



Was There an Increase in Cat-scratch Disease in Children During the COVID-19 Pandemic?

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Abstract

Background: Cat-scratch disease (CSD) is a zoonotic infection caused by *Bartonella henselae* that presents as usually local lymphadenopathy and is classified as localized, disseminate (hepatosplenic) or atypical syndrome according to clinical presentation. In this study, we present the cases of CSD that occurred during the COVID-19 pandemic.

Methods: This retrospective single-center study was performed between January 2014 and December 2022 and included 23 patients aged <18 years diagnosed with CSD.

Results: Among the 23 patients, 86% (n=20) were admitted between 2020 and 2022, had a mean age of 143.82±47.5 months, and 47.8% (n=11) were female. The most common symptoms were lymphadenopathy (87%), fever (8.7%), stomach aches (8.7%), and weight loss (13%). According to the clinical classification of CSD, 82% of the patients had localized CSD, 15% had disseminated CSD, and 0.04% had atypical (uveitis) CSD. Azithromycin therapy was preferred for localized CSD, combination therapy (rifampin or gentamycin) for disseminated CSD, and steroids for uveitis. Overall, the treatment outcomes were favorable, and a curing was achieved in the localized group. Notably, splenic lesions persisted for over 2 years in one patient.

Conclusions: Our study highlights the significant emergence of CSD during the COVID-19 pandemic and emphasizes its importance in children. The increase in cases may be attributed to alterations in pet acquisition practices and a surge in outdoor activities after lockdown restrictions were lifted.

Keywords: *Bartonella henselae*, cat-scratch disease, children, COVID-19, lymphadenopathy

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COVID-19 Pandemisi Sırasında Çocuklarda Kedi Tırmağı Hastalığı'nda Artış Oldu Mu?

Öz

Giriş: Kedi tırmağı hastalığı (KTH), *Bartonella henselae*'nin etken olduğu, genellikle lokal lenfadenopati şeklinde ortaya çıkan ve klinik tabloya göre lokalize, yaygın (hepatosplenik) veya atipik sendrom olarak sınıflandırılan zoonotik bir enfeksiyondur. Bu çalışmada, COVID-19 pandemisi sırasında ortaya çıkan CSD vakalarını sunulmuştur.

Yöntemler: Bu retrospektif tek merkezli çalışma Ocak 2014 ile Aralık 2022 tarihleri arasında yürütülmüş olup KTH tanısı alan 18 yaş altı 23 hastay dahil edildi.

Bulgular: Hastanın %86'sı (n=20) 2020 ile 2022 yılları arasında başvurmuş olup, ortalama yaşları 143,82±47,5 ay ve hastaların %47,8'i (n=11) kadındı. En sık görülen semptomlar lenfadenopati (%87), ateş (%8,7), mide ağrısı (%8,7) ve kilo kaybı (%13) idi. Klinik sınıflandırmasına göre hastaların %82'sinde lokalize, %15'inde yaygın ve %0,04'ünde atipik KTH vardı. Lokalize KTH için azitromisin tedavisi, yaygın KTH için kombinasyon tedavisi (rifampin veya gentamisin) ve üveit için steroidler tercih edildi. Genel olarak, tedavi sonuçları olumlu ve lokalize grupta kür sağlandığı görülse de bir hastada dalak lezyonları 2 yıldan uzun süre devam etti.

Sonuçlar: Çalışmamız, KTH'nın çocuklarda önemini ve COVID-19 pandemisi sırasındaki artışına dikkat çekmektedir. Bu durum evcil hayvan edinme davranışlarındaki değişikliklere ve karantina kısıtlamalarının kaldırılmasından sonra açık hava aktivitelerindeki artışa bağlanabilir.

Anahtar kelimeler: *Bartonella henselae*, kedi tırmağı hastalığı, çocuklar, COVID-19, lenfadenopati.

INTRODUCTION

Cat-scratch disease (CSD) is a zoonotic disease caused by *Bartonella henselae*. It usually develops as acute or subacute local lymphadenopathy, but can also manifest as atypical syndromes, including hepatosplenic CSD, fever of unknown origin (FUO), neurological manifestations, endocarditis, bacteremia, retinitis^{1,2}. Cats are the primary reservoir of *B. henselae*, with exposure to cats being identified in 90% of CSD cases¹. *B. henselae* is a gram-negative, aerobic, facultative intracellular bacillus, with approximately 50% of cats, particularly small cats, being asymptomatic carriers, which serve as transmitters through biting or scratching³. The incidence and seasonality of CSD were associated with cats' breeding period and an increase in flea breeding in the late summer months⁴. A case series including various countries reported the highest incidence in autumn and winter, with January being the peak month in the USA^{5,6}. Although CSD can occur at any age, the highest incidence is in the 5- age range, with 32.5% of all cases occurring in children aged < 14 years⁶. CSD case series have also been reported in with one study reporting immunoglobulin (Ig) M seropositivity in 9.9% of

suspected cases, suggesting that the actual number of cases is much greater in patients with increased contact with stray cats^{7,8}. During the pandemic, there was increased contact between children and both stray and domestic animals. A study conducted in Argentina reported a 98% increase in the frequency of CSD during the COVID-19 pandemic compared to the period before March 2020⁹. In this context, our study aimed to present the demographic, clinical, and laboratory parameters of patients diagnosed with CSD; outline our treatment approach; and assess the impact of the COVID-19 pandemic on the incidence of CSD.

METHODS

Population

This study included 23 patients aged <18 years diagnosed with CSD between January 2014 and December 2022. The study period (2014-2022) was chosen to encompass the years before and during the COVID-19 pandemic, allowing for a comparative analysis of incidence. A retrospective analysis was conducted using patients' medical records to evaluate their demographic, clinical, and microbiological characteristics.

Laboratory assessment

CSD was diagnosed based on serological tests or histopathological examinations, in addition to clinical presentations. According to serological tests, CSD was defined as follows:

- Positive serology with IgM titers against *B. Henselae* or positive serology with IgG titers against *B. Henselae* $\geq 1/256$
- An IgG titer of $<1:64$ suggested that the patient did not have CSD. If titers of $1/64$ or $1/128$ represented possible CSD, repeated testing was performed for 10–14 d. A titer of $\geq 1:256$ was considered indicative of CSD¹⁰.

Clinical definitions

Localized CSD was defined as the presence of regional lymphadenopathy, whereas disseminated CSD was characterized by hepatosplenic involvement with or without lymphadenopathy. Atypical CSD included neuroretinitis, fever of unknown origin, endocarditis, osteomyelitis, encephalopathy, and other atypical manifestations¹¹.

The study also ruled out other alternative diagnoses (viruses; Epstein-Barr virus, Cytomegalovirus, etc.; bacteria, such as gram-positive agents, especially those causing suppurative bacterial lymphadenopathy, such as *Mycobacterium tuberculosis*-granulomatosis infections) through clinical or laboratory evaluation.

Statistical Analysis

Statistical data were analyzed using IBM SPSS Statistics for Windows, version 21. The values for numerical variables are given as mean \pm standard deviation, depending on the normal distribution, while categorical variables were compared using the χ^2 or Fisher's exact test. Statistical significance was set at $p < 0.05$. This study was approved by the Istanbul University Clinical Research Ethics Committee (2023/220, 17.02.23-162871).

RESULTS

This study included 23 patients diagnosed with CSD. Except for three patients, most patients

($n=20$) were admitted between 2020 and 2022. The mean age of the patients was 11.98 ± 3.95 (3–17,5) years; 47.8% ($n=11$) were female and 52.2% ($n=12$) were male.

The most commonly reported symptoms at presentation were lymphadenopathy (87%, $n=20$) (Figure 1), fever (8.7%, $n=2$), stomachache (8.7%, $n=2$), weight loss (13%, $n=3$), rash (4.3%, $n=1$), and blurred vision (4.3%, $n=1$). Lymphadenopathy was observed in all patients; however, it was the primary presenting symptom in 87% of cases. The remaining three patients initially presented with fever, abdominal pain, or blurred vision. The duration of symptoms ranged 0–12 months, with an average of 2.95 ± 3.57 months. A history of intimate contact with a cat was reported in 69.6% ($n=16$) of patients (Table 1).

Systemic lymphadenopathy was observed in 21.7% ($n=5$) of patients, local lymphadenopathy in 73.9% ($n=17$), axillary lymphadenopathy in 21.7% ($n=5$), cervical lymphadenopathy in 13% ($n=3$), inguinal lymphadenopathy in 13% ($n=3$), submandibular lymphadenopathy in 13% ($n=3$), epitrochlear lymphadenopathy in 8.7% ($n=2$), and supraclavicular lymphadenopathy in 4.3% ($n=1$). Two patients with submandibular and inguinal lymphadenopathies are shown in Figure 1. Hepatosplenic involvement, including hepatomegaly, splenomegaly, calcific nodules, and microabscesses in the liver and spleen, was observed in three patients. One of the patients developed uveitis.



Figure 1. Two patients with a submandibular (left) and an inguinal lymphadenopathy (right)

The distribution of patients according to the clinical classification of CSD was as follows: localized, 82.6% ($n=19$); disseminated/atypical,

17.4% (n=4), which included three patients with hepatosplenic involvement and one with ocular involvement (uveitis).

Laboratory parameters

Positive serological results for IgM antibodies against *B. Henseleae* were found in 56.5% (n=13) of the patients, while 43.5% (n=10) were negative. At admission, positive serology for IgG antibodies (titer $\geq 1:256$) was found in 9 patients. A further 7 patients had low or negative titers at initial testing but showed a significant seroconversion (titer $\geq 1:256$) upon repeat testing 10-14 days later (Table 1).

Table I: Demographic, clinical characteristics, and laboratory parameters of patients with CSD

	Mean \pm SDS/n(%)	Min-max
Age (m)	143.82 \pm 47.5	37-211
<u>Sex (n/%)</u>		
Female	11 (47.8)	
Male	12 (52.2)	
Symptom duration (m)	2.95 \pm 3.57	0-12
<u>Contact (n/%)</u>		
Yes	16 (69.6)	
No	7 (30.4)	
Therapy time(d)	8.69 \pm 8.76	5-45
<u>Symptoms (n/%)</u>		
Papule	0 (0)	
Lenfadenomegaly	20(87)	
Loss of weight	3 (13)	
Fever	2 (8.7)	
Rash	1 (4.3)	
Blurred vision	1 (4.3)	
Stomachache	2 (8.7)	
<u>Laboratory</u>		
Hemoglobin (g/dL)	12.7 \pm 1.21	(10.7-15)
WBC (μ L)	7356 \pm 2185	(3000-12800)
Neutrophil (μ l)	4001 \pm 1796	(1570-7900)
Lymphocyte (μ l)	2586 \pm 902	(1200-5100)
Platelets (μ l)	319173 \pm 1139	(210000-737000)
CRP (mg/L)	7.87 \pm 15.79	(0.27-75)
ALT(U/L)	25.39 \pm 15.66	(8-79)
AST (U/L)	21.65 \pm 7.92	(8-43)
Bartonella IgM positivity	13 (56.5)	
Bartonella IgG positivity	16 (69.5)	

ALT; Alanine aminotransferase, AST: aspartate aminotransferase, CSD: Cat-scratch disease, CRP; C-reactive protein, d: day, m: month, SDS: standard deviation score, WBC: White Blood Cell

Radiological evaluations were performed using ultrasonography/computed tomography (USG/CT). One patient had a subcapsular hypointense lesion in the spleen, one had multiple nodular calcific lesions in the liver, and one had multiple nodular lesions in the liver and spleen (Figure 2).

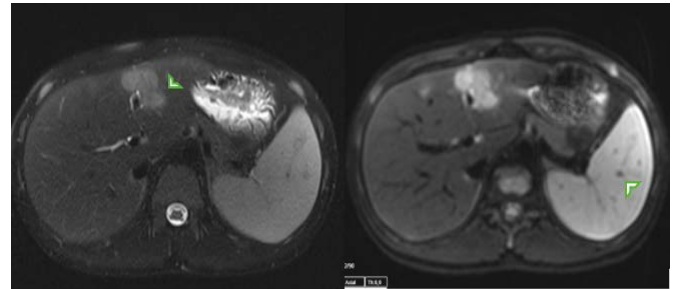


Figure 2. Patients with hepatosplenic CSD. A hyperintense nodular lesion measuring 64 x 46 x 73 mm in the liver (left); multiple hypointense lesions 6 mm, 10 mm, and 16 mm in size in the spleen (right).

Nine patients underwent lymph node biopsy due to suspected malignancy or other infectious causes, and histopathological examination revealed non-necrotizing/necrotizing granulomatous lymphadenitis. Additionally, a patient who underwent a liver biopsy was found to have chronic granulomatous infection and fibrosis. No coinfections or comorbidities were detected in any patient.

Comparison of the characteristics of the disseminated and localized groups

The mean duration of therapy in the disseminated group was 21.75 \pm 15.5 d, while that in the localized group was 5.94 \pm 2.83 d. The duration of therapy was significantly longer in the disseminated group than in the localized group ($p=0.002$). Lymphadenomegaly was significantly more common in patients with localized CSD ($p=0.002$). No significant differences were observed in other variables, including sex, contact, disease duration, or other laboratory parameters (Table 2).

Table II: Comparison of localized/disseminated and atypical CSD cases

	Disseminated and atypical CSD (n:4)		Localized CSD (n:19)		p
	Mean±SDS/ n(%)	Min-max	Mean±SDS/ n(%)	Min-max	
Age (m)	137.75 16.78	123-156	145.105 51.98	37-211	0.59
<u>Sex (n/%)</u>					
Female	1 (0.43)		10 (43)		0.59
Male	3 (13)		9 (39)		
Symptom duration(m)	0.62±0.75	(0-1.5)	3.44±3.75	(0.5-12)	0.257
<u>Contact</u>					
Yes	2 (8.6%)		14 (60%)		0.55
No	2 (8.6%)		5 (21.7)		
Therapy time(d)	21.75±15.5	(14-45)	5.94±2.83	5-14	0.002
<u>Symptoms</u>					
Papule	0 (0)		0 (0)		
Lenfadenomegaly	1 (4.3)		19 (82)		0.002
Loss of weight	1 (4.3)		2 (8.7)		0.45
Fever	1 (4.3)		1 (4.3)		0.32
Rash			1 (4.3)		NS
Blurred vision	1 (4.3)				0.174
Stomachache	2 (8.7)				0.24
<u>Laboratory</u>					
Hemoglobin (g/dL)	12.9±1.15	(11.7-14)	12.74±1.23	(10.7-15)	NS
WBC (µL)	7650±2146	(5200-9200)	7392±2274	(3000-12800)	NS
Neutrophil (µl)	4706±2027	(2900-6900)	3979±1800	(1570-7900)	NS
Lymphocyte (µl)	2396±949	(1400-3290)	2616±917	(1200-5100)	NS
Platelets (µl)	296666±43003	(254000-340000)	318315±124870	(210000-737000)	0.317
CRP (mg/L)	27.16±42	(2.5-75)	5.23±6.45	(0.27-21)	0.317
ALT(U/L)	32±9.53	(21-38)	24.94±16.5	(8-79)	NS
AST (U/L)	29.33±7.02	(22-36)	20.42±7.74	(8-43)	0.589
Bartonella IgG positivity	3 (75)		11 (57.8)		NS
	2(50)		12(63)		NS

ALT;alanin aminotransferase, AST;aspartate aminotransferase, CSD; Cat-scratch disease, CRP;c-reactive protein, d;day, m;month, SDS; standard deviation score, Not Significant; NS, WBC; White Blood Cell

In the disseminated/atypical group, the prevalence of Bartonella IgM and IgG positivity was 75% (n=3) and 50% (n=2), respectively, whereas in the localized group, it was 57.8% (n=11) and 63% (n=12), respectively.

Treatment and prognosis: Only 15 of the 23 patients (65.2%) received only azithromycin, with 8 patients having received a combination regimen of azithromycin (rifampin and gentamycin). The mean duration of therapy was 8.69±8.76 d (range 5–45 d). The patient with uveitis was administered doxycycline, rifampin,

and prednisolone. The patient with hepatosplenic involvement was treated with azithromycin, rifampin, and trimethoprim-sulfamethoxazole because of failure of therapy with a combination of azithromycin and gentamycin. Two patients required drainage due to lymph node abscesses.

Follow-up: In the localized CSD group, lymphadenomegaly regressed in all the patients after treatment. In the patient with microabscesses in the liver and spleen, the liver lesion regressed in the fourth month, whereas

the splenic lesion persisted into the second year, although its size decreased. The patient with only a hepatic abscess showed regression of the lesion size on USG at the first-year follow-up.

DISCUSSION

B. henselae is typically considered as a differential diagnosis when there is a history of contact with cats and other typical findings. The number of cases observed at our institution increased, especially during dates that coincide with the COVID-19 pandemic. We observed an average of 0.5 cases per year pre-pandemic (2014-2019) compared to 6.6 cases per year during the pandemic (2020-2022). The habit of acquiring pets during the lockdown period and preference for outdoor areas in the post-lockdown period increased contact with animals.

In a Spanish study, the incidence rate of CSD inpatients was 0.93 cases per million person-years, with an overall mortality rate of 1.3%¹². As reported in the USA (9/100 000), the highest overall average incidence rate was in patients aged 5–9 years old (3.0/1.000.000), and 42.6% of cases were in children (0–14 years) in Spain^{6,12}. The median age of the patients was 16 years in Turkey⁷. Although only pediatric patients were evaluated in our study, the mean age in our patient group (11,98±3.95 years) was greater than the age range of 5–9 years, which has been reported to have the highest incidence.

In various case series of pediatric patients with CSD, the most common presentation was solitary lymphadenopathy, although the rate varied. The most common clinical finding in Spain was the presence of isolated lymphadenopathies (84.7%),¹³ whereas the rate of localized disease among patients with CSD was reported 55.6% in Chile¹¹. This rate was higher (80%) in a study by Fusani et al.⁵. In the present study, the majority of the patients presented with regional lymph node

involvement and were diagnosed with localized CSD (82.6%). The head, neck, and axillary regions are the most commonly involved sites^{11,13}. Additionally, the axillary station was the most common site of involvement. However, lymph node involvement was observed in more than one region. Another common finding, painful erythematous papules and vesicles at the site of bacterial inoculation, was not observed in our patients¹⁴. A maculopapular rash, which is not a typical finding, was observed in only one patient.

Atypical/systemic CSD constitutes 1.5% of all CSD cases, with an overall incidence rate of 0.7 per 100,000 individuals in the USA. While most atypical cases were observed in individuals between the ages of 15 and 49 years, 36.2% of these cases were in children under 14 years of age¹⁵. There is no gold standard method for the diagnosis of systemic CSD, and the diagnosis can be made based on the presence of high clinical suspicion using various laboratory methods¹⁶. In a prevalence study, the seroprevalence of *B. henselae* in volunteer blood was 6% (48/800), and histories of tick bites and rabbit husbandry were risk factors¹⁷. The fact that this rate was found in healthy volunteers suggests that it may be more common than previously believed.

The most prevalent atypical presentations of systemic CSD were ocular (48.7%), hepatosplenic (24.6%), and neurological (13.8%), with hepatosplenic disease exhibiting a stronger correlation with younger age¹⁵. Patients usually present with prolonged fever, abdominal pain, weight loss, lymphadenomegaly, hepatosplenomegaly, and hematogenous or lymphatic spread, all of which are thought to be involved in the disease pathogenesis¹⁴. No atypical findings were observed in immunosuppressed patients. Hepatosplenic abscesses were successfully treated in immunocompromised children¹⁸. A case of hepatic abscess 1 month after azithromycin treatment for a typical diagnosis

of CSD has also been reported. Although fever was a common finding in a study evaluating patients with Bartonella-related systemic findings, it was present in only one of our patients with systemic findings¹⁹.

Imaging modalities are valuable tools for the diagnosis of medical conditions. In particular, abdominal imaging is effective in detecting microabscesses in the liver or spleen of >50% of patients with hepatosplenic CSD²⁰. In some cases, no lesions can be detected on USG, whereas abscesses can be seen on CT or vice versa²¹. Patients may present with granulomatous hepatitis with an increase in liver enzymes²² as well as abscesses in both the liver and spleen¹³. Although USG/MR is a very helpful diagnostic method, in some cases, the presence of “stellate microabscesses” can be demonstrated in tissues when liver/spleen/lymph node biopsy is performed for differential diagnosis¹. We performed a liver biopsy in only one patient.

The most common ocular manifestations of CSD are optic nerve lesions, uveitis, neuroretinitis, optic neuropathy, disc swelling, and retinal vessel occlusion. Ophthalmological involvement was reported in 4.4% of patients in a large CSD series in Israel (141/3222). In a national CSD surveillance study, decreased or blurred vision and neuroretinitis were reported in 94 (88%) and 14 (64%) patients with ocular diseases, respectively, during the follow-up period²³. Although neuroretinitis occurs in 1–2% of infected patients²⁴, CSD is the most common cause of neuroretinitis, and seropositivity is detected in two-thirds of cases²⁵. Although there is no definitive treatment guideline for CSD, a combination of doxycycline and rifampicin/azithromycin is recommended, especially for immunosuppressed patients²⁶. In addition, the addition of systemic steroids to treatment, rather than antimicrobial therapy alone, has a positive effect on visual outcomes²⁷.

Although treatment is not recommended for uncomplicated isolated lymphadenomegaly, antimicrobial treatment is recommended because of the risk of dissemination, with treatment decreasing the duration of symptoms. Oral azithromycin for 5 days was effective in reducing lymph node volume, as measured by three-dimensional USG in a previous prospective study²⁸. Although typical CSD lymphadenomegaly resolves within 2–6 months, we preferred azithromycin treatment for 5 d in patients with isolated lymphadenomegaly, and only two patients required lymph node drainage during follow-up.

No clear treatment guidelines are available for hepatosplenic CSD, and prolonged monotherapy or combination therapy is generally preferred²⁹. In hepatosplenic CSD, a combination of azithromycin and rifampicin is considered first-line treatment; however, Arisoy et al. showed that fever resolves more rapidly when rifampicin is administered along with TMP-SMX for 14 d^{20,21}. In one of our cases, we opted for a combination therapy comprising azithromycin, rifampin, and trimethoprim-sulfamethoxazole because the symptoms did not regress with a combination of azithromycin and gentamicin. Depending on the clinical findings and resolution of the lesions, the duration of treatment can be prolonged, but varies between 14 and 28 days on average²⁰. The resolution time of hepatic/splenic lesions varies, and cases of resolution on the 45th day or fifth month of the disease have been reported^{19,30}. In the second year follow-up of one of our patients, although the lesion in the spleen was reduced in size, it was not completely resolved, and follow-up with intermittent USG was continued.

This study has several limitations. Firstly, it was conducted at a single center, which may restrict the generalizability of the findings to a more extensive population. Secondly, the evaluation

of systemic CSD was limited by the relatively small number of patients available for detailed analysis. Despite these limitations, this study provides important perspectives on the pediatric presentation of CSD and highlights the need for future multicenter investigations to validate and extend these findings to diverse populations.

CONCLUSION

In pediatric patients, CSD should be considered as a potential differential diagnosis for patients presenting with localized lymphadenomegaly, fever of unknown origin, ocular or neurological involvement, particularly in regions where the incidence of cat contact is increased. This study highlights the diverse clinical presentations of CSD, which have increased during the COVID-19 pandemic. The increase in cases may be attributed to alterations in pet acquisition practices and a surge in outdoor activities after lockdown restrictions were lifted. Further research is warranted to explore the nuances of CSD, its evolving epidemiology, and optimal management strategies for atypical manifestations.

Ethics Committee Approval: This study was approved by the Istanbul University Clinical Research Ethics Committee (2023/220, 17.02.23-162871).

Conflict of Interest: The authors declared no conflicts of interest.

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REFERENCES

1. Leigh, M., Howard & Kathryn, M. Edwards. Bartonella infections. In: Feigin, R.D., Cherry, J.D., eds. Textbook of Pediatric Infectious Diseases. 8th ed. Philadelphia: Elsevier; 2019:1240-45.
2. Bass JW, Vincent JM, Person DA. The expanding spectrum of Bartonella infections: II. Cat-scratch disease. *Pediatr Infect Dis J*. 1997 Feb;16(2):163-79.
3. Klotz SA, Ianas V, Elliott SP. Cat-scratch Disease. *Am Fam Physician*. 2011;83(2):152-155. PMID: 21243990.
4. Zangwill KM, Hamilton DH, Perkins BA, et al. Cat scratch disease in Connecticut. Epidemiology, risk factors, and evaluation of a new diagnostic test. *N Engl J Med*. 1993 Jul 1;329(1):8-13.
5. Fusani L, Venturini E, Chiappini E, Galli L. Cat scratch disease in childhood: a 10-year experience of an Italian tertiary care children's hospital. *World J Pediatr*. 2023 Feb;19(2):189-193.
6. Nelson CA, Saha S, Mead PS. Cat-Scratch Disease in the United States, 2005-2013. *Emerg Infect Dis*. 2016 Oct;22(10):1741-6.
7. Uluğ M. Evaluation of Cat Scratch Disease Cases Reported from Turkey between 1996 and 2013 and Review of the Literature. *Cent Eur J Public Health*. 2015 Jun;23(2):170-5.
8. Arıcı N, Aksaray S, Ankaralı H. Bartonella henselae IgM seropositivity in both adult and pediatric patients with diverse clinical conditions in Turkey. *Acta Microbiol Immunol Hung*. 2021 Mar 2.
9. Praino ML, Infectologist P, Julia G, et al. 1357. Collateral consequence of COVID pandemic: Increased incidence of cat scratch disease. *Open Forum Infect Dis*. 2022 Dec 15;9(Suppl 2):ofac492.1186.
10. Spach DH, Kaplan SL. Microbiology, epidemiology, clinical manifestations, and diagnosis of cat scratch disease. Uptodate, Ed. Edwards MS, Stout J [updated 2024 Nov]. Available at: (Accessed on Nov 24).
11. Sandoval AC, Reyes FT, Prado MA, Peña AL, Viviani TN. Cat-scratch Disease in the Pediatric Population: 6 Years of Evaluation and Follow-up in a Public Hospital in Chile. *Pediatr Infect Dis J*. 2020 Oct;39(10):889-893.
12. Rodríguez Alonso B, Alonso-Sardón M, Rodrigues Almeida HM, et al. Epidemiological of cat scratch disease among inpatients in the Spanish health system (1997-2015). *Eur J Clin Microbiol Infect Dis*. 2021 Apr;40(4):849-857.
13. Deregibus MI, Bagnara EI, Buchovsky A. Cat-scratch disease: Experience in a tertiary care

- children's hospital. Arch Argent Pediatr. 2023 Feb 1;121(1):e202202592.
14. Florin TA, Zaoutis TE, Zaoutis LB. Beyond cat scratch disease: widening spectrum of Bartonella henselae infection. Pediatrics. 2008 May;121(5):e1413-25.
15. Nawrocki CC, Max RJ, Marzec NS, Nelson CA. Atypical Manifestations of Cat-Scratch Disease, United States, 2005-2014. Emerg Infect Dis. 2020 Jul;26(7):1438-1446.
16. Margileth AM. Recent Advances in Diagnosis and Treatment of Cat Scratch Disease. Curr Infect Dis Rep. 2000 Apr;2(2):141-146.
17. Yilmaz C, Ergin C, Kaleli I. Pamukkale Üniversitesi Kan Merkezine başvuran donörlerde Bartonella henselae seroprevalansinin araştırılması ve risk faktörlerinin irdelenmesi [Investigation of Bartonella henselae seroprevalence and related risk factors in blood donors admitted to Pamukkale University Blood Center]. Mikrobiyol Bul. 2009 Jul;43(3):391-401. Turkish. PMID: 19795614.
18. Del Pozo AA, Angulo-Cruzado M, Amenero-Vega R, et al. Hepatosplenic abscesses in an immunocompetent child with cat-scratch disease from Peru. Ann Clin Microbiol Antimicrob. 2019 Jul 15;18(1):23.
19. Choi JH, Thomas P, Gray JM, et al. Hepatic Lesions in an Immunocompetent Child Previously Treated for Cat Scratch Disease With Azithromycin. Clin Pediatr (Phila). 2023 Mar;62(3):191-194.
20. Johnson SC, Kosut J, Ching N. Disseminated Cat Scratch Disease in Pediatric Patients in Hawai'i. Hawaii J Health Soc Welf. 2020;79(5 Suppl 1):64-70. PMID: 32490388
21. Arisoy ES, Correa AG, Wagner ML, Kaplan SL. Hepatosplenic cat-scratch disease in children: selected clinical features and treatment. Clin Infect Dis. 1999 Apr;28(4):778-84.
22. VanderHeyden TR, Yong SL, Breitschwerdt EB, et al. Granulomatous hepatitis due to Bartonella henselae infection in an immunocompetent patient. BMC Infect Dis. 2012 Jan 23;12:17.
23. Habot-Wilner Z, Trivizki O, Goldstein M, et al. Cat-scratch disease: ocular manifestations and treatment outcome. Acta Ophthalmol. 2018 Jun;96(4):e524-e532.
24. Carithers HA. Cat-scratch disease. An overview based on a study of 1,200 patients. Am J Dis Child. 1985 Nov;139(11):1124-33.
25. Weiss AH, Beck RW. Neuroretinitis in childhood. J Pediatr Ophthalmol Strabismus. 1989 Jul-Aug;26(4):198-203.
26. Acar A, Çakar Özdal P, Başarır B, et al. A Case Series of Cat-Scratch Disease with Ocular Manifestations: Clinical Findings and Treatment Approach. Turk J Ophthalmol. 2023 Aug 19;53(4):226-233.
27. Kalogeropoulos D, Asproudis I, Stefaniotou M, et al. Bartonella henselae- and quintana-associated uveitis: a case series and approach of a potentially severe disease with a broad spectrum of ocular manifestations. Int Ophthalmol. 2019 Nov;39(11):2505-2515.
28. Bass JW, Freitas BC, Freitas AD, et al. Prospective randomized double blind placebo-controlled evaluation of azithromycin for treatment of cat-scratch disease. Pediatr Infect Dis J. 1998 Jun;17(6):447-52.
29. Rolain JM, Brouqui P, Koehler JE, et al. Recommendations for treatment of human infections caused by Bartonella species. Antimicrob Agents Chemother. 2004 Jun;48(6):1921-33.
30. Liao HM, Huang FY, Chi H, Wang NL, Chen BF. Systemic cat scratch disease. J Formos Med Assoc. 2006 Aug;105(8):674-9.