Role of magnetic resonance imaging in the differential diagnosis of spinal signal changes caused by infection and tumor in the early stages

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Research Article

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Abstract

Background: Spinal infection and tumor have abnormal magnetic resonance imaging (MRI) manifestations in the early stages; however, they are not easy to identify before symptoms appear. These conditions have their own characteristics and need to be correctly diagnosed.

Methods: MRI images of six cases of pyogenic spondylitis, eight cases of tuberculous spondylitis, eight cases of *Brucella* spondylitis, and twelve cases of spinal tumor were reviewed. MRI images of the involved vertebral body, vertebral facet joint, spinous process, paravertebral soft tissue, and posterior dural sac of the vertebral body were analyzed using T2-weighted imaging, T1-weighted imaging, lipgraphy and enhanced MRI, respectively.

Results: MRI data of spinal infection, spinal tuberculosis, spinal tumor, and spinal *Brucella* infection differed in their characteristics.

Conclusions: MRI could provide early diagnostic evidence for spinal lesions.

Introduction

Metastatic cancer, spinal infection, and spinal tuberculosis are frequently seen in the clinic. The spine is easily affected by disease^[1], which can involve the vertebral bodies with or without involvement of intervertebral discs, posterior neural elements, and paraspinal soft tissues. Affected patients can present with similar symptoms, such as backache. When typical signs of spinal inflammation are not present, clinical manifestation can be very similar to metastasis and may not be easily differentiated. Because these diseases require very different treatments, some develop quickly and can cause great harm to people. Thus, correct diagnostic imaging would help to determine suitable procedures and optimal treatment methods. Magnetic resonance imaging (MRI) is an effective tool for the early diagnosis of spinal disease according to its characteristics.

Material And Methods

We performed a retrospective analysis of MRI images of six cases of pyogenic spondylitis, eight cases of tuberculous spondylitis, eight cases of *Brucella* spondylitis, and twelve cases of spinal tumor. The vertebral bodies, vertebral facet joints, spinous processes, paraspinal soft tissues, and the dural sac of the vertebral body were imaged and analyzed using T2-weighted imaging (T2WI), T1-weighted imaging (T1WI), fat-suppressed T2WI, and enhanced MRI. The characteristics of different diseases were summarized according to their different manifestations on MRI.

Results

MRI features of spinal tuberculosis

Before symptoms, such as spinal pain and deformity, marked changes had taken place in the vertebral bodies and surrounding tissues, which were easy to ignore in the early stages of disease. Invasion of diseased tissues into the vertebral bodies was obvious, often with simultaneous fracture. There was no damage to the intervertebral discs in the early stages of disease, but destruction occurred in the middle and later stages, which was accompanied by loss of vertebral height. MRI of the diseased vertebrae showed a low signal or an equal low mixed signal on T1WI and an inhomogeneous high signal on T2WI. With an abnormal signal from the vertebral endplate and under endplate, enhanced MRI showed strip or focal enhanced endplate signals and heterogeneous enhanced vertebral body signals. MRI showed a small cystic or irregular patchy high signal intensity on T2WI. Involvement of paravertebral soft tissues showed irregular flaky abnormal signals on MRI. The edge of the abscess had obvious circular enhancement on enhanced MRI, which was related to the abundance of capillaries in the periosteum and proliferative granulation tissue around the abscess, but there were no blood vessels in the abscess (Fig. 1). This was one of the key features that distinguished abscess from tumor.

MRI features of suppurative spondylitis

Suppurative spondylitis appeared as a hypointense or isohypointense signal on T1WI, a heterogeneous high signal on T2WI, and abnormal vertebral endplate and under endplate signals. We also observed strip or focal endplate enhancement and heterogeneous vertebral body enhancement on MRI and a small cystic or irregular patchy high signal intensity on T2WI. Paravertebral soft tissue involvement showed an irregular flaky abnormal MRI signal.

In the early stages of disease, pyogenic spondylitis appeared as diffuse vertebral body lesions, which involved intervertebral discs and paravertebral tissues. Early invasion and destruction occurred in intervertebral discs, and vertebral height was reduced. The range of lesions in paravertebral tissues did not exceed the height of the vertebral bodies. A low signal or equal low mixed signal was observed on T1WI, and an inhomogeneous high signal was observed on T2WI (Fig. 2).

Pyogenic spondylitis was different from tuberculous spondylitis on MRI. The manifestations were as follows: 1) vertebral body involvement: fewer vertebral bodies were involved in pyogenic spondylitis compared with tuberculous spondylitis, and except for the edge and middle of the vertebral bodies, there was no vertebral body destruction or collapse. The incidence of vertebral body involvement in tuberculous spondylitis was higher compared with other types of infectious spondylitis, and the range of abscess was often extensive, which led to paravertebral abscess and epidural abscess. 2) Paravertebral soft tissue swelling in suppurative spondylitis often had an irregular margin and an unclear boundary and solid soft tissue granuloma enhancement or multiple small abscess enhancements with thick abscess walls and fuzzy edges on enhanced MRI. The involved margin of paravertebral soft tissues in tuberculous spondylitis was often clear and regular, and the abscess wall was often thin and smooth.

MRI features of *Brucella* spondylitis

Vertebral bone marrow edema was the main manifestation of *Brucella* spondylitis infection in the early stages, which appeared as a low-density shadow under the articular surface or in vertebral bodies, and infiltrative bone destruction. The MRI signal of vertebral body lesions was non-specific and showed an abnormal sheet signal. *Brucella* spondylitis appeared as a low signal on T1WI, a slightly high signal on T2WI, and a high signal on fat-suppressed T2WI. When granulation tissue was formed in the bone destruction area, lesions did not show mild or obvious enhancement. When osteohyperplasia appeared in the vertebral bodies, T1WI still showed a low signal and the T2WI signal became complex and presented a low, equal, or high mixed signal, and the sclerosis edge showed a low signal. When the intervertebral disc was damaged, the intervertebral space was narrow and the signal intensity on T2WI was not uniform. Paravertebral abscess showed a low signal intensity on T1WI and a high signal intensity on T2WI. The abscess wall was enhanced, and the wall was thick and irregular without an obvious flow phenomenon (Fig. 3).

The differences between *Brucella* spondylitis and tuberculous spondylitis on MRI were as follows: 1) low-density shadows under the articular surface or in the vertebral bodies were often present in *Brucella* spondylitis, which appeared as diffuse bone infiltration. There was no special MRI signal from the vertebral body. Tuberculous spondylitis was characterized by “mouse nibbled” bony destruction. Bone destruction foci appeared as multiple, irregular, low-density lesions, especially in the articular surface and below the articular cartilage. *Brucella* spondylitis vertebral bodies had diffuse infiltration, but the upper and lower margins of vertebral bodies showed no obvious change. There was no vertebral squeezing or flattening. Flattening of vertebral bodies in tuberculous spondylitis occurred because early lesions were present in the corresponding upper and lower endplates of vertebral bodies, and *Bacillus* destroyed the intervertebral disc and invaded the adjacent vertebral body in the later stages. Because of progressive destruction of the vertebral body by tuberculous granulation tissue and the mechanical relationship in humans, vertebral bodies became compressed and flattened. Paravertebral abscess was observed in *Brucella* spondylitis and tuberculous spondylitis, but paravertebral abscess in *Brucella* spondylitis was connected with the destructed vertebra, the abscess had an irregular shape and a clear boundary, and the adjacent psoas muscle was displaced. Absence of abscess flow phenomenon was a characteristic of *Brucella* spondylitis. Tuberculous spondylitis had paravertebral abscess formation and the abscess was calcified. The abscess often entered the spinal canal and flowed to the next vertebral body. The paravertebral abscess of *Brucella* spondylitis was limited and rarely invaded the appendages and surrounding muscles. Tuberculous spondylitis often invaded the appendages and often presented cold muscle abscess. The abscess also flowed along the muscles. Most cases of *Brucella* spondylitis did not present sequestrum formation. However, multiple, irregular, scattered sequestra could be seen in the destruction area of tuberculous spondylitis, which were also observed with abscess. In the later stages of *Brucella* spondylitis, perivertebral hyperplasia and lace vertebrae formation were characteristic manifestations.

The differences between *Brucella* spondylitis and purulent spondylitis were as follows. First, the appendages were more often infiltrated in pyogenic spondylitis, although this was not generally accompanied by vertebral collapse. *Brucella* spondylitis lesions always appeared, mainly under the

articular surface and/or in the vertebral bodies, but the surrounding accessories were less involved. Due to neutrophil-secreted proteolytic enzymes, a clear abnormal signal was observed in the intervertebral disc, and destruction in the middle and edge of the vertebral body and vertebral deformation often appeared in bone. Second, damage was always observed in the vertebral bodies in *Brucella* spondylitis, but the height of the vertebral bodies often did not change significantly, and no vertebral compression or flattening occurred. There was no facet joint or osteophyte formation in pyogenic spondylitis. In the late stages of *Brucella* spondylitis, the edge of the vertebral body often presented with lace-like hyperplasia.

MRI features of spinal tumor

Typical spinal metastases had abnormal vertebral body signals, multiple vertebra involvement, jumping sign, multiple accessory bone involvement, avoidance of intervertebral discs, and enlargement of the intervertebral space caused by pathological fracture. Fat replacement of bone marrow was an early sign of spinal metastasis. Metastasis had a focal low signal intensity, which could easily be distinguished from the high signal intensity of normal bone marrow with T1WI. A few metastases with hemorrhage had a short T1 (high signal intensity). Deposition of yellow bone marrow might have led to an increase in fat in the center of the lesion, presenting as a bull's eye sign. With T2WI, bone metastases showed brighter signals than normal bone marrow because they contained more water than normal bone marrow. With T2WI, there was a bright line around the metastasis (halo sign). With T2WI, low, equal, high, and mixed signals were observed. If a low signal was observed with both T1WI and T2WI, this signal change often indicated osteosclerosis. Most cases had primary lesions and multiple bone destruction of vertebral bodies and appendages, which started with appendage involvement. T1WI showed an irregular low signal, while T2WI showed a high signal. Edema was observed around the tumor with a fuzzy boundary, and jumping images in the destruction of vertebral bodies were observed on MRI. The abnormal signal generally did not cross the intervertebral space, and "lip-like" bone sclerosis was observed at the edge of the vertebral body. The intervertebral disc was generally not involved, and the intervertebral space generally showed no obvious change. The local mass on MRI showed the signal characteristics of soft tissue, rather than a fluid signal similar to abscess. The enhancement scan showed obvious enhancement (Fig. 4).

Discussion

The pathological and anatomical changes in the spine and its surrounding tissues caused by spinal disease were related to the arterial and venous blood supply around the vertebral bodies^[2].

The arterial blood supply of vertebral bodies

The arteries supplying blood to the vertebra were segmental arteries, which came from the vertebral artery, posterior intercostal artery, lumbar artery, and lateral sacral artery from top to bottom. The arteries ran along the ventral surface of the spinal nerve into the intervertebral foramen, also known as the spinal artery. After entering the intervertebral foramen, the spinal artery was divided into three branches: 1)

dorsal collateral artery, which mainly supplied blood to the pedicle, lamina, transverse process, spinous process, dura mater, and epidural space. The dorsal branches of the superior and inferior spinal artery anastomosed with each other and were accompanied by the posterior side of the internal vertebral venous plexus. 2) The middle branch artery, which supplied the dura mater, including the spinal nerve root, and penetrated into the dura with the nerve root to supply blood to the spinal cord. 3) The abdominal collateral artery, which supplied the vertebral bodies, the anterolateral part of the dura mater, and the tissue of the epidural space. The typical abdominal collateral artery was divided into ascending and descending terminal branches, which entered to the center of the rear of the two adjacent vertebral bodies oblique upward and downward, respectively, and penetrated into the vertebral body at the deep surface of the posterior longitudinal ligament. Therefore, each vertebral body received a blood supply from four arteries from the rear: two on each side, one upper, and one lower.

Venous return to the spine

The veins of vertebra constitute the vertebral venous plexus, which is the same length as the spine, and is divided into two parts: the internal vertebral venous plexus and the extravertebral venous plexus. The internal vertebral venous plexus is located in the spinal canal, is densely distributed between the dura mater and the periosteum, and is divided into two parts, each with two longitudinal venous trunks and many anastomotic branches. The intravertebral venous plexus collects venous blood from the vertebra and spinal cord and passes it into the intervertebral vein (segmental vein) at the intervertebral foramen. The external vertebral venous plexus is also divided into two parts: anterior and posterior. These parts collect venous blood from the vertebra and surrounding soft tissues. The internal vertebral venous plexus is connected with the extravertebral venous plexus at the gap between the intervertebral foramen and the ligamentum flavum on both sides, and is also connected with the intracranial basal venous plexus at the foramen magnum. Therefore, inflammation of the posterior abdominal wall and lumbar back can spread to the brain through the vertebral venous plexus at any time^[3].

Blood supply to the cartilage endplate

The main role of the cartilage endplate is load-bearing. It also participates in nutrient exchange and infiltration of intervertebral discs. The nutrient exchange channel and osmotic function of the cartilage endplate are closely related to spondylitis. Although the blood vessels passing through the cartilage endplate are occluded in adulthood in humans, many pores in the endplate remain open, which can maintain the characteristics of the semi-permeable membrane of the cartilage endplate^[4]. This particularity enables small molecular components, such as water, to diffuse with changes in osmotic pressure. Under a scanning electron microscope, the vascular buds, which did not penetrate the interface between the cartilage endplate and nucleus pulposus, were still densely distributed in the cartilage endplate. In the area near the nucleus pulposus, these small vascular buds were more densely distributed. These small blood vessels were intertwined in the form of branches and had expanded branches. Along with these microvascular buds, many tiny lymphatic vessels existed. There were also dense microvessels, blood sinuses, and lymphatic capillaries at the two ends of the vertebral body adjacent to the cartilage

endplate, especially at the central part of the cartilage endplate. After bacteria enter the human body, because of the abundant blood supply of the vertebral endplate, this site is the first to be invaded by bacteria. Bacteria then further invade the intervertebral disc and vertebral body, causing discitis and vertebritis. The vascular supply of intervertebral discs changes significantly with age. Infants and children have a more abundant vascular supply compared with adults. The capillary networks at the edges of vertebra adjacent to intervertebral discs are denser in infants and children compared with adults. Several blood vessels penetrate deep into the infant's intervertebral disc, which are not present in most adolescents and all adults^[5].

Occurrence and development of spinal infection

Embolism is caused by bacteria staying in the terminal vertebral artery. At present, the most commonly accepted hypothesis for vertebral bone infection is that the infected bacterial embolus diffuses in the arterial system and stays in one place to form a bacterial mass, resulting in arterial infarction and subsequent infection. Because of the abundant capillary networks at the edges of vertebra adjacent to intervertebral discs and the number of arteries at this position, bacteria often stay here, and osteomyelitis is the most common occurrence at the edge of the vertebral body. In adults, the epiphyseal artery is the terminal artery, and septic embolism can lead to a large area of purulent infarction. Then, the infection may extend to the contralateral vertebral endplate through the periosteal aorta or through the intervertebral artery to the endplate artery of the adjacent vertebral body, leading to infection in the adjacent vertebral body, which does not necessarily affect the central part of the vertebral body.

Another route of bloodborne transmission is the venous route. The epidural venous plexus in the central canal is a series of valveless veins. Inflammatory bowel disease, urinary tract infection, and pelvic infection spread to the vertebral body through venous blood. In cases of penetrating trauma or direct exposure related to skin rupture and open wounds, bacteria may be directly inoculated in the spine. Spinal surgery, negligent exposure related to non-spinal surgery, and secondary hospital infection are also sources of spinal infection^[6].

In both children and adults, epiphyseal infection can affect the corresponding intervertebral disc. Because the adult intervertebral disc is almost without a vascular supply, there is no immediate bloodborne immune defense mechanism. Infection occurs easily and develops rapidly. Bloodborne pathogens mainly involve intervertebral discs. In children with vertebral infection, because of the continuous blood supply of intervertebral discs, the chance of disc infection is reduced. Sometimes it may only present as discoid inflammation. Children may experience abdominal pain, which is often their initial symptom, when the anterior longitudinal ligament is pulled during lumbar extension. Prevertebral and paravertebral abscesses are caused by continuous spread of infection from the spine to adjacent soft tissues^[7].

In addition, involvement of paravertebral veins provides a route for abscess formation; thus, infection can enter adjacent soft tissues, and abscesses can form from phlebitis or thrombophlebitis. In extensive paravertebral inflammation, secondary involvement of adjacent vascular structures, such as the aorta,

may lead to vasculitis and mycotic aneurysm. In the middle and late stages of disease, if the abscess around the vertebral body is not completely absorbed, granulation tissue can be produced around the injured vertebrae, which is evenly distributed around the vertebral body. Sometimes we can see formation of the epidural mass behind the vertebral body, which is an equal strength mass formed by the vertebral cortex, posterior longitudinal ligament, dura, and cerebrospinal fluid. With T2WI, subdural effusion may be equal to the high signal intensity of cerebrospinal fluid, so it may be missed at diagnosis. At this time, the image can be added to differentiate between epidural abscess and subdural effusion. In the absence of detectable bone or disc disease, hematogenous dissemination of purulent organisms rarely causes an epidural abscess alone.

Pathophysiology of spinal disease

Spinal tuberculosis

Spinal tuberculosis is a chronic and destructive disease, which tends to be in a relatively late stage when symptoms appear^[8]. The morphological and pathophysiological changes of spinal tuberculosis are the most serious changes observed in infectious spondylitis. Tuberculosis is most common in thoracic vertebra, followed by lumbar and cervical vertebra. Infection begins in the vertebral body, and the most common site is the anterior and upper part of the vertebral body. Tuberculosis can selectively affect part or all of the vertebral body (pedicle, lamina, posterior spinous process) without involving adjacent intervertebral discs^[9]. Mycobacteria lack proteolytic enzymes to digest the nucleus pulposus, which can avoid destruction of intervertebral discs in the early stages of disease. In the late stages of disease, with the destruction of the rich blood supply area by *Mycobacterium tuberculosis* and its impact on marginal blood vessels supplying nutrition to the intervertebral disc, the intervertebral disc will lose its nutritional supply and undergo pathological changes. In other words, intervertebral disc damage will occur only when spinal tuberculosis infection is relatively advanced.

Cancellous bone is abundant in the vertebral body. When spinal tuberculosis occurs, damage occurs to the vertebral body. The vertebral endplate is easy to damage, and when bone destruction is serious, compression fracture will occur. Infection involves the tissue around the vertebral body and formation of paravertebral abscess. When liquefaction of the cheese-like material in spinal tuberculosis occurs, a localized abscess will form on the periosteum on one side of the vertebral body, and periosteal hyperplasia and granuloma formation will occur under inflammatory stimulation. If inflammation cannot be controlled in time, pus may continue to peel off the vertebral periosteum, the abscess will continue to increase in size, and the periosteum will burst. The huge abscess will flow to different places through the space. A cervical tuberculous abscess can form a retroesophageal abscess, and a lumbar tuberculous abscess can flow to the anteromedial thigh. Spinal tuberculosis is associated with bone abscess, but paraspinal soft tissue abscess is often beyond the scope of the affected vertebral body and intervertebral disc^[10].

Suppurative spondylitis

Staphylococcus aureus, *Escherichia coli*, *Salmonella*, *Pseudomonas aeruginosa*, and *Klebsiella pneumoniae* are common pathogens of spinal infection. *Aureus* is the most common pathogenic bacterium that causes suppurative spondylitis, which mostly occurs in adults. It is mainly associated with bloodstream infection, and a few cases are caused by trauma, intervertebral disc surgery, lumbar puncture, and acupuncture. The onset of disease is acute, and symptoms are obvious, often involving chills and high fever and lower back pain with thorn process percussion pain^[11].

Suppurative spondylitis progresses rapidly, and MRI signal changes can be found after 1 week. Early lesions easily diffuse the entire vertebral body, which can lead to osteolytic bone destruction in different areas of the vertebral body. In the early stages, the pathogen can easily affect the intervertebral disc, mainly because during inflammation, a large number of neutrophils secrete proteolytic enzymes, which directly dissolve and destroy the intervertebral disc. *Aureus* spondylitis often appears in the early stages of paravertebral soft tissue signal abnormalities, is prone to paravertebral abscess and can present with a number of small abscess foci. The main reason is that the abscess under the action of proteolytic enzymes is more likely to spread to paravertebral tissue, but the range is generally not more than the height of the diseased vertebral body^[12].

Expansion of adjacent vertebral body lesions is caused by direct invasion of intervertebral discs. Plain MRI findings of hematogenous suppurative spondylitis are characteristic and can reflect pathophysiological changes. If collapse and deformity are not observed in the spine, early suppurative spondylitis is usually limited to the spinal marrow and does not extend to the paravertebral region. In the early stages of spondylitis, bone marrow edema in the vertebral body is first found on MRI, which shows a low signal on T1WI and a high signal on T2WI. After using an enhancer, the signal difference increases. In the early stages of infection, lesions begin to occur below the endplate. As the disease progresses, it gradually involves the intervertebral disc between the endplate and the involved vertebrae. Early discitis may be difficult to detect, and normal discs show a high signal intensity on T2WI. The decrease in T2WI signal of intervertebral discs can be regarded as a reliable early sign of an improvement in discitis.

Bone marrow edema adjacent to the endplate may be a part of normal intervertebral disc degeneration. In a severely degenerated spine, intervertebral disc degeneration produces edema or fluid in the intervertebral space. At this time, T2WI shows a high-intensity signal, accompanied by signal changes in the endplate. However, careful examination of the cortex of the endplate without erosion should indicate presence of degenerative changes in the spine rather than intervertebral disc infection. Fat-suppression techniques combined with intravenous contrast agents have recently been recommended because they can improve the significance of enhanced structures by eliminating signals from adjacent adipose tissue^[13].

***Brucella* spondylitis**

Brucellosis is an infectious and allergic disease caused by the *Brucella* genus. Cattle, sheep, and other livestock are the main sources of infection. Bacteria enter the human body through the skin, respiratory

tract, digestive tract, conjunctiva, and other routes. The incidence rate in pastoral areas is high, but with the development of population mobility and non-pastoral farming, and an increase in the number of mutton-eating people, the trend of spread to pastoral areas is increasing.

The upper endplate of the vertebral body has a rich blood supply, which is the first site of *Brucella* invasion. When the cells further invade the intervertebral disc and vertebral body, discitis and vertebritis occur. With discitis, the intervertebral space becomes narrow and bone near the upper and lower edges of the vertebral body is damaged. Bone destruction in *Brucella* spondylitis is limited and mild. At the same time, there is new bone formation. The speed of bone formation and repair is faster than bone destruction. Bone hyperplasia of the vertebral periosteum is characterized by lip-like protrusions of the vertebral body edge, and osteophytes are formed nearby. A bone bridge is gradually formed in adjacent vertebra, and the edge of the vertebral body presents a "lace-shaped" change. In general, the state is relatively complete or only slightly wedge-shaped. *Brucella* can also invade the soft tissue around the vertebral body, forming paravertebral abscess and intraspinal abscess, which compresses the spinal cord, nerves, and cauda equina, resulting in corresponding neurological damage. Multiple granulomas and multiple small purulent foci can coexist^[14].

MRI is a reliable follow-up tool in the treatment of suppurative spondylitis. As spinal infection improves, the abnormal high signal intensity on T2-weighted sagittal images of infected vertebral bodies gradually decreases, and the signal intensity on T1-weighted images gradually increases, which suggests that cellular bone marrow is replaced by fat, indicating healing^[15].

Pathological fracture of the vertebral body may occur when tumor growth invades vertebral bone. Pathological fracture is characterized by compression and deformation of the involved vertebral body to varying degrees, which can bulge to the four sides and cause obvious kyphosis. Discoid fracture may involve chronic uniform compression of trabecula or cortical structures when all vertebral bodies are eroded by the tumor. The inverted wedge fracture is due to the abundant blood supply of the posterior upper quarter of the vertebral body, which is also the site of early metastasis. In the process of metastatic invasion, the load-bearing time of the lesion is long, so compression occurs relatively early and is more marked. At the same time, it can be accompanied by enlargement and deformation of involved vertebral appendages^[16].

Second, compression of the spinal canal, spinal cord, and nerve root can be observed. The results show that the epidural mass protruded into the spinal canal, and epidural fat was compressed, deformed, and even disappeared. The subarachnoid space was compressed and occluded; the spinal cord was mostly compressed, with varying degrees of compression and displacement. Compression of lateral recess could lead to stenosis.

In addition, a paravertebral soft tissue mass can be observed. The mass is often connected with the vertebral body, with the lesion at its center, and the upper and lower range is limited. The larger soft tissue mass is located on the side of the junction between the vertebral body and the appendix, and its signal is

similar to that of vertebral body disease. The intervertebral disc was rarely involved in the metastatic tumor, so there was no obvious change in the intervertebral space. This may be related to the avascular structure of the hyaline cartilage endplate, fibrocartilage ring, and nucleus pulposus. Metabolism of the intervertebral disc is realized by diffusion of the endplate. The cartilage endplate and fibrocartilage ring act as a barrier to tumor expansion^[17].

Bacteria and tumor cells have distinct characteristics and different manifestations of spinal invasion. Combined with clinical manifestations, we should carefully identify differences in spinal diseases using MRI to facilitate early drug intervention and unnecessary invasive operations. This study found that MRI can provide early diagnostic evidence for spinal lesions.

Conclusion

Early spinal MRI can distinguish between spinal tuberculosis, suppurative spondylitis, *Brucella* spondylitis, and spinal tumor, which avoids delays in drug therapy before the pathological results of spinal lesions become available. Careful analysis of MRI images and the clinical manifestations of disease are prerequisites for a correct diagnosis.

Declarations

Ethics approval and consent to participate

The Institutional Review Board of Department of Orthopaedics, Fujian Medical University Affiliated Quanzhou First Hospital approved this retrospective study and waived, the requirement for written informed consent due to its retrospective nature.

Consent for publication

Not applicable.

Availability of data and materials

The datasets analyzed in this study are available from the corresponding author on request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Jianhui Shi: contributed to the conception of the study;

Qiaofeng Chen:performed the data analyses and wrote the manuscript;

Shanpeng Wu :helped perform the analysis with constructive discussions;

Tianxiang Lu:contributed significantly to analysis and manuscript preparation;

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Figures

Figure 1

MRI of lumbar Brucella infection. (A)-(C) Sagittal position of lumbar spine (A) T1 image, (B) T2 image (C) T2-weighted FSE image (D) Coronal position of lumbar spine (E) Horizontal position of lumbar spine.

Figure 2

MRI of suppurative spondylitis. (A)-(D) Sagittal position of lumbar spine (A) T1 image, (B) T1 enhanced MRI image (C) T2 image (D) T2-weighted FSE image (E) Horizontal position of lumbar spine. /

Figure 3

MRI of spinal tuberculosis. (A)- (B) thoracic sagittal position view. (A) T1 image, (B) T2 image (C) thoracic coronal position view. (D) thoracic horizontal position view.

Figure 4

MRI of spinal tumors. (A)-(D) Sagittal position of thoracic vertebrae: (A) T1 MRI image, (B) T1 enhanced image (C) T2 image (D) T2 enhanced image (E) – (G) Sagittal position of lumbar vertebrae. (E) T1 image, (F) T1 enhanced image (G) T2 image. (H) Horizontal position of lumbar spine.