Kozu et al. Advances in Rheumatology (2018) 58:28
https://doi.org/10.1186/s42358-018-0024-x

Advances in Rheumatology

RESEARCH Open Access

Juvenile dermatomyositis: is periodontal disease associated with dyslipidemia?

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Abstract

Background: Association between periodontal disease and dyslipidemia was recently reported in healthy adults. However, a systematic evaluation of concomitant periodontal diseases and lipid profile was not carried out in juvenile dermatomyositis (JDM).

A cross-section study was performed in 25 JDM patients and 25 healthy controls, assessing demographic data, periodontal evaluation, fasting lipoproteins and anti-lipoprotein lipase antibodies. Disease parameters, laboratorial tests and treatment were also evaluated in JDM patients.

Results: The mean current age was similar in patients and controls (11.5 ± 3.75 vs. 11.2 ± 2.58 years,$p = 0.703$). Regarding lipid profile, the median triglycerides [80(31–340) vs. 61(19–182)mg/dL,$p = 0.011$] and VLDL[16(6–68) vs. 13(4–36)mg/dL,$p = 0.020$] were significantly higher in JDM patients versus controls. Gingival vasculopathy pattern was significantly higher in the former group (60% vs. 0%,$p = 0.0001$), as well as the median of gingival bleeding index (GBI) [24.1(4.2–69.4) vs. 11.1(0–66.6)%,$p = 0.001$] and probing pocket depth (PPD) [1.7(0.6–2.4) vs.1.4(0–2.12) mm,$p = 0.006$]. Comparison between JDM patients with and without dyslipidemia revealed that the median of dental plaque index (PI) [100(26.7–100) vs. 59(25–100)%,$p = 0.022$], PPD[1.9(0.6–2.4) vs. 1.4(1.2–1.8)mm,$p = 0.024$] and clinical attachment level (CAL) [1.31(0.7–1.7) vs. 0.8(0.6–1.7)mm,$p = 0.005$] were significantly higher in patients with dyslipidemia. Further analysis between JDM patients with and without gingivitis revealed that the median of current age [12.4 (8.3–18.4) vs. 9.2 (5.5–17.5) years, $p = 0.034$] and disease duration [7.09 ± 3.07 vs. 3.95 ± 2.1 years, $p = 0.008$] were significantly higher in the former group.

Conclusion: Our study showed that gingival inflammation seems to be related to dyslipidemia in JDM patients, suggesting underlying mechanisms for both complications.

Keywords: Juvenile dermatomyositis, Dyslipidemia, Periodontal disease, Gingivitis

Background

Juvenile dermatomyositis (JDM) is a multisystemic disease of unknown etiology characterized by chronic inflammation of striated muscles and skin [1, 2]. The survival rate, outcome and health related quality of life of JDM populations has been improving in the last years and some aspects, such as periodontal diseases [3, 4] and dyslipidemia [5–9] are particularly relevant for these patients.

Periodontal disease comprises gingivitis and periodontitis. It is defined as an immune inflammatory periodontal disorder characterized by chronic localized infections associated with inflammation [3]. Periodontitis is a progressive phenomenon, beginning with gingivitis, followed by the destruction of the periodontal ligaments and bone reabsorption and ending in dental attachment loss [4]. Gengivitis and/or periodontitis have been seldom studied in pediatric autoimmune diseases, like juvenile idiopathic arthritis and juvenile systemic lupus erythematosus [10–15] Recently the association between periodontal disease and dyslipidemia was reported in healthy adults [16].

Data of gingivitis and periodontal diseases are scarce in JDM patients. Of note, the association between gingival vasculopathy pattern, characterized by gingival erythema, capillary dilation and bush-loop formation and disease

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activity was previously suggested [4, 17]. Moreover, dyslipidemia has been rarely reported in JDM patients [5–9], and was observed in 36% of JDM patients, characterized by low high-density lipoprotein (HDL) and high triglycerides (TG) levels [9].

However, to our knowledge, a systematic evaluation of concomitant periodontal diseases and lipid profile assessment was not carried out in JDM population.

Therefore, the aims of this study were to assess periodontal involvement and lipid profile in JDM patients and healthy controls, and to evaluate the possible associations between periodontal disease and dyslipidemia in this chronic inflammatory myopathy.

Materials and methods

JDM patients and controls

A cross-section study was performed from January 2009 to January 2011, and involved 52 JDM patients who were followed at the Pediatric Rheumatology Unit of our University Hospital. All of them fulfilled Bohan and Peter criteria for JDM diagnosis [18]. Exclusion criteria were: diabetes mellitus (fasting glycemia > 126 mg/dL); renal insufficiency (creatinine clearance < 70 ml/min/1.73m^2); proteinuria > 0.3 g/24 h; liver and thyroid dysfunction; neoplasia; infection in the last 15 days; hospitalization in the last month; previous/current smoking and alcohol use; current pregnancy; hormonal therapy; presence of orthodontic appliances and the use of lipid-lowering, anticonvulsant and antihypertensive drugs (thiazide, diuretics or betablockers). Twenty-seven JDM patients were excluded due to refusal to participate in this study ($n = 20$), presence of orthodontic appliances ($n = 4$) and incomplete evaluation ($n = 3$). Therefore, the final group was comprised by 25 JDM patients.

The healthy control group included 25 children and adolescents recruited from the families of JDM patients (either cousins or siblings) in order to minimize differences in some interfering aspects such as nutrition, constitutional and genetic factors. This study was approved by the Local Ethics Committee of our University Hospital and an age-appropriate written informed consent was obtained from all participants and their legal guardians.

Methods

Demographic, anthropometric data and body composition

Current age and gender were recorded for all subjects. For JDM patients, age at disease onset, and disease duration were also studied. Anthropometric data of patients and controls included blood pressure, weight in kilograms, height in meters, and body mass index (BMI) defined by the formula weight/height^2 (kg/m^2). For measurement of body composition, fat and lean mass and fat percentage were evaluated by dual-energy x-ray absorptiometry (DXA) using the densitometer Hologic QDR 4500 with pediatric software (Discovery model; Hologic Inc. Bedford, MA, USA). A questionnaire of life style/life habits was applied to all JDM patients and healthy controls to assess information regarding breastfeeding, weekly physical activity in hours, and familial history of coronary disease and dyslipidemia [9].

Clinical evaluation and treatment

All JDM patients were examined by the same pediatric rheumatologist to assess the following disease parameters scores: Disease Activity Score (DAS) [19], Childhood Myositis Assessment Scale (CMAS) [20], Manual Muscle Testing (MMT) [20], Myositis Disease Activity Assessment Analogue Scale (MYOACT) [21] and Myositis Intention to Treat Activity Index (MYTAX) [21]. Functional ability score was assessed according to the validated Brazilian version of Childhood Health Assessment Questionnaire (CHAQ) [22].

Current use and cumulative dose of prednisone, hydroxychloroquine, immunosuppressive drugs (methotrexate, cyclosporine, azathioprine and cyclophosphamide) were also determined.

Periodontal assessment

Periodontal assessment was performed in all subjects by three standardized epidemiological parameters: gingival index (GI), dental plaque index (PI) [23] and gingival bleeding index (GBI) [24]. Clinical dental attachment was evaluated by three other indices: probing pocket depth (PPD), cementoenamel junction (CEJ) and clinical attachment level (CAL), at six sites per tooth [25]. The number of decayed, missing and filled teeth (DMF-T) was also counted [26]. PI was used to evaluate the level of oral hygiene, which was calculated according to the number of dental surfaces stained by a dental plaque disclosing agent, multiplied by 100 and divided by the total number of surfaces [23]. GBI was used to evaluate gingival inflammation, and was expressed as the number of bleeding surfaces after probing with a periodontal probe, which was then multiplied by 100 and divided by the total number of surfaces [24]. PPD was determined as the distance from the bottom of the pocket to the gingival margin (normal range < 3 mm). CEJ was measured as the distance from the gingival margin to the cementoenamel junction, identifying hyperplasia or recession. CAL was calculated as the sum of PPD and CEJ (normal range < 3 mm) [25]. Gingivitis was defined as inflammation of the gingiva in the absence of clinical attachment loss [27] with GBI > 25% [28]. Gingival vasculopathy pattern was defined according to the presence of concomitant gingival erythema, capillary dilation and bush-loop formation pattern, as previously described [3].

Laboratory analysis

Biochemical analyses were performed for JDM patients and controls on serum samples obtained after 12-h over-night fasting at the study entry.

Lipid profile

Total cholesterol (TC) and triglycerides (TG) were measured enzymatically on a Technicom RA 1000 System analyser (Boehringer Mannheim, Argentina and Merck, Alemanha) [29, 30]. High-density lipoprotein (HDL) cholesterol was obtained by colorimetric method (Roche Diagnostics) after precipitation of very low-density lipoprotein (VLDL) cholesterol and low-density lipoprotein (LDL) cholesterol by phosphotungstic acid and magnesium chloride [31]. Levels of VLDL were estimated using the formula of TG levels divided by 5 (TG/5), since all samples had a TG level < 400 mg/dL [32]; and LDL cholesterol levels were estimated using the following equation: TC - (HDL + VLDL) [32]. Normal values were defined according to national norms for metabolic data for children and adolescents as follow: TC ≤ 170 mg/dL, HDL ≥ 45 mg/dL, LDL ≤ 130 mg/dL, and TG ≤ 130 mg/

Table 1 Demographic and anthropometric data, body composition, laboratorial findings and periodontal evaluation in juvenile dermatomyositis (JDM) patients and healthy controls

Variables	JDM patients ($n = 25$)	Controls ($n = 25$)	p
Demographic data			
Current age, years	11.5 ± 3.75	11.2 ± 2.58	0.703
Female gender	14 (56)	13 (52)	0.500
Anthropometric data			
BMI, kg/m^2	19.1 (12.7–30)	17.1 (14.4–27.5)	0.954
Systolic blood pressure, mmHg	90 (80–118)	90 (80–110)	0.099
Dyastolic blood pressure, mmHg	60 (50–85)	60 (45–70)	0.211
Body composition			
Fat percentage	27.5 (10.9–45.4)	22.5 (10.2–42.3)	0.230
Fat mass, kg	10.7 (2.9–27.1)	6.7 (2.8–30.5)	0.274
Lean mass, kg	24.8 ± 6.4	25.4 ± 7.6	0.599
Lipid profile			
Dyslipidemia	17 (68)	10 (40)	0.087
Total cholesterol	151 (102–227)	151 (121–207)	0.941
≥ 170 mg/dL	8 (32)	4 (16)	0.320
HDL, mg/dL	44 (0–72)	50 (30–65)	0.117
≤ 45 mg/dL	10 (40)	7 (28)	0.550
LDL, mg/dL	87 (56–148)	91 (54–140)	0.675
≥ 130 mg/dL	1(4)	2(8)	1.000
VLDL, mg/dL	16 (6–68)	13 (4–36)	0.020
Triglycerides, mg/dL	80 (31–340)	61 (19–182)	0.011
≥ 130 mg/dL	5 (20)	1 (4)	0.189
Anti-LPL antibody	1 (4)	0	1.000
Periodontal assessment			
Gingival vasculopathy pattern	15 (60)	0 (0)	0.0001
DMF-T	2 (0–5)	2 (0–3)	0.862
PI, %	90.6 (25–100)	71.5 (19.8–100)	0.051
GBI, %	24.1 (4.2–69.4)	11.1 (0–66.6)	0.001
PPD, mm	1.7 (0.6–2.4)	1.4 (0–2.12)	0.006
CEJ, mm	−0.1 (−0.8–0)	−0.1 (−0.9–0)	0.570
CAL, mm	1.25 (0.7–1.7)	1 (0.6–1.7)	0.071

Values expressed in mean ± SD, median (range) and *n* (%); *BMI* (body mass index), *HDL* (high density lipoprotein), *LDL* (low density lipoprotein), *VLDL* (very low density), *anti-LPL* (anti-lipoprotein lipase antibody), *DMF-T* (decayed, missing and filled tooth index), *PI* (plaque index), *GBI* (gingival bleeding index), *PPD* (probing pocket depth), *CEJ* (cementoenamel juntion), *CAL* (clinical attachment level)

dL [33]. Dyslipidemia was defined when subjects presented lipid abnormalities in at least one of these lipid parameters [33].

Anti-lipoprotein lipase (LPL) antibodies

Anti-LPL IgG isotype antibodies were measured by double-enzyme-linked immunosorbent assay (ELISA). Costar polystyrene plates were coated overnight with commercially available LPL from bovine milk (5 μg/ml; Sigma, St Louis, MO) and then blocked with 15% adult bovine serum in Tris buffered saline (ABS-T) for one hour at room temperature. The test was performed with serum samples diluted at 1:100 in ABS-T incubated for one hour at room temperature. Anti-LPL IgG isotype antibodies were determined by alkaline phosphatase conjugated goat anti-human IgG (Sigma). The reaction was developed by means of p-nitrophenylphosphate and optical density (OD) was read at 405 nm with a labsystems Multiskan MS (Labsystems, Helsinki, Finland). Positive results were defined as OD values ≥3 standard deviation (SD) above the mean OD values of the 25 healthy control serum samples (cut-off value 0.36). To ensure consistency between assays, serial dilutions of known positive serum samples were included in each study [34].

Inflammatory profile and muscle enzymes

The following tests were performed in JDM patients only. Erythrocyte sedimentation rate (ESR) was evaluated using the Westergren method and C-reactive protein (CRP) by nephelometry. Skeletal muscle enzymes included creatinine kinase (CK) and aldolase measured by kinetic automated method, and lactate dehydrogenase (LDH), aspartate aminotransferase (AST) and alanine aminotransferase (ALT) by kinetic method.

Statistical analysis

Data were presented in median (range) or mean (± SD) for continuous variables according to abnormal or normal distribution, respectively. Data were presented in number (percentage) for categorical variables. For continuous variables data were compared using Mann-Whitney test or t test to evaluate differences between JDM patients and controls, JDM patients with and without gingivitis, and also JDM patients with and without dyslipidemia according to demographic, anthropometric, laboratorial, treatments and dental parameters. For categorical variables, differences were assessed by Fisher exact test. *P*-values < 0.05 were considered as significant.

Results

JDM patients vs. healthy controls

Demographic, anthropometric data, body composition, laboratorial exams and periodontal evaluation in JDM patients and healthy controls are shown in Table 1. The mean of current age was similar in JDM patients and healthy controls (11.5 ± 3.75 vs. 11.2 ± 2.58 years, $p = 0.703$). Although trend of a higher frequency of dyslipidemia was observed in JDM patients compared to controls [68% vs. 40%, $p = 0.087$] it was not statistically significant. The median TG [80 (31–340) vs. 61 (19–182) mg/dL, $p = 0.011$] and VLDL [16 (6–68) vs. 13 (4–36) mg/dL, $p = 0.020$] were significantly higher in JDM patients versus controls. The frequency of gingival vasculopathy pattern was significantly higher in the former group (60% vs. 0%, $p = 0.0001$), as well as

Table 2 Demographic data and periodontal assessment in juvenile dermatomyositis (JDM) patients with and without dyslipidemia

Variables	JDM with dyslipidemia (*n* = 17)	JDM without dyslipidemia (*n* = 8)	*p*
Demographic data			
Current age, years	12.2 (5.5–18.4)	8.5 (7.2–17.8)	0.103
Age at disease presentation, years	6.56 ± 2.73	4.83 ± 2.46	0.159
Disease duration, years	5.49 ± 3.45	5.2 ± 1.55	0.840
Female gender	9 (52)	5 (62)	1.000
Periodontal assessment			
Gingival vasculopathy pattern	11 (64)	4 (50)	0.667
Gingivitis (GBI > 25%)	10 (59)	2 (25)	0.202
DMF-T	2 (0–12)	1 (0–6)	0.102
PI, %	100 (26.7–100)	59 (25–100)	0.022
GBI, %	29.7 (4.2–69.4)	19.6 (13–50)	0.923
PPD, mm	1.9 (0.6–2.4)	1.4 (1.2–1.8)	0.024
CEJ, mm	−0.12 (−0.8–0)	−0.1 (−0.5–0.04)	0.923
CAL, mm	1.31 (0.7–1.7)	0.8 (0.6–1.7)	0.005

Values expressed in mean ± SD, median (range) and *n* (%); *GBI* (gingival bleeding index), *DMF-T* (decayed, missing, filled teeth index), *PI* (plaque index), *PPD* (probing pocket depth), *CEJ* (cementoenamel juntion), *CAL* (clinical attachment level)

Table 3 Demographic and anthropometric data, body composition, laboratorial findings, juvenile dermatomyositis (JDM) scores and treatment in JDM patients with and without gingivitis

Variables	*JDM with gingivitis (GBI > 25%, n = 12)*	*JDM without gingivitis (GBI ≤ 25%, n = 12)*	*p*
Demographic data			
Current age, years	12.4 (8.3–18.4)	9.2 (5.5–17.5)	0.034
Age at disease presentation, years	4.8 (3.7–7.4)	5.2 (4.1–9.5)	0.580
Disease duration, years	7.09 ± 3.07	3.95 ± 2.1	0.008
Female gender	9 (75)	5 (41)	0.182
Anthropometric data			
BMI, kg/m^2	19.6 (14.5–30)	19.2 (15.3–25)	0.954
Body composition			
Fat percentual	28.2 (11.1–45.4)	26 (10.9–42)	0.789
Fat mass, kg	10.8 (5.0–27.1)	8.2 (2.9–20.2)	1.000
Lean mass, kg	26.4 (15.4–43.8)	27.2 (20–44.5)	0.423
Lipid profile			
Total cholesterol	151 (115–206)	151 (102–227)	0.913
≥ 170 mg/dL	5 (41)	3 (25)	0.666
HDL, mg/dL	43 (0–65)	49 (17–72)	0.328
≤ 45 mg/dL	9 (75)	4 (33)	0.099
LDL, mg/dL	92 (76–148)	77.5 (56–129)	0.265
≥ 130 mg/dL	1 (9)	0	1.000
VLDL, mg/dL	16 (9–42)	14.5 (9–68)	0.957
Triglycerides, mg/dL	82 (31–168)	72.5 (46–340)	0.935
≥130 mg/dL	1 (8)	3 (33)	0.316
Anti-LPL antibody	0	1 (8)	1.000
Muscle enzymes			
AST, U/L	28 (13–122)	29.5 (15–82)	0.703
ALT, U/L	36 (22–123)	34 (29–79)	0.744
CK, U/L	84 (33–478)	125 (62–179)	0.399
LDH, U/L	184 (107–1234)	216.5 (153–562)	0.355
Aldolase, U/L	5.7 (3.4–10.8)	6.35 (4.6–14.6)	0.231
Inflammatory profile			
ESR, $mm/1^{ST}hour$	20 (2–40)	19 (7–54)	0.624
CRP, mg/dL	0.59 (0.16–5.5)	1.61 (0.15–26)	0.242
JDM scores			
CMAS, 0–52	52 (10–52)	52 (17–52)	0.848
MMT, 0–80	80 (42–80)	80 (38–80)	0.742
DAS, 0–18	4 (0–12)	3 (0–18)	0.807
Cutaneous DAS, 0–9	1 (0–8)	1 (0–9)	0.663
Muscle DAS	2 (0–9)	2 (0–9)	0.853
MYOACT, 0–1	0.05 (0–0.16)	0.02 (0–0.3)	0.724
MITAX, 0–1	0.01 (0–0.23)	0 (0–0.28)	0.462
CHAQ	0 (0–1.75)	0.625 (0–2.5)	0.164
Treatment			
Prednisone			
Current use	1 (8)	8 (66)	0.303

Table 3 Demographic and anthropometric data, body composition, laboratorial findings, juvenile dermatomyositis (JDM) scores and treatment in JDM patients with and without gingivitis *(Continued)*

Variables	*JDM with gingivitis (GBI > 25%, n = 12)*	*JDM without gingivitis (GBI ≤ 25%, n = 12)*	*p*
Cumulative dose, g	13.6 (4.9–51.5)	15.1 (3.9–31.2)	0.531
Methotrexate			
Current use	3 (25)	8 (66)	0.277
Cumulative dose, g	2.2 (0.3–16.9)	1.9 (0.37–4.98)	0.79
Cyclosporine			
Current use	0	3 (25)	0.230
Cumulative dose, g	0 (0–3.6)	0	0.805

Values expressed in mean ± SD, median (range) and *n* (%), *GBI* (gingival bleeding index), *BMI* (body mass index), *HDL* (high density lipoprotein), *LDL* (low density lipoprotein), *VLDL* (very low density), *anti-LPL* (anti-lipoprotein lipase antibody), *AST* (aspartate aminotransferase), *ALT* (alanine aminotransferase), *CK* (creatine kinase), *LDH* (lactate dehydrogenase), *ESR* (erythrocyte sedimentation rate), *CRP* (C-reactive protein), *CMAS* (Childhood Myositis Assessment Scale), *MMT* (Manual Muscle Testing), *DAS* (Disease Activity Score), *MYOACT* (Myositis Disease Activity Assessment Analogue Scale), *MYTAX* (Myositis Intention To Treat Activity Index), *CHAQ* (childhood assessment questionnaire)

the median of GBI [24.1 (4.2–69.4) vs. 11.1 (0–66.6)%, $p = 0.001$] and PPD [1.7 (0.6–2.4) vs. 1.4 (0–2.12) mm, $p = 0.006$] (Table 1).

No differences were observed in breastfeeding duration [4 (0–7) vs. 3 (0–12) months, $p = 0.548$], weekly physical activity [4 (0–14) vs. 5 (5–30), hours, $p = 0.182$] and familial history of coronary disease (32% vs. 52%, $p = 0.251$) between JDM patients and healthy controls.

Periodontal assessment and dyslipidemia in JDM patients

Further analysis between JDM patients with and without dyslipidemia revealed that the median of PI [100 (26.7–100) vs. 59 (25–100)%, $p = 0.022$], PPD [1.9 (0.6–2.4) vs. 1.4 (1.2–1.8) mm, $p = 0.024$] and CAL [1.31 (0.7–1.7) vs. 0.8 (0.6–1.7) mm, $p = 0.005$] were significantly higher in JDM patients with dyslipidemia compared to those without this complication (Table 2).

Gingivitis (GBI > 25%) was observed in 12/24 (50%) of JDM patients and 9/12 (75%) had concomitantly gingivitis with gingival vasculopathy pattern that extends over the upper and/or lower teeth. Further analysis between JDM patients with and without gingivitis revealed that the median of current age [12.4 (8.3–18.4) vs. 9.2 (5.5–17.5) years, $p = 0.034$] and disease duration [7.09 ± 3.07 vs. 3.95 ± 2.1 years, $p = 0.008$] were significantly higher in the former group. No differences were observed in BMI, body composition, anti-LPL antibodies, muscle enzymes, inflammatory parameters, JDM scores and treatments in both groups (Table 3). None of the patients were classified as presenting lipodystrophy (data not shown).

Discussion

According to our study, gingival inflammation seems to be related to dyslipidemia in JDM patients, suggesting common underlying mechanisms for both complications.

The great advantage of the study was to assess a concomitant evaluation of periodontal and lipid parameters in JDM patients and healthy controls. Tobacco use, alcohol intake, diabetes mellitus and some medications may be related to periodontal disorders and therefore they were considered as exclusion criteria [13]. Moreover, the control group included only JDM siblings or relatives, to minimize risk factors associated with periodontal diseases and dyslipidemia, particularly tooth brushing habits, nutrition and genetic factors. However, the major limitations of the present study were a relative small sample, due to the restricted inclusion and exclusion criteria, and a cross-sectional study design.

We confirmed previous evidences of altered lipid profile in JDM populations [5–9]. Dyslipidemia occurred in approximately 70% of our JDM patients, with high levels of VLDL and triglycerides. The presence of anti-LPL antibodies, a specific autoantibody associated with hypertriglyceridemia in systemic lupus erythematosus patients [34], was not a possible explanation for the lipoprotein abnormalities seen in our JDM patients.

The gingival inflammation, that used to be considered a process restricted to oral cavity, can now be considered as responsible for systemic inflammation and associated to disease activity in children [4] and adult patients [35]. It is believed that in predisposed individuals, a metastatic inflammation can occur, that is, a systemic inflammatory reaction to the presence of microorganisms (aerobic and anaerobic bacteria) in the chronically inflamed gingiva and periodontal ligaments. Thus, in the presence of gingivitis or periodontitis, inflammatory cytokines such as IL1 and IL6 are produced and exert local and systemic action, leading to an increase in circulating immune complexes. Therefore, periodontal abnormalities could act as a trigger for local and systemic inflammatory process, which in turn predisposes to metabolic changes, explaining the association between the expressive plaque indexes found in our patients with JDM with dyslipidemia [16, 36].

In addition, three-quarter of our JDM patients with gingivitis (gingival bleeding index > 25%) had a concomitant

clinical gingival vasculopathy pattern that extends over the upper and/or lower teeth, indicating a diffuse oral involvement. Of note, gingival alterations were not associated with treatment particularly with glucocorticoid and cyclosporine. This latter immunosuppressive drug is a well-known cause of gingival enlargement [37]. Moreover, gingival alterations could not be attributed to disease activity, since it was similar in JDM patients with and without gingivitis, with both groups presenting inactive disease or mild to moderate disease activity.

It was observed, however, that the group of patients with JDM and gingival alterations was composed by older individuals with longer disease duration. This fact suggests that the persistence of a chronic inflammatory process related to JDM disease could be responsible for the greater periodontal involvement and reinforces that periodontitis is a progressive phenomenon, beginning with gingivitis, followed by the destruction of the periodontal ligaments and bone reabsorption and ending in dental attachment loss [4]. In fact, although initial aspects of periodontitis, such as increased PCS and PCI, were associated with patients with JDM and dyslipidemia, none of the patients studied was classified as having periodontitis (PCI > 3 mm), which is rarely observed in pediatric age group [15]. Future prospective studies should be necessary to clarify this point.

The worst oral hygiene observed in the group of JDM patients with abnormal lipid profile alert to the risk of bacterial plaque accumulation inducing local inflammatory process, a condition that could contribute to aggravate periodontal and systemic inflammation, leading to lipid changes and increased risk of coronary artery diseases. Therefore, it is necessary to encourage patients and family members to maintain an adequate oral health [4, 36].

Conclusions

Gingival inflammation and attachment loss observed in the studied sample were associated with dyslipidemia and dental changes were more evident the longer was the disease duration.

Gingivitis and poor oral hygiene are important concerns in JDM dyslipidemic patients, especially after long-term disease. These findings indicate the importance of oral health prevention and treatment for these patients, aiming to reduce their early cardiovascular risk.

Acknowledgements

We thank Prof. Dr. Jose Siqueira Tadeu and Dr. Priscila Ribas Guardieiro for their suggestions and comments, and Dr. Ulysses Doria Filho for statistical analysis.

Funding

This study was supported by grants from Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP 2008/58238–4 to CAS), Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq 303422/2015–7 to CAS and 301805/2013–0 to RMRP), Federico Foundation (to CAS and RMRP) and by Núcleo de Apoio à Pesquisa "Saúde da Criança e do Adolescente" of Universidade de São Paulo (NAP-CriAd) to CAS.

Authors' contributions

All authors contributed equally to this paper. All authors read and approved the final manuscript.

Ethics approval and consent to participate

This study was approved by the Local Ethics Committee of our University Hospital and an age-appropriate written informed consent was obtained from all participants and their legal guardians.

Consent for publication

The consent for publication was also obtained from patient's parents or their legal guardian.

Competing interests

The authors declared that they have no competing interests.

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Received: 11 July 2018 Accepted: 31 July 2018
Published online: 05 September 2018

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