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# Clinical Presentation of Cat Scratch Disease in Pediatric Patients—A Single-Center Study

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## What is already known on this topic?

- *Diagnosis of CSD has always been challenging and is performed by using combination of epidemiological, bacteriological, and histological criteria.*

## What this study adds on this topic?

- *Regional, especially axillary, lymphadenopathy unresponsive to non-specific antibiotic treatment should raise suspicion for CSD. Lymphadenopathies in CSD if not treated properly enlarge and become cystic suppurative lesion; their common ultrasonographic findings are conglomerated lymph nodes with lobulated contours and cortical thickening usually without calcification. Azithromycin, the first choice agent in CSD treatment, is very effective antibiotic. However, in some cases, it may be necessary to extend the duration of azithromycin treatment or to add other antibiotics and/or steroids to the treatment, such as in disseminated disease.*

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## ABSTRACT

**Objective:** Cat scratch disease (CSD) is the infectious disease caused by *Bartonella henselae*. Its typical presentation is regional lymphadenopathy. Also it may present with symptoms related to involved organs or disseminated disease with fever of unknown origin (FUO). Here children with CSD are evaluated to increase awareness about disease.

**Materials and Methods:** A total of 29 children diagnosed with CSD between 2019 and 2022 were involved in the study. Patients' demographic characteristics, clinical, laboratory and radiological findings, treatments, and outcomes were analyzed.

**Results:** Seventeen of the patients were male, 12 were female, and their mean age was 116.5 ± 51 months. About 69.6% of them had a history of cat contact. Twenty-seven patient (93.1%) had lymphadenopathy, mostly axillary involved (61.5%). Other manifestations were disseminated disease presented with FUO, neuroretinitis, and encephalopathy. Twenty-seven patients (93.1%) had received antibiotics before admission without any improvement. Ultrasound showed that the affected lymph nodes were conglomerated, lobulated contoured, and cortical thickened, with one-third having cystic suppurative components. Serologic tests were positive in 24 of 27 patients. Twenty-one patients gave response to 5 days azithromycin treatment, in 8 patients this treatment extended to 10-14 days, rifampicin with/without doxycycline was given to 6 patients, and steroids were given to 3 patients.

**Conclusion:** In case of regional lymphadenopathy, especially axillary, not responding to non-specific antibiotics CSD should be suspected. Cat contact history and serological and ultrasonographic findings are useful for diagnosis. Even if CSD responds well to azithromycin, sometimes prolongation of azithromycin and addition of other antibiotic or steroid may be required.

**Keywords:**Cat scratch, *Bartonella henselae*, children

## INTRODUCTION

Cat scratch disease (CSD) was first described in 1931,[1] it is a zoonotic infectious disease characterized by subacute regional lymphadenopathy. The most common etiologic agent is *Bartonella* spp., a small gram-negative bacterium. There are about 25 species of the *Bartonella* and about half of them are confirmed as human pathogens. *Bartonella henselae* is the most common serotype causing CSD. Rarely other *Bartonella* spp. have been reported to cause this disease.[2] The main reservoir of *B. henselae* are cats, and transmission to human occur mainly via scratches or bites of infected cat. So, history of close contact with cat is significant for diagnosis but not always necessary.

Classic presentation of CSD is lymphadenopathy near the inoculation site. Three to 10 days after inoculation, disease usually begins with a single or group of erythematous papules

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called "primary lesion" which persist 1-3 weeks.[3] Regional lymphadenopathies usually emerge 2 weeks after inoculation. Infected lymph nodes are characteristically tender and have erythema of the overlying skin, and about 5-10% of them suppurate.[4] In case of disseminated disease, patients have fever, abdominal pain, hepatosplenomegaly, and weight loss, and rarely disease can be life threatening. Other clinical manifestations are usually related to visceral organ involvement like neuroretinitis, encephalitis, or endocarditis.[3]

Today, probable diagnosis of CSD can be made in the presence of typical clinical features with a recent history of cat scratching or contact. Additionally, serological tests and if diagnosis is uncertain and tuberculosis or lymphoma are in differential diagnosis, lymph node or tissue biopsy can be performed. Early diagnosis of CSD is important when it is in the differential diagnosis of the diseases that require invasive interventions for diagnosis, such as mycobacterial infection and malignancy. Therefore, physician awareness about disease is critical for early diagnosis. Here we wanted to increase awareness of physician about disease by evaluating demographic features, clinical laboratory and radiologic findings, treatments, and outcomes of the children with CSD to make early diagnosis possible.

## MATERIALS AND METHODS

### Study Design

This retrospective descriptive study was conducted in the Medeniyet University Göztepe Prof Dr. Süleyman Yalçın City Hospital, a hospital providing tertiary health care services. Children aged between 0 and 18 years, diagnosed with CSD by pediatric infectious disease specialist between September 1, 2019, and December 31, 2022, were involved in the study. We identified patients who had an International Classification of Diseases code (ICD) for CSD or bartonellosis (ICD10 A28, A28.1, A44.9) and underwent serology and/or PCR testing for *B. henselae* infection. Then the medical records of these patients were reviewed for their demographic features, animal exposure, disease associated symptoms, physical examination findings, imagining results, serology, histopathologic results, antibiotic treatment, and overall outcomes. Finally, we included patients with clinical features compatible with CSD and with at least 1 positive serological, histopathological, or molecular result.

### Inclusion and Exclusion Criteria

Patients with clinical manifestation compatible with CSD and had either positive B. henselae enzyme immunosorbent assay (EIA, B. henselae IgG 1:64 with/without IgM 1:16) or positive the indirect fluores- cence assay titer (IFA, B. henselae IgG >1:64), or positive B henselae real-time polymerase chain reaction (rt-PCR) on lymph node biopsy, abscess fluid and/or other biopsy material; his- topathological examination suggesting CSD with granulomatous inflammation with/without satellite abscess formation were included to study. Patients' serological tests were done by reference clinical laboratory.

This study was approved by decision of Clinical Research and Ethics Committee of Medeniyet University Göztepe Prof. Dr. Süleyman Yalçın City Hospital (approval number: 0558; date: September 21, 2022).

### Statistical Analysis

The statistical analyses were performed using SPSS Version 21.0 (IBM SPSS Corp.; Armonk, NY, USA). Demographic and clinical characteristics were analyzed using descriptive statistics. While discrete data were summarized as count and percentiles, continuous variables were presented as mean and SD or median and interquartile range (IQR) where appropriate.

## RESULTS

### Patient Characteristics

The 29 patients were diagnosed with CSD in about 3 years. Their median age was 116.5 ± 51 months (14-203) of them 17 (58.6%) were male. Three of them had comorbidity; intracranial mass, ataxia telangiectasia, and idiopathic thrombocytopenic purpura. Sixteen of 23 patients (69.6%) had cat exposure, 3 (13.0%) had dog exposure. Median time between contact and diagnosis was about 2.7 months. Four patients (13.8%) described inoculation lesion; among them, 1 was observed at admission (Figure 1). All patients had received antibiotics before admission, mainly beta lactam antibiotics.

Twenty-six patients presented with lymphadenopathy (89.7%), 1 presented with fever (3.5%), 1 with seizures (3.5%), and 1 with blurred vision (3.5%). On physical examination, 12 patients had axillary lymphadenopathy (46.2%), 3 had axillar and cervical (11.5%), 1 had axillary and antecubital, 8 had cervical (30.8%), 1 had antecubital, and 1 had supraclavicular lymphadenopathy (Table 1). Eighteen of the 26 patients (62.1%) had tenderness and erythema on the lymphadenopathy. One patient had a fever lasting for more than 3 weeks with unknown origin despite intensive outpatient examinations for the last week. One patient admitted with convulsion and altered mental status. Another patient was admitted due to blurred vision.

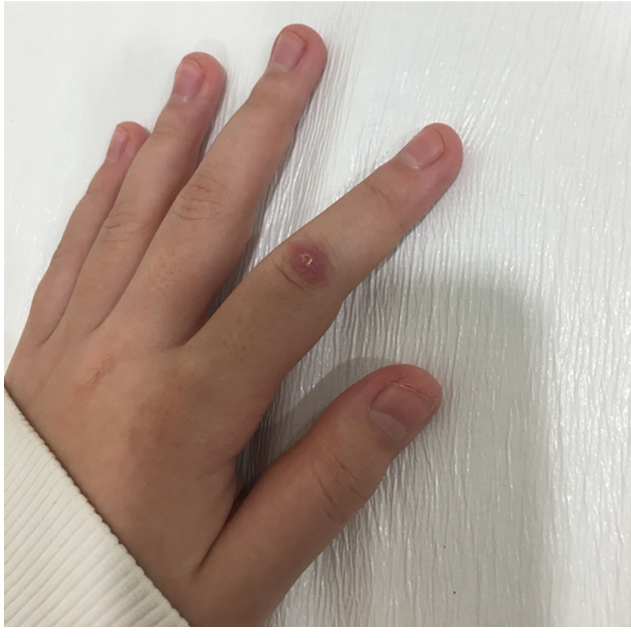

**Figure 1.** Cat scratch disease (CSD) after 4 weeks of scratch, inclusion lesion.

**Table 1.** Demographic and Clinical Characteristics of the Children with Cat Scratch Disease (n = 29)

| Characteristics | Frequency, n | Percent (%) |
|---|---|---|
| ***Sex*** | | |
| Male | 17/29 | 58.6 |
| Female | 12/29 | 41.4 |
| **Age** | | |
| Mean ± SD, months | 116 ± 51 | |
| **Comorbidities** | 3/29 | 10.4 |
| Ataxia-telangiectasia | 1 | |
| Intracranial tumor | 1 | |
| Idiopathic thrombocytopenia | 1 | |
| **Animal exposure** | | |
| Cat | 16/23 | 69.6 |
| Dog | 3/23 | 13.0 |
| No exposure | 4/23 | 17.4 |
| **Time of contact before admission, months** | 2.7 | |
| **Cause of admission** | | |
| Lymphadenopathy | 26/29 | 89.7 |
| FUO* | 1/29 | 3.5 |
| Neuroretinitis | 1/29 | 3.5 |
| Encephalitis | 1/29 | 3.5 |
| **Physical examination** | | |
| Lymph node involvement | 26/29 | 89.7 |
| Axillar | 12/26 | 46.2 |
| Cervical | 8/26 | 30.8 |
| Axillar and cervical | 3/26 | 11.6 |
| Axillar and antecubital | 1/26 | 3.9 |
| Antecubital | 1/26 | 3.9 |
| Supraclavicular | 1/26 | 3.9 |
| Pain and tenderness on lymph node | 18/26 | 69.2 |
| Inoculation lesion at admission | 4/29 | 13.8 |
| Splenomegaly | 4/29 | 13.8 |
| **Laboratory findings** | | |
| White blood count, $mm^3$ | 9.4 ± 3.3 | |
| Neutrophils count, $mm^3$ | 5.79 ± 3.2 | |
| CRP at diagnosis, mean ± SD, mg/L | 16,84 ± 25 | |
| ESR at diagnosis, mean ± SD, mm /hour | 32.13 ± 32 | |
| TST negative | 12/12 | |
| Quantiferon, negative | 4/4 | 100 |
| *B. henselae* IGG > 64 (IFA) | 21/24 | 87.5 |
| *B. henselae* IGG = 64 (EIA) | 3/3 | 100 |
| Positive *B. henselae* PCR | 1/9 | 11.1 |
| Characteristic histopathologic findings | 5/9 | 55.6 |
| **Surgical intervention/biopsy** | | |
| Fine needle aspiration | 2/9 | 0.22 |
| Excisional biopsy | 3/9 | 0.33 |
| Lymph node biopsy+abscess drainage | 2/9 | 0.22 |
| Abscess drainage | 2/9 | 0.22 |
| **Treatment** | | |
| Azithromycin, 5 days | 21/29 | 72.4 |
| Azithromycin, >5 days | 8/29 | 27.6 |
| +doxycycline | 4 | 13.8 |
| +rifampicin | 4 | 13.8 |
| +Methylprednisolone | 3 | 10.4 |

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; TDT, tuberculin skin test.

## Laboratory Findings

Laboratory examination of patients revealed mean white blood count (WBC) 9.4 ± 3.3 /$mm^3$ and mean neutrophil count 5.79 ± 3.2 /$mm^3$. Twelve of 24 patients (50%) had elevated C-reactive protein (CRP) (>5 mg/L) and erythrocyte sedimentation rate (ESR) (>20 mm/h) (Table 1). Serological tests for brucellosis, acute cytomegalovirus, Ebstein-Barr virus, and Toxoplasma gondii infections were negative. *Bartonella henselae* IgG-IFA was performed in 24 patients, 21 had titer >1:64, and in 3 it was 1:64. Two patients with negative results had axillary and 1 had antecubital lymphadenopathy, their diagnosis was made by histopathological examination. *Bartonella henselae* IgG-EIA was performed in 3 patients, and titer was IgG ≥1:64 in all. Tuberculosis skin test (TST) was applied in 12 patients and QuantiFERON test in 5, all of them resulted negative.

One patient was diagnosed with neuroretinitis by the ophthalmologist, she had cat scratch history and positive *B. henselae* IgG test. The patient admitted with convulsion and altered mental status diagnosed with encephalitis, his CSF culture remained sterile and multiplex PCR was negative, and he had no clinical and laboratory signs for other possible etiological causes of meningoencephalitis. He had exposure with street cats and had positive *B. henselae* IgG IFA test in serum. There was no any validated test we can apply to demonstrate *B. henselae* or *B. henselae* IgG in CSF. Clinical response to azithromycin observed with improving of his mental status.

## Imaging Features

Imagining tests results of patients were analyzed. Twenty-six patients who presented with lymphadenopathy revealed conglomerated lymph nodes with lobulated thickened well-defined contours on ultrasound (Table 2). In 11 of them (37.9%) whose lymph nodes size larger than 4 cm, cystic component in lymph node progressed to abscess formation (Table 2). However, no calcification was observed in any affected lymph nodes of patients. Abdominal ultrasound was performed in 19 patients; 1 had hepatosteatosis and 3 had mild splenomegaly (13.8%). Additionally, the ultrasonography of the patient, who was hospitalized with a diagnosis of FUO, revealed axillary lymphadenitis and splenomegaly with cystic lesions. Then this patient was diagnosed as disseminated CSD.

## Management

Surgical intervention was needed for 9 patients because in 5 diagnoses of CSD is uncertain, in 4 axillary lesion was >5 cm in diameter and not respond to medical treatment. The 6 of the patients who had biopsy and/abscess drainage had characteristic histopathologic findings, necrotic granulomatous reaction in some with microabscesses, and also one's biopsy specimen was positive for B. *henselae* PCR. No fistulation was observed in any of them after surgical drainage or biopsy.

Classic 5-day azithromycin treatment was initiated in all patients; it was extended to 10-14 days in 8 patients. Additionally, 6 patients received rifampicin with or without doxycycline. Also, 3 patients received steroids, 1 for neuroretinitis, 1 for complicated lymphadenopathy, and 1 for painful lymphadenopathy in antecubital fossa. Prednisone was initiated with dose of 1 mg/kg (maximum daily dose 60 mg/day) for 5 days and tapered over 2 weeks.

**Table 2.** Sonographic Features of the Lymph Nodes of Children with Cat scratch Disease (n = 25)

| Lymph Node Diameter | Number of Patients (n) | Conglomerated | Cortical Thickness | Lobule | Solid Nodular Lesion | Cystic/Abscess Formation | Calcification |
|---|---|---|---|---|---|---|---|
| <40 mm | 16 | + | + | + | + | – | – |
| >40 mm | 11 | + | + | + | – | + | – |

## DISCUSSION

Cat scratch disease has worldwide distribution. Its incidence varies by region and reported as 6.4 cases/100.000 population in southeastern US.[5] There are no data about its incidence in Turkiye. This study is not appropriate to reflect the incidence in Turkiye as it was conducted during the period when isolation was implemented in the country due to COVID-19 pandemic and many people adopted cats. Additionally, the region the hospital located is a district of Turkiye with a high animal lover and cat population.

Cat scratch disease is most commonly seen in children and young adults. Children under 14 years of age account for 32.5% of all cases in the US, and with the highest incidence observed among children 5-9 years of age (9.4 cases/100 000 population).[6] Similiarly in this study mean age of children was 5-14 years.

Cat scratch disease is a disease generally underrecognized. Here all patients had received non-specific antibiotics before admission with no clinical improvement. And also they described the time of the appearance of primary lesion as approximately 2.7 months before admission. Actually, inoculation lesion usually begins 1-3 weeks after the inoculation of microorganism.[3] Therefore, this long period before diagnosis and inappropriate treatment are sign of delay in diagnosis. History of animal contact has been reported in almost half of the patients with CSD, here about 82.6% of patients had animal contact.[6] So, history of cat contact, presence of inoculation lesion, and unresponsiveness to antibiotic treatment seems as important clues for diagnosis of CSD.

Cat scratch disease characteristically present with regional lymphadenopathy in about 85%-90% of patient.[7] In accordance with this, lymphadenopathy was the most common manifestation of the disease in this study, the axillar lymph nodes were most frequently involved as expected, probably due to hands are the most bitten or scratched area and their lymphatics drain to axillary lymph nodes.[4,8] The second most affected area are cervical lymph nodes. Mazur-Melewska et al[9] reported similar distribution of affected lymph nodes; mainly axillary and epitrochlear lymph nodes (46%), followed by head and neck (26%) and groin (17.5%). In most patients, a single lymph node is affected, and nodes are usually solid, painful, and mobile. However, as the diagnosis is delayed and the disease progresses, the lymph nodes became cystic. Ultrasound findings are not specific but shows multiple, hypoechoic, and highly vascularized nodes and increased echogenicity in the surrounding soft tissue.[9,10] In this study, lymph nodes smaller than 4 cm were mainly solitary, while larger ones were cystic, even had abscess formation. After the treatment, lymphadenopathies may persist for weeks or months.[3]

The other commonly reported clinical manifestation of disease is prolonged fever or FUO.[11] In one study, *B. henselae* infection estimated as 5% of all pediatric cases of FUO.[12] Here 1 patient who had general aches, malaise, and fever for 3 weeks without origin was diagnosed with disseminated CSD after detailed history taking, abdominal ultrasound, and serologic test. In disseminated CSD hepatosplenic manifestation present with organomegaly, fever, and abdominal pain.[13] In accordance to this, our patients' ultrasound revealed axillary lymphadenitis and splenomegaly with cystic lesions. Therefore, it is critical to consider CSD when look for etiology of FUO.

Neuroretinitis and encephalitis were other manifestations seen in this study. Actually following the lymph nodes, the most commonly involved organ is reported to be eye.[13] Ocular manifestations present as neuroretinitis or Parinauld oculoglandular syndrome (bulbar conjunctivitis, preauricular lymphadenopathy, and conjunctival granuloma).[14,15] Neuroretinitis is usually characterized by painless decreased visual acuity, dyschromatopsia, and visual field anomalies due to optic disc edema and macular exudates associated with inflammation.[14,15] We routinely consult our patients for ophthalmological examination when they have disseminated disease or when they have ocular symptoms. One patient consulted us for treatment of CSD presented with a retinal infiltrate on her left eye, diagnosed with CSD neuroretinitis due to the presence of characteristic a macular star and optic nerve edema after ophthalmological evaluation. She was only 3 years old, she probably did not notice or express any symptoms, even though she had. It may be better to routinely consult patient younger than 6 years routinely for ophthalmologic examination, until child can express his symptoms.

Neurologic manifestation made 13.8% of CSD, the most common neurological involvement is encephalopathy.[13,16] One patient was admitted with convulsion following abrupt confusion and disorientation. Even if there was no definitive evidence for CSD encephalopathy, exposure history, positive *B. henselae* IgG IFA test in serum, lack of any other identifiable cause for encephalopathy, and response to azithromycin treatment support CSD diagnosis.

Other rare manifestations are due to cardiac, lung, or bone involvements.[3] These rare situations are usually associated with immunocompromising diseases. We did not observe cardiopulmonary, orthopedic, or skin involvement in either our patients who received chemotherapy due to intracranial tumor or patient with ataxia telangiectasia.

Diagnosis of CSD is difficult with routine laboratory tests and additionally it is difficult to isolate *B. henselae* in culture. For this reason, serological tests, indirect fluorescence assay (IFA), and enzyme immunoassay (EIA) are used.[7] Positive IgM levels

indicate acute disease but it remains positive for a short time, and IgG remains positive for longer time, but its sensitivity and specificity seem to vary between studies.[17] On the other hand, false-positive *Bartonella* serology is 4%-6% in the general population.Recently, PCR, a molecular method, has become more widely used, but although the specificity of this test is high, its sensitivity varies between 40% and 76%.Recently, PCR, a molecular method, has become more widely used, but although the specificity of this test is high, its sensitivity varies between 40% and 76%.[18] Our patients' routine laboratory tests resulted in normal range, mainly IFA IgG levels, were used for diagnosis, 3 patients had *B. henselae* IgG (IFA) titer equal to 1/64, 2 of them had axillary, and 1 had antecubital lymphadenopathy diagnosed with histopathological examination.

In CSD, surgical lymph node excision is rarely needed, it is performed when diagnosis is uncertain or to provide symptomatic relive in case of painful suppurative nodes. In this study, 9 patients undergone biopsy with/without drainage. Although fine needle aspiration is not preferred for having potential to cause fistulation, fistulation was not observed in any of our patients. Histopathological findings of biopsy material depend on the stage of the disease and are not specific; the initial change is lymphoid hyperplasia, followed by stellate granuloma formation. The centers of the lymph nodes become necrotic and acellular, then microabscesses develop and may coalesce at a later stage make abscess.[7] In fact, these stages explain the cystic appearance of lymph nodes on ultrasound as they grow in diameter. In histopathologic examination, Warthin-Starry stain has remarkable role it suggests a diagnosis of CSD if demonstrated *B. henselae* bacilli in areas of necrosis of involved lymph nodes. Unfortunately, it was not done in our hospital. In the case report of Aslan et al, Warthin–Starry staining made diagnosis in absence of microbiologic evidence.[19] *Bartonella henselae* PCR positivity in another's tissue sample established the diagnosis, but it should be known that false-negative results are common when performed after 6 weeks of disease.[20]

Cat scratch disease usually has self-limited course in healthy children, but some develop disseminated diseases so antibiotic treatment is suggested for all children with CSD.[1] Azithromycin, rifampin, ciprofloxacin, doxycycline, and trimethoprim-sulfamethoxazole are commonly used drugs.[21] There is no consensus on which antibiotic should be chosen for treatment, instead, antibiotic choice depends on the case reports, small studies, and expert opinion. In this study, majority of patients were treated with oral azithromycin and had good clinical response, only 6 patients required additional antibiotics and 3 ones required steroid addition. Oral azithromycin treatment is safe and cost-effective, so it could be considered as first-line treatment for uncomplicated patients. Further research is needed to determine optimal therapy options for atypical presentations.

This study has some limitations: due to its retrospective design, the data of some cases could not be obtained and they could not be included in the study. This caused the study group to remain small, reducing the power of our findings. A larger sample, including different manifestation of disease, may provide more information about the nature of the disease and treatment options.

## CONCLUSION

In conclusion, in case of hyperemic, indurated, tender lymphadenopathy unresponsive to nonspecific antibiotic treatment, CSD should be suspected, cat contact history and inoculation lesion should be asked. Also, CSD should be considered in etiology of FUO. Serological tests are useful for diagnosis but cannot exclude diagnosis. Ultrasound will show multiple hypoechoic nodes with increased echogenity in the surrounding soft tissue. If the diagnosis is uncertain, histopathological examination is suggested, and Warthin-Starry staining is helpful in the absence of microbiologic evident. Oral azithromycin treatment is safe and cost-effective, so it could be considered as first-line treatment for uncomplicated patients.

**Availability of Data and Materials:** The data that support the findings of this study are available on request from the corresponding author.

**Ethics Committee Approval:** This study was approved by Ethics Committee of Medeniyet University Göztepe Süleyman Yalçın City Hospital (approval no: 0558, date: September 9, 2022).

**Informed Consent:** Verbal and written informed consent was obtained from the patients who agreed to take part in the study.

**Peer-review:** Externally peer-reviewed.

**Author Contributions:** Concept – S.O.D., Design – S.O.D.; Supervision – S.O.D., K.K.; Resources – S.O.D.; K.K., G.B., F.E.; Materials – S.O.D., K.K., G.B., F.E.; Data Collection and/or Processing – S.O.D., K.K.; Analysis and/or Interpretation – S.O.D., G.B., K.K., F.E.; Literature Search – S.O.D., K.K.; Writing – S.O.D.; Critical Review – S.O.D.

**Declaration of Interests:** The authors have no conflicts of interest to declare.

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