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Review Article

Synovial chondromatosis of the temporomandibular joint

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ABSTRACT

When cartilage-like tissue is present in the synovial membrane of joints, it is known as synovial chondromatosis (SC). The main cause is believed to be the metaplasia of embryonal mesenchymal tissue in the synovial membrane. The metaplastic foci grow within the joint space and can detach from the synovial wall, forming free-floating bodies. While SC is more commonly associated with trauma in the temporomandibular joint (TMJ), the exact relationship between trauma and SC remains unclear. Improved diagnostic techniques, such as CT and MRI, have led to increased recognition of SC in the TMJ. Gender differences in the prevalence of TMJ SC have been observed, with women being more affected. Diagnosis involves imaging techniques like X-rays, CT scans, MRIs, and surgical removal of loose bodies is the preferred treatment. Arthroscopy is recommended for small, isolated loose bodies, while open surgery may be necessary for complete removal. Long-term follow-up is essential to monitor for recurrence. Histopathological findings reveal a mixture of cartilage and bone, with ossification and calcification present.

Keywords: SC, Cartilage-like tissue, Metaplasia, Joint space, Trauma, Diagnosis, Surgical removal

INTRODUCTION

A condition known as SC primarily affects the large limb joints, specifically the knee, hip, and elbow. In 1958, Jaffe provided the first histomorphologic description, which described the disease's histologic diagnosis.[1] Along with fibromatosis and nodular fasciitis, SC is classified as a tumorous lesion of the tendon and fascia. It belongs to the group of chondrogenic neoplasms that develop outside of the skeleton, together with soft tissue chondroma and chondrosarcoma.[1] Pain, restricted mouth opening, preauricular edema, malocclusion, crepitus, and a clicking sound when opening the mouth are the most typical clinical indications and symptoms of SC in the TMJ.[2] Georg Axhausen[1] provided the first precise, scientific description of SC of the temporomandibular

joint in 1993, referring to it as metaplastic chondrogenesis in the synovial membrane. SC nomenclature is inconsistent. This clinical disease is also known as synovial chondrometaplasia, osteochondromatosis, and synovial chondrosis. Early diagnosis of SC is quite uncommon due to the low prevalence of the illness in the temporomandibular joint area. It frequently takes several months or even years before the diagnosis is established. Uncertain factors may be contributing to the temporomandibular joint disorder. The detachment of chondrocytes in the embryo contrasts with true secondary reactive metaplasia following trauma or persistent aberrant loading. Traditional x-ray examinations, computer tomography, magnetic resonance imaging, and arthroscopy of the temporomandibular joint are all examples of imaging diagnosis. Usually, only after an arthroscopic or open examination of the joint and a histopathologic analysis can the final diagnosis be determined. Three phases in the development of SC of the temporomandibular joint were documented by Blankenstijn et al.[3] In first stage, the synovial membrane undergoes metaplasia without loose bodies. The second phase sees the loose bodies detached as a result of increasing metaplasia. These are partly encircled by a synovial membrane and contain active chondrocytes. The synovial membrane area no longer exhibits metaplastic activity in the final stage. It is possible for the loose bodies to age and calcify. The recommended course of treatment is an arthrotomy of the afflicted temporomandibular joint and excision of the loose bodies. Some writers advise synovectomy, including diskectomy, in situations with villous-cartilaginous harm to the synovial membrane to stop recurrence.[1] SC of TMJ cannot resolve on its own and does not improve with conservative therapy. The only effective therapy for SC in TMJ is surgery.[4] A recurrence of the lesion is possible if it is not completely removed.[5] However, after arthrotomy in big joints, a 30% SC recurrence incidence has been documented.[6] This review article will discuss SC of the temporomandibular joint.

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on 11 June, 2023 in the Medline, PubMed and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed SC of the temporomandibular joint. There were no restrictions on date, language, participant age, or type of publication.

DISCUSSION

The cartilagenous metaplasia of embryonal mesenchymal tissue that remains in the subintimal layer of the synovial membrane is the most widely recognised idea about the cause of SC.[7] All intracapsular structures, such as ligaments, tendons, and fibrocartilage, are produced from embryonic synovial mesenchyme, which comprises synovial and sub-synovial tissues. A villous process of the synovium acts as a pedicle for some of these metaplastic foci, which are fed by the blood supply of the synovial tissue and nutrients in the synovial fluid. The foci form a perichondrium which allows the chondrocytes to grow by proliferation. Calcification of the cartilaginous foci occurs in advanced stages of the disorder, and, as growth continues, they detach from the synovial wall and become free-floating bodies within the joint space. A number of authors suggest that SC of TMJ can be triggered by trauma despite the lack of association with trauma in other joints. The difficulty in establishing a relationship with trauma is increased because symptoms may not present for weeks or months after the traumatic episode Traumatic episodes can dislodge cartilagenous tissue, bony tissue, or both, into the joint space where they may lodge in synovial tissue. If these fragments are then overgrown by a layer of synovium, nourishment is provided and the cells form distinct calcified bodies.

TMJ was assumed to be infrequently impacted by SC. However, the reports of SC in TMJ have grown in recent years, which may be connected to a better diagnostic rate utilising CT and MRI and the improvement of doctors' analytical skills.[8,9] Women were more likely to experience TMJ SC, whereas men were more likely to experience SC in the larger joints.[10,11] The prevalence of SC in TMJ was gender-specific and comparable to that of TMD.[12,13] It may be connected to the way oestrogen affects TMJ. Most SC in TMJ happens on one side of the patients.[14] The majority of publications state that SC is typically only present in the upper compartment.[2] Seven examples of SC in the lower compartment of TMJ were documented in three articles.[15,16] The narrower joint compartment and smaller synovial membrane area in the lower compartment of TMJ may be to blame for the low prevalence rate of SC there.[17] A clinical sign of tumour-like diseases, including diffuse synovial giant cell tumours, synovial cysts, and SC, is preauricular edema.[18] It is challenging to distinguish SC from TMD or osteoarthritis because of the other vague symptoms. SC has a local invasive behaviour that can cause bone erosion by affecting the glenoid fossa, disc, and condyle and potentially extending into the brain later on.[19] As a result, it is challenging yet crucial to treat and discriminate against SC in the beginning. X-rays, CT scans, and MRIs are crucial diagnostic methods for identifying SC in TMJX-rays and CT scans can reveal multiple granular calcification shadows, sclerosis of the condyle and glenoid fossa, and changes to the joint spaces.[2] The loose body is the most valuable diagnostic sign. However, SC in Milgram's stage 1 or 2 lacks calcified loose bodies, which provides difficulty in CT or X-ray-based diagnosis, wrong treatment, expansion of lesion, and delay of surgery. Uncalcified loose bodies, joint capsules, disc position, effusion, and invasion of neighbouring tissues

can all be seen on MRI scans. The major symptoms of SC in TMJ are synovial thickening, joint capsule expansion, cartilage nodules, or calcified loose bodies presenting low signal intensity dispersed in the effusion that provide high signal intensity. MRI has a significant reference value for the diagnosis of SC in TMJ. Given that MRI has an estimated diagnostic accuracy of roughly 96.06%, it may be a viable diagnostic technique for identifying SC in TMJ.[2] It is important to identify SC in TMJ with calcified loose bodies from osteoarthropathy, exfoliative osteoarthritis, chondrosarcoma, and osteosarcoma. When an image is inconclusive, an arthroscopic examination aids in the diagnosis by identifying specific SC features, including the following: villous or polypoid nodular protrusions on the hyperplastic synovial membrane; and the synovial membrane without metaplasia is a papillary protrusion-like aggregation, with a pink or bright red colour because it is rich in capillaries.

The use of the arthroscope has therapeutic benefits. For starters, resection was restricted to the synovium that was affected rather than necessitating open arthrotomy, which is a far more invasive procedure. Second, an open arthrotomy would not have been able to examine or treat the particles due to their size and placement.[7] For instance, they are likely that the particles at the medial superior capsule wall would have been impossible to identify or reach in an open surgery. The process is influenced by the particle size. The existence, size, and position of any particles discovered during the arthroscopy diagnostic process will have been determined, even if they are too large to be removed using arthroscopic tools. However, switching to a bigger cannula system could offer sufficient clearance for the removal of large particles.[7] Typically, a 2.0-mm cannula is used with a working instrument with a diameter of 1.7 mm. For the removal of larger loose bodies, a 3.0 mm cannula system with 2.6 mm to 2.9 mm instrumentation may be employed. For the removal of loose bodies, 2.0 mm or 3.0 mm cannulas can be used with biopsy forceps, disc graspers, and motorised shavers. For orthopaedic surgeries, methods for removing big pieces that do not fit via the cannula system have been described; however, in a tiny joint like the TMJ, an open arthrotomy will likely be necessary for the full removal of such fragments. This does not rule out the potential for approaches to be created in the future for this purpose.[7] The surgeon can choose the examination techniques of X-ray, CBCT, MRI, and arthroscopy based on their ease of acquisition, the complexity of the diagnosis, the trauma the examination causes, and the patient's approval of the procedure. The only effective therapy for TMJ-SC is surgery. Arthroscopy, synovectomy, removal of loose bodies, discectomy, etc. are all available as treatments. Because arthroscopy provides the benefit of minor trauma, some academics advise using it to treat SC.[2] Additionally, arthroscopy has a broad field of view and can uncover abnormalities in the joint cavity that are difficult to see directly. However, arthroscopy is only appropriate in situations where there are only small, isolated, loose bodies (3 mm) in one compartment. According to reports, arthroscopy can only partially remove loose bodies and aberrant synovial membranes, but open surgery can effectively remove both of these conditions. Open surgery makes it challenging to directly see the fossa, particularly the anteromedial portion of the fossa. To have a clear view of the fossa and remove the disease entirely, a condyle osteotomy and/or zygomatic arch amputation may be done. For SC in TMJ, open surgery and arthroscopy have also been mentioned. This method's benefit is the total removal of lesions with little osteotomy and a speedy recovery. For instance, arthroscopy was chosen since it is an established method in our clinical setting and enabled us to clearly remove the lesion while preventing the condylar from being amputated. According to certain publications, surgical techniques should be chosen based on Milgram's stage of SC. Milgram's stage 1 instances necessitate a thorough resection of the synovial tissue. In stage 2, it is necessary to clean the synovial tissue and loose bodies. In stage 3, only loose bodies or masses ought to be eliminated. Except for the removal of the loose bodies and aberrant synovium, further surgical operations, such as discectomy, costochondral graft, and deep temporal fascial fat flap, are occasionally necessary. It appears that the kind of surgery was chosen based on the surgeon's training and the structures of the SC involved. Long-term follow-up is required to rule out postoperative recurrence, even though SC has a very low postoperative recurrence rate. Early recurrence is primarily caused by an insufficient synovectomy. The glenoid fossa or condyle's residual minute bone erosions may be the root of a long-term recurrence. SC recurs because of aggressive metaplasia on the small bone degradation.[20] Since MRI is more sensitive to SC diagnosis and early detection is crucial for choosing the best course of therapy and offering a better prognosis, MRI should be conducted both before and after surgery. In some cases, even condylectomy has been performed to get a better view of the medial aspect of the joint. More modern surgical techniques have made this kind of approach unnecessary.

Histopathological findings from study suggest that all the loose bodies were surrounded by a thin layer of cellular fibrous tissue.[21] Most of them consisted of a mixture of cartilage and bone in variable proportions. Enchondreal ossification and calcification could be found in the majority of the particles. Some areas of the chondroid tissue showed a considerable cellular and nuclear plemorphism, and even binucleated cells could be encountered. A few bodies were composed only of mature lamellar bone with fatty marrow tissue. Furthermore, resorption laqunae with giant cells of the osteoclast type were present in some bony areas. Histological examination of the disk revealed dense hyalinized fibrous tissue without any sign of inflammation. Within the fibrous tissue, there were several nodes of metaplastic bone with lamellar architecture in the center. In the synovial membrane, cartilage nodules of various sizes can be observed. These

nodules are frequently surrounded by fibrosed connective tissue that has a sparse lymphoplasmacellular infiltrate. On occasion, a thin synovial membrane layer covers the surface of the loose bodies. Cellular pleomorphism and increased proliferation are well recognised, although malignant degeneration is quite uncommon.

One study reported operative findings consisting of loose bodies which were predominately white in colour, firm or hard in consistence, and rice- or pearl-like in shape usually with a smooth or sometimes a lobulated surface.[21] In a few instances, their surface was rough. Some of the nodules loosely attached to synovium.

CONCLUSION

SC is characterized by the presence of cartilage-like tissue in the synovial membrane of joints. The main cause is believed to be the metaplasia of embryonal mesenchymal tissue in the synovial membrane. Metaplastic foci grow within the joint space and can detach from the synovial wall, forming free-floating bodies. SC is more commonly associated with trauma in TMJ, although the exact relationship remains unclear. Improved diagnostic techniques such as CT and MRI have led to increased recognition of SC in TMJ, with women being more affected than men. Diagnosis involves imaging techniques such as X-rays, CT scans, and MRIs, while surgical removal of loose bodies is the preferred treatment. Arthroscopy is recommended for small, isolated loose bodies, while open surgery may be necessary for complete removal. Long-term follow-up is essential to monitor for recurrence. Histopathological findings reveal a mixture of cartilage and bone, with ossification and calcification present. Further research and exploration of surgical techniques are needed to optimize the management of SC in TMJ.

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