disease, vascular lesions, and infections causing traverse myelitis such as herpes zoster, empyema and meningitis [8] [9]. Vertebral Hemangiomas (VHs) are benign vascular neoplasms mostly formed by endothelial cells that grow within marrow spaces in bones encasing bony trabeculae. They are generally considered neoplasms, but because of their lack of aggressive histopathological features, these lesions have also frequently been referred to as hamartomas or vascular malformations. A vertebral hemangioma may rarely predispose to the development of a vertebral fracture and thereby cause acute compression of the spinal cord. To our knowledge, acute BSS has never been described in patients with traumatic vertebral fractures in which the affected vertebral body presented an asymptomatic capillary hemangioma.

Demographics: Vertebral hemangiomas represent the most common form of benign spinal tumors. The prevalence of VHs accounts for 26% to 28% of all skeletal hemangiomas. These benign tumors are found in 10% to 12% of the general autopsy series [10] [11]. However, a retrospective analysis of 198 cases published by Slon *et al.* confirmed a prevalence of 26.0%, in which hemangiomas were multiple in one-third of the cases [12] [13]. Hemangiomas are independent of sex, appearing in females (28.6%) and males (23.5%) but they are age-dependent. The mean age of affected individuals (65.8 years) is higher than the age of unaffected individuals (56.2 years) [14]. Hemangiomas most commonly occur in vertebral bodies of the thoracic and lumbar spine, although cervical lesions are rarely reported and account for about 7% [15] [16]. A third of all cases involve occasional extensions to the posterior arch and multiple hemangiomas [4] [17].

Etiology: Vertebral hemangiomas are benign tumors derived from newly formed blood vessels that replace the bone marrow as they develop. They are thought to be dysembryogenetic disturbances and affect the proper differentiation of blood vessels. They have been proposed as hamartomas because they could be considered more likely to be congenital vascular malformations [18]. The exact causes of this disease are yet unknown, although it is assumed that genetic predisposition is a determining factor. Angiogenesis may play a role in disorganized vascular components present in normal tissue. It is a known fact that cytokines, such as basic Fibroblast Growth Factor (bFGF) and Vascular Endothelial Growth Factor (VEGF) stimulate angiogenesis. Peaks of these angiogenic factors or drops in angiogenesis inhibitors (eg, interferon-gamma, Tumor Necrosis Factor [TNF]-β, and Transforming Growth Factor [TGF]-β) have been implicated in their development [19].

Some researchers focus on an increased amount of estrogen circulating in the blood, which may explain why female patients may suffer from hemangiomas slightly more frequently than male patients (ratio 1:1.5). Slon *et al.* reported that VHs appeared in 42 females (60%) and 28 (40%) males [14]. Moreover, pregnancy may be considered a risk factor that could reveal or aggravate hemangioma symptoms [20]. Under these circumstances, the proposal that endothelial proliferation stimulated by estrogen may result in mitogenic enlargement of hemangiomas during pregnancy has been considered. However, the existence of estrogen or progesterone receptors in vertebral hemangiomas is not conclusive, so there may exist other factors that influence the greater size of pregnancy-related hemangiomas [21] [22]. Among these, Vijay *et al.*, and later Wang *et al.*, postulated that hemodynamic venous changes during the enlargement of the gravid uterus caused compression of the inferior vena cava. This would elevate pressure in the paravertebral venous plexus and probably increase congestion of the hemangioma by inducing a 30% to 50% rise in blood volume thereby increasing the size of the lesion [22] [23].

Pathology: Up to 75% of osseous hemangiomas occur in the vertebrae but they can also appear in the calvarium, calcaneus, and long bones [24]. Macroscopically, VHs look like a soft, dark red mass that may include gross intralesional sclerotic bone trabeculae and scattered blood-filled cavities. Histologically, hemangiomas are classified by type of vascular channel into capillary, cavernous, arteriovenous, and venous [13]. These lesions, however, are infrequently pure in histological terms. Non-vascular components can also be observed in angiomatous lesions (cavernous hemangioma in particular) and may include fat, smooth muscle, fibrous tissue, hemosiderin, and thrombus. Lesions are mostly composed of thin-walled blood vessels aligned in a single layer of flat endothelium mostly set in a substratum of reactive overgrowth and edematous adipose marrow stroma [25]-[27]. Capillary hemangiomas represent the most frequent variant in this group of lesions. Well-structured bone and chondrocytes have been identified in radiological images featuring reinforced trabecula and striations, so vertebral fractures are not

the initial clinical picture [13]. Lesions containing less fat and more vascular stroma tend to be less common. They are located between T3 and T9 [27]. Cavernous hemangiomas are usually larger and less well-circumscribed and have a greater tendency to involve deeper structures. Large cavernous vascular spaces separated by scant connective tissue stroma constitute these well-defined lesions of variable sizes between 1 - 2 cm in diameter. They may sometimes show vascular replacement of the marrow, potentially with calcified or cystic foci and a reactive sclerotic margin.

Immunohistochemical analysis of these lesions is based on a positive result for ERG transcription factor, known to be expressed in endothelial cells. ERG is a highly specific marker for benign and malignant vascular tumors in which nuclear positivity is uniformly detected. CD34 is a sialomucin that can be found in endothelial cells, stem cells of the hematopoietic system, and dendritic fibroblasts. It is expressed in 90% of benign or malignant endothelial neoplasms. Muscle actin immunostaining may be especially prominent in some types of cellular hemangiomas [28]. Today, immunohistochemical analysis of these lesions should be considered as part of the diagnostic criteria to rule out any associated malignancy or make a differential diagnosis.

Clinical Manifestations: Despite recent advances in diagnostics, predicting the biological behavior of spinal hemangioma is still not feasible [29]. According to Boriani *et al.*, VHs can be classified into four categories based on a patient's lesions and symptoms: mild bony destruction with no symptoms (Type I); bony destruction with pain (Type II); aggressive lesions with epidural and/or soft tissue extension (Type III); and aggressive, neurological deficit with epidural and/or soft-tissue extension (Type IV) [30].

Most VHs are asymptomatic, but they may evolve and follow a course resembling that of malignant spine tumors. Symptomatic vertebral hemangiomas are rare. Between 0.9% and 1.2% of spinal hemangiomas result in pain and neurological deficits [29] [31]. Local pain is the most frequent symptom. Symptomatic lesions containing less fat and more vascular stroma tend to be less common and are most frequently located between T3 and T9. Active lesions with spinal cord or nerve root compression are rarely seen but with the first neurologic manifestations, they typically progress slowly over several weeks to months associated with increasing back pain and they depend on the compression level rather than being the cause of it [11].

Clinical manifestations usually appear when hemangiomas reach the vertebral surface where they cause changes such as deformity and invasion of the epidural space and adjacent areas, especially the spinal cord (cortical layer hypertrophy) [29] [32]. Vertebral bodies are the most affected location, but involvement of the posterior arches may be observed with even extension beyond vertebral limits and paravertebral distribution, which could lead to erroneous diagnoses such as spondylitis with paravertebral abscess [33]. It is important to differentiate between intra and extraosseous hemangiomas because extraosseous hemangiomas are fre-

quently associated with symptomatic lesions, this differentiation can be easily made with MRI [34]. Exceptionally, the presence of acute epidural bleeding originating from the hemangioma itself may cause neurologic compression [35]. Circulatory disturbances, either due to pathologic clotting or vessel recanalization, exhibit some potential to alter hemangioma stroma volume via spinal cord blood flow disturbances, which should be considered as an aggressive manifestation [29] [32] [36]. Medullary ischemia, possibly induced by direct vascular steal from the anterior spinal artery, seems highly unlikely to occur.

Despite the outstanding anatomical changes that these lesions can display, spontaneous vertebral fractures are extremely rare as initial clinical symptoms [35] [37]. Pathologic burst fracture can be problematic considering that these highly vascular lesions can subsequently bleed causing a hematoma and possibly cord compression. In our case, such bleeding generously happened along the longus colli muscle. Some other factors have been proposed as predisposing to develop pathological fractures. In 2017, Knechtle *et al.* described the case of a male patient with a pathologic fracture in a Th5 vertebral body containing hemangioma associated with osteopenia. In this case, hemangioma associated with decreased testosterone and vitamin D levels were the most determinant pathological factors [38]. In our patient, the only factor related to the C5 fracture was the presence of a thinned trabecula containing a capillary hemangioma inside the vertebral body evidenced by the histological analysis of the resected bone fragments. From our point of view, this fracture occurred in a structurally weakened and biomechanically vulnerable vertebra. In addition to the presence of the vascular lesion, an outstanding aspect of this case was the clinical picture of an acute cervical Brown-Sequard syndrome produced by an expelled posterior wall fragment; a situation never before described in association with hemangiomas.

Diagnosis: VHs comprise a stroma within an osseous network that originates from thickened vertical striation observed as linear, parallel condensations resembling vertical bars. These bars are considered typical of VHs on plain radiographs [15]. VHs may also be visualized as osteolytic lesions that, in combination with trabeculations resembling a "honeycomb", are classically called the "corduroy sign" [29]. They constitute the characteristic appearance of multiple punctate areas of bone sclerosis on axial CT images, creating the "white polka-dot" sign. CT identification of VHs is possible in approximately 80% of cases [33]. Fatty stroma and serpentine vascular channels are responsible for the low-density components on plain radiographs and CT images. In their report, Laredo *et al.* studied CT attenuation values of the stroma in VHs and determined that such stroma could have either fat or soft-tissue attenuation or both. In their series, a predominantly fatty content, as shown by negative attenuation values on CT scans or by increased signal intensity on T1-weighted MR images, was found in all asymptomatic VHs, whereas, soft-tissue content with low signal intensity was prevalent in compressive lesions [15]. Different reports confirm that the fatty component of hemangiomas shown in MR imaging is usually interspersed between vascular channels ex-

hibiting a high T1 signal. Fat overgrowth exhibits a signal very similar to subcutaneous adipose tissue. Associated vascular components demonstrate increased signal intensity with a serpentine pattern on T2-weighted MR imaging, which suggests slow blood flow [24] [27] [39].

Hemangiomas presenting with posterior element extension, paraspinal involvement and only discreet amounts of intertrabecular fatty stroma are more likely to be associated with symptoms, so they are considered aggressive [40]. Vertebral body weakness has been documented in large hemangiomas, which increases the chances of vertebrae to develop vertebral fractures. Hemangiomas associated with epidural extension and posterior longitudinal ligament displacement that adopt a bilobular appearance in axial images display the so-called "curtain sign"; such spinal epidural hemangiomas are of the cavernous type [27]. VHs are thus considered to have well-defined radiological features in plain X-rays, CT, and MRI. Lesion aggressiveness strictly with the development of pathological fractures, however, makes it difficult to differentiate VHs from other pathologies like primary bone malignancies, lytic metastasis, multiple myeloma, or even infectious processes [27]. In our case, without the evidence of previous radiological image it was important to realize that by the time the spinal compression had occurred, classic VHs radiological features were quite difficult to be demonstrated, so we could not have suspected this diagnosis until the surgical event.

Treatment Strategies: It is recommended to follow all asymptomatic patients detected with VHs with annual neurological and radiological examinations. Various treatment options are available for VHs that considered as aggressive without consensus strategy and it is usually matter of local habit [41], so treatment needs to be individualized. The standard goal should focus on pain control, neurological preservation, and promoting adequate spinal stability.

Traditionally, percutaneous techniques like direct ethanol injections or vertebroplasty (alone or in combination with surgery) have been deemed good options for treating symptomatic VHs. In the case of ethanol injection, the technique has been confirmed to destroy vascular endothelium and induce irreversible sclerosis of the hemangiomatous venous pool. The lesion shrinks once deprived of its blood supply relieving neurological signs and symptoms [42]. However, if it is to be efficacious, ethanol must be injected directly into the vascular spaces of the hemangioma. Once the needle is positioned with fluoroscopic guidance, we suggest combining this procedure with intraoperative angiography or a CT-scan image to confirm correct needle positioning within the vascular spaces.

Although this technique is considered quite safe, when Heiss *et al.* detailed their experience with direct percutaneous ethanol injection, they described its tendency to produce severe vertebral osteonecrosis when the amount of pure ethanol injected exceeded 12 ml [43]. Nevertheless, Doppmann *et al.* reported that a maximum dose of 50 ml of ethanol opacified with metrizamide (10 - 12 ml ethanol + 3.75 g of metrizamide) achieved acceptable thrombosis of the lesion. Sometimes these vascular changes may predispose to various types of pathological fractures

and consequently the persistence of vertebral pain due to poor bone quality. In this author's report, both cases that experienced this complication, required surgical excision of the affected vertebral body complemented with internal fixation and stabilization [44]. In these cases, ethanol-induced devascularization of the vertebral hemangioma greatly reduced the operative blood loss generally associated with this type of surgery. It may be assumed that alcohol ablation might be a good way to treat VH's per se, but it does not restore osseous characteristics and strength to the vertebral body predisposing it to pathological fractures. Therefore, it is feasible and recommended to supplement this treatment option with Polymethylmethacrylate (PMMA) augmentation or surgical excision and instrumentation. Other reported complications with this option include transient neurological deterioration (including Brown-Sequard syndrome) and recurrence [45] [46].

Percutaneous vertebroplasty offers immediate pain control due to the stabilizing properties of internal consolidation of microfractures in trabecular and cancellous bone, thus promoting a considerable hardening of hemangiomatous vessels with a low rate of complications. Deramond *et al.* reported pain control in 90% of cases when performing vertebroplasty with PMMA injection in selected patients [47]. We recommend using fluoroscopic imaging to continuously monitor polymethylmethacrylate injections because of increased chances for intravascular acrylic migration and invasion of the spinal canal by the mixture in the presence of an inadequate posterior cortical wall or foraminal leak. This has been demonstrated by Chiras *et al.* who confirmed that in their series of 258 vertebroplasties, including 78 by angioma, 13 cases developed foraminal cement migration [48]. It is well known that neither kyphoplasty nor vertebroplasty have any direct antitumor biological effect. External beam radiotherapy is usually recommended when an early recurrence is suspected [49].

Different modalities of radiotherapy have been proposed as another option to treat painful symptomatic VHs, but to date, there is no overall consensus on a single option for cases with neurologic manifestations, specifically in patients with acute severe deterioration. Although some neurological benefits have been described with radiotherapy, these usually take several months to occur [50]. With the development of intensity-modulated radiation therapy, it may be possible to safely deliver higher doses of radiation while avoiding complications. Pinar-Sendero *et al.* described an interesting intra corporeal radiation delivery procedure performed during percutaneous kyphoplasty. During a standard pedicular-access kyphoplasty, the authors introduced specially designed metallic sleeves to guide a 4.2 mm diameter Intrabeam sheet. A single intralesional dose of 8 Gy into 10 mm was delivered for 120 seconds. After radiation, bone cement was injected bilaterally without complications. Throughout the 30-month follow-up, the patient remained asymptomatic, free of radiological and neurological signs of tumor recurrence [49].

Postoperative complications in patients who previously received radiation ther-

apy are well recognized. These include wound complications such as delayed union, dehiscence, and Surgical Site Infection (SSI), all of which should be considered. According to Ghogawala, these complications may arise in 32% of patients who undergo irradiation before surgical decompression [51]. SSI after surgery for spinal metastasis was about 31.8% in patients previously irradiated compared to 1.1% for those that had not received radiation treatment [52] [53]. These same authors confirmed that wound dehiscence only occurred in patients who had received > 40 Gy of preoperative irradiation and was especially frequent in patients whose irradiation treatment was ≥12 months. Supporting this, Devalia *et al.* suggested postponing surgery for previously irradiated patients between 3 and 6 weeks after completing radiotherapy because the acute effects of irradiation lead to vascularization disorders and tissue remodeling due to increased apoptotic cell death [54]. Conversely, radiotherapy has demonstrated significant results after surgical resection, especially in cases with partial or incomplete resection, so it is widely recommended as an acceptable co-adjuvant to surgery especially when radiation is delivered under stereotactic control [50].

Endovascular embolization has also been used as a stand-alone therapy for VHs. Hekster *et al.* were the first to report the reversal of spinal cord compression after percutaneous embolization of feeding vessels followed by radiotherapy [55]. However, this treatment has been challenged in the presence of acute spinal cord compression and neurological deterioration. Frequently, hypervascular tumors (cavernous type) with flow-void areas and large cells producing spongy patterns on CT sometimes produce little evidence of spinal cord compression but prominent neurologic deficit. It is of value to consider embolization as an initial treatment for these cases to attempt to resolve these signs. Nevertheless, recanalization of the intentionally occluded vessels and clinical recurrences do exist, so it is still controversial to think about embolization as an isolated treatment option. Moreover, in cases with recurrent or residual disease, a second attempt to embolize the lesion is not feasible as previously occluded feeding arteries can cause reflux of the embolization material into the lumbar and intercostal arteries, which could lead to spinal cord infarction with severe pain and paresis [56] [57].

Embolization followed by surgery is another widely considered option to reduce blood loss during surgical resection. In different reports, several authors have demonstrated good results in achieving gross total tumor removal when preoperative transarterial embolization was performed [58]. In their reports, there was no tumor recurrence during a mean follow-up of 2.4, 3.9, and 10.7 years, respectively. It is widely suggested to consider embolization before resecting VHs to attempt to control surgical blood loss [59]-[61]. There are, however, reports of preoperative embolization failing to reduce intraoperative blood loss. It is believed this is due to the possibility of adjacent recruitment irrigation derived from adjacent tissues that cannot be modified by embolizing these secondary nutrient arteries or with intralesional arterio-venous shunts [56]-[58]. Given this, we strongly recommend the participation of a vascular surgical team to devascularize the accessory vessels

of the lesion followed by resecting the tumor. For this kind of hypervascular spinal tumor, Handa *et al.* strongly recommend total en bloc spondylectomy detecting and resecting tumor vessels, as it tends to decrease intraoperative blood loss when compared with gradual or piecemeal excision [61]. Despite various maneuvers to reduce intraoperative bleeding, en bloc spondylectomy still produces significant blood loss and therefore, increased perioperative morbidity [62].

Surgical options have been proposed in different modalities depending on tumor location, neurological status, spinal stability, and co-morbidity factors. In fact, surgery is indicated for cases in which rapid or progressive neurological symptoms predominate either due to compressive myelopathy or radiculopathy. Neural element decompression and spine stabilization are considered the main goals of any surgical option. The recommendation is to choose interventions according to the size of the lesion and its location. It is essential to plan surgery taking into account technical and anatomical limitations conditioned by the extent of the injury. Surgeries may range from simple decompressive procedures to complex vertebral body resections with subsequent spinal reconstruction modalities that are available from a wide variety of stabilizing devices. Anterior corpectomy is recommended when isolated vertebral body involvement is the issue. If the compression is anterior without involvement of the posterior spinal elements, the resected vertebral body can be replaced with a tricortical bone graft, a cage filled with bone graft reinforced with anterior or lateral segmental plates depending on the affected spinal segment, or with an expandable titanium vertebral body replacement device, with or without complementary pedicle stabilization. In our case, we opted for the stand-alone anterior approach due to the characteristics of the spinal cord compression and the fact that the selected vertebral body replacement device offered appropriate stability. No complementary posterior approach was necessary because both the ligaments and articular elements were preserved.

The posterior approach is recommended in cases when neural arch involvement or associated epiduritis are evident. The extent of decompressive laminectomy with articular processes and pedicular resection depends on tumor invasion [11] [63]. Spinal pedicular instrumentation and arthrodesis should be included when extensive posterior element removal is required. In cases of epidural space, pedicle, or laminar infiltration by angioma, surgeons should expect excessive bleeding during the procedure. This will limit the extent of resection and increase the risk of producing either neural element or vascular injury while attempting to control bleeding. As previously stated, preoperative spinal angiography and embolization are excellent options and widely recommended to locate feeding vessels and reduce intraoperative bleeding of these highly vascular lesions [41] [63]. In cases of fractured vertebral hemangiomas co-existing with mild spinal cord or nerve root compression, either vertebroplasty or kyphoplasty combined with simultaneous posterior decompressive and stabilizing procedures are considered viable treatment options [55]. In their series, Li *et al.* confirmed the effectiveness of posterior decompression combined with bone cement augmentation and internal

fixation to treat pathological fractures due to vertebral hemangiomas with fewer complications and acceptable neurological outcomes.

As surgical techniques evolve and creative devices become available, treatment of this spinal pathology can progress reducing patient's vulnerability to complications and morbidity. To illustrate, Canbaya *et al.* treated a case of vertebral fracture by hemangioma that produced moderate neurological deficit and pain. They described an ingenious decompressive-stabilizing technique that consisted of ablating the vertebral hemangioma with radiofrequency probes applied to the lesion via a bilateral pedicular approach. The hemangioma was then generously packed with autologous cancellous bone graft and 5 cc of hemostatic sealant. According to the authors, this resulted in a considerable reduction of surgical bleeding and very good physiological strengthening of the vertebral body. By applying a complementary short stabilizing pedicular system, they confirmed good results after several months without evidence of tumor recurrence and acceptable bone reconstitution [64]. However, even though it is a technically simple, accessible, and apparently effective management, these authors must validate their data with a larger number of cases and long-term follow-up.

4. Conclusion

Vertebral hemangiomas are considered lesions with a well-defined etiology but an uncertain evolution. Their behavior can be benign and completely asymptomatic or frankly aggressive causing pain, neurological manifestations, and compromising spinal stability. The clinical characteristics of our case are special because the presence of Brown-Sequard syndrome associated with a severe vertebral fracture containing a vertebral hemangioma has not been previously reported. However, due to the lack of prior radiological images, the characteristics of the bone lesion that could have influenced the weakening of the fractured vertebral segment are unknown. There is an increasing amount of information about the behavior of these hemangiomas, so therapeutic decisions are diverse with acceptable results. Nevertheless, in the case of aggressive hemangiomas considering clinical picture with neurological compromise, biomechanical instability and invasiveness of the lesion, definitive treatment protocols should focus on surgery and total excision of the lesions as a gold standard.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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