



Short communication

Intimate partner violence and temporomandibular joint disorder

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ABSTRACT

Objective: To assess the relationship between Intimate partner violence (IPV) (a highly prevalent form of domestic abuse) with the subsequent development of temporomandibular joint disorder (TMD).

Methods: A retrospective open cohort study using a UK primary care database was undertaken. 18,547 women exposed to IPV were matched by age to 74,188 unexposed women. Defined through clinical codes the outcome of interest was TMD, and adjusted incidence rates (aIRR) were used to describe the relationship after considering covariates of interest.

Results: 94 individuals in the exposed group were clinically coded with TMD during the study period translating to an incidence rate (IR) or 1.59 per 1000 person years. This was in comparison to 342 outcomes in the unexposed group (IR 1.21 per 1000 person years). The unadjusted IRR was 1.31 (95% CI 1.04–1.65; $p < 0.020$) and after adjustment for important covariates increased to 1.45 (95% CI 1.14–1.84; $p < 0.002$).

Conclusion: Our results suggest that the development of TMD may be associated with exposure to IPV in women. **Clinical significance:** In the first cohort to do so, we have identified a moderate association between Intimate partner violence exposure and subsequent development of TMD. This highlights an opportunity for screening of abuse in individuals presenting with TMD.

1. Introduction

Temporomandibular joint disorder (TMD) is the name given to a number of disorders affecting the temporomandibular joint, muscles of mastication surrounding the jaw and related structures [1]. In the United States, TMD have been identified as a common condition with self-reported estimates of prevalence affecting 5% of adults [2]. Previous estimates suggested the lifetime prevalence of TMD related pain was between 3–62% [3]. The pain caused by TMD can be debilitating for patients who experience it, affecting many aspects of their working and social life [4]. Therefore, it is important to have an understanding of the cause of the condition to highlight any suitable opportunities for intervention.

The aetiology of TMD is multifactorial in nature, with risk factors thought to range from; individual genetic and sex-based (more common in females) factors, dental related causes including occlusion issues and bruxism, to stress, depression and anxiety related states [5]. There has been evidence to suggest that patients who have experienced TMD, may

also present with functional and structural changes in the thalamus, somatosensory cortex and some have dysfunction in the pain inhibition systems [6].

Of those patients who present with chronic pain initiated by TMD, some have an increase in generalised pain sensitivity, which can be characteristic of central sensitisation of the nervous system [7]. There has been extensive work exploring changes in the stress response following abuse (childhood and adulthood abuse), which are linked to the development of central sensitisation in somatic disorders which may include TMD in those experiencing chronic pain [8]. Older research suggested that individuals presenting with TMD did, although not statistically significantly differently, self-report a higher prevalence of physical/sexual abuse than individuals without TMD [9]. Another study suggested that individuals with TMD who had experienced physical or sexual abuse experienced worse TMD pain compared to other patients with TMD [10]. However, both of these studies are small and not generalisable to a wider population. Intimate partner violence (IPV) is a form of adulthood abuse which includes physical and sexual abuse but

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also extends to other forms of abuse including emotional abuse and neglect [11]. IPV is estimated to affect one in three women globally, and has a range of physical and psychological consequences [12,13]. The association between IPV and TMD has not been previously tested. Considering the prevalence of IPV, it is important to ascertain of the burden of TMD disease in this cohort in order to provide an opportunity to suggest intervention if required and also to provide further insight into aetiology of TMD exploring whether IPV is a risk factor. Therefore, our aim was to conduct a retrospective cohort study using UK primary care data to explore this relationship

2. Materials and methods

We conducted a population based retrospective cohort study using ‘The Health Improvement Network’ (THIN) database. The THIN database captures electronic records of approximately 3.6 million UK based patients, which are deemed to be representative of the general population in demographic structure and comorbidity burden [14,15]. The database uses a clinical coding system hierarchy called Read codes [16] to categorise symptoms, diagnoses and clinical outcomes. The validity of such coding is dependent on the accuracy of the individual inputting the data on the system. The study period was between 1st January 1995 to 1st December 2017. During this period, we highlighted all women with a Read code relating to being a survivor of IPV forming our exposed group (Read codes: 14 × 3.00, 14 × 8.00, 14XD.00, 14XE.00 and 14XG.00). These individuals were then matched on age (+/- 1 year) to four controls from the database who did not have IPV (unexposed group). Each individual in the exposed group, had an index date assigned to either the earliest date of exposure in the study period or was given as the study start date if their exposure was prior to the study start date. The same index date was assigned to their counterpart in the unexposed group mitigating immortality time bias [17].

The outcome measure was the development of TMD. This was deemed by the presence of a diagnostic Read Code of TMD (Read codes: J046.00, J046011, J046100, J046100, J046400 and J046z00). If an individual in the exposed or unexposed group had the outcome of interest prior to their index date, they were excluded from the study. Baseline data relating to the individual’s alcohol use, smoking status, body mass index (BMI) and deprivation were also collected.

Baseline data were described using means and proportions. Following this a Poisson regression was used to determine an incidence rate ratio (IRR) to describe the IRR of TMD comparing the exposed to the unexposed group. 95% confidence intervals are given with statistical significance set at $p < 0.05$. An adjusted IRR (aIRR) is also given which adjusts for other factors which may have an independent effect on TMD development which include age, smoking status, BMI and deprivation.

3. Results

The baseline characteristics of the group are described in Table 1. 18,547 women with IPV exposure were matched to four controls in the unexposed group. However, 343 (1.9%) individuals in the exposed group had a pre-existing diagnosis of TMD as did 1507 (2.0%) individuals in the unexposed group, therefore these individuals were then excluded from the main results presented in Table 2. At study entry individuals in the exposed group had a higher prevalence of smoking, increased deprivation, increased prevalence of excessive drinking although reduced prevalence of non-excessive drinking and also marginally higher prevalence of individuals who were obese (> 30 kg/m²).

During the study period, there were 94 incident outcomes of TMD in the exposed group which translated to an incidence rate (IR) or 1.59 per 1000 person years. This was in comparison to 342 outcomes in the unexposed group (IR 1.21 per 1000 person years). The unadjusted IRR was 1.31 (95% CI 1.04–1.65; $p < 0.020$) and after adjustment for important covariates increased to 1.45 (95% CI 1.14–1.84; $p < 0.002$).

Table 1
Baseline characteristics.

Baseline Characteristics (Standard Deviation or Percentage)		
	Exposed Group	Unexposed Group
Number of patients	18,547	74,188
Follow-up period (person years)	2.2 (SD 2.3)	3.2 (SD 2.8)
Age (years)	36.9 (SD 12.5)	36.9 (SD 12.5)
Body mass index		
< 25 kg/m ²	7,916 (42.7%)	32,330 (43.6%)
25-30 kg/m ²	3,999 (21.6%)	16,346 (22.0%)
> 30 kg/m ²	3,568 (19.2%)	13,934 (18.8%)
Not available	3,064 (16.5%)	11,578 (15.6%)
Current Smoking status	8,096 (44.7%)	16,039 (21.6%)
Drinking Status		
Non-drinker	5,149 (27.8%)	13,771 (18.6%)
Drinker not excess	8,353 (45.0%)	44,112 (59.5%)
Excessive drinker	1,870 (10.1%)	1580 (2.1%)
Not available	3,175 (17.1%)	14,725 (19.9%)
Townsend index for deprivation		
(Least deprived) 1	1773 (10.1%)	14,160 (20.2%)
2	2,104 (12.0%)	12,881 (18.4%)
3	3,149 (17.9%)	13,548 (19.3%)
4	4,215 (24.0%)	12,601 (18.0%)
5	4,266 (24.3%)	9330 (13.3%)
Not available	2,068 (11.8%)	7691 (11.0%)
Temporomandibular joint disorder at baseline		
TMD	343 (1.9%)	1507 (2.0%)

Table 2
: Risk of developing TMD following exposure to IPV.

	Temporomandibular Joint Disorder	
	Exposed	Unexposed
Number of Patients	18,204	72,681
Numbers of Incident Outcomes	94	342
Person-years	59,138	282,410
Incidence Rate (per 1000 person years)	1.59	1.21
Unadjusted Incidence Rate Ratio (95% Confidence intervals)	1.31 (1.04-1.65)	
p-value	0.020	
Adjusted Incidence Rate Ratio (95% Confidence intervals)*	1.45 (1.14-1.84)	
p-value	0.002	

* Adjusted Incidence rate ratio: adjusted for BMI, age, smoking status and Townsend deprivation index at baseline.

These results suggest that exposure to IPV is associated with the subsequent development of TMD.

4. Conclusions

This brief report summarises our new finding that incident TMD diagnosis is associated with IPV exposure in women (aIRR 1.45 (95% CI 1.14–1.84; $p < 0.002$)), when tested in a UK cohort. We have also identified the IR of the development of TMD in an IPV cohort is 1.59 per 1000 person years.

This type of study design presents with several limitations. The primary limitation relates to the accuracy of recording of both the exposure and outcome of interest. It is clear that the exposure (IPV) appears to be under recorded when compared to global estimates of IPV prevalence [12]. Although not directly comparable due to geographical variation and method of recording, when comparing the outcome (TMD) the overall prevalence of TMD in the control population is slightly lower than estimates in the US [2]. However, under recording of exposure is likely to mean we have possibly underestimated our effect size. We were unable to meaningfully explore a breakdown of the

types of IPV (emotional, sexual, physical and neglect) due to the limited numbers of individuals recorded in these particular categories. We were also unable to explore the effect of the association on a breakdown of the different types of TMD, specifying what the causative factor was for the diagnosis. These should be explored in other cohorts to identify if there is either a dose-response relationship or different relationships appear depending on the type of abuse and type of TMD diagnosed.

Considering the limitations of this study, our results pose particular significance as this is the first cohort globally to assess the outcome of incident TMD following exposure to IPV. Although we were unable to assess the severity of TMD pain symptoms nor define the nature of the abuse (emotional, physical sexual or neglect) we are able to confirm the relationship that exists suggesting that a history of abuse is associated with TMD [9,10]. This is of particular interest in terms of gaining further insight into the aetiology of TMD. As we excluded individuals with TMD prior to the start of the study, we were able to suggest the explore the temporality of the relationship by suggesting IPV exposure precedes TMD diagnosis. This is a relationship that needs to be tested in other cohorts to confirm this relationship. Also although we were unable to explore the biochemical relationship in this study, by defining the temporality of the relationship we can add to the debate surrounding whether it is possibly central sensitisation that occurs following abuse which could precipitate TMD in a subset of patients who experience chronic pain as a result of the diagnosis [8].

In summary, in this study, we have been able to identify for the first time the relationship between TMD to exposure to IPV in women. This is particularly important as it may indicate a potential screening opportunity for individuals who present with TMD to a dental physician to explore a history of abuse, a discussion which recent literature suggests currently happens infrequently in this setting [18]. There are several, simple approaches which can be integrated into daily dental practice such as the AVDR approach; asking about abuse, validating it is not the survivor's fault, documenting and referring appropriately [19]. These approaches have been shown to work effectively in dental settings putting minimal strain on clinicians [18]. Further work is still required in other cohorts to identify if this relationship persists elsewhere and further work is needed to explain the pathway behind this relationship.

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Declaration of interests

None

Author contributions

This study contributed to the PhD thesis for the main author JSC. All authors were involved in conceptualisation of the study. JSC conducted data extraction, analysis and drafted the first version of the manuscript with TT. The final manuscript was reviewed and authorised by all authors.

Conflicts of interest

None.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:<https://doi.org/10.1016/j.jdent.2019.01.008>.

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