

Hostility, pain sites and somatization in a selected population of Craniomandibular Disorders subjects with long-Standing Tension-Type Headache.

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Abstract

Introduction: Chronic Tension-Type Headache is a common/disabling headache both in the general and in clinical populations. How headache becomes chronic has encouraged extensive research in this field of sciences. **Goals:** Evaluate some clinical parameters including hostility, somatization and pain sites in subjects with chronic tension-type headache. **Methods:** The medical and dental records of fifty clinical cases presenting with 10-year or longer tension-type chronicity, fifty subjects with 1-2 years tension-type headache chronicity, forty-five subjects with tension-type headache of 3-5 years chronicity and 26 tension-type headache subjects with 6-9 years duration were retrieved from a database, retrospectively evaluated and compared regarding hostility, pain sites and somatization scores. Clinical examination, palpation, self-report, questionnaires, criteria for tension-type headache, craniomandibular disorders and bruxing behavior and instruments to evaluate somatization and hostility, were used to gather data. Data from these four subsets of Craniomandibular Disorders and tension-type headache subjects were evaluated using Kruskal-Wallis nonparametric statistics. **Outcome:** Mean age in the subgroups with different tension-type headache chronicities were as follows: 1-2 years (mean age=26,8); 3-5 years (mean age=31,3); 6-9 years (mean age=32,4) and 10 years or longer (mean age=40 years), respectively. A statistically and significant age difference was observed only when the subgroups 1-2 years of tension-type headache chronicity (mean=26,8) and 10 years or longer of tension-type headache chronicity (mean 40,0) were compared ($p<0,05$). Means in hostility in the same subgroups are described as follows: 1-2 years tension-type headache chronicity (21,7); 3-5 years tension-type headache chronicity (21,7); 6-9 years tension-type headache chronicity (19,1) and 10 years or longer tension-type headache chronicity (16,3). Kruskal-Wallis statistics ($p<0,001$): Group 1-2 years tension-type headache chronicity versus 3-5 years tension-type headache chronicity subgroup ($p>0,05$); group 1-2 years tension-type headache chronicity versus 6-9 years tension-type headache chronicity ($p>0,05$); group 1-2 years tension-type headache chronicity versus 10 years or longer tension-type headache chronicity group ($p<0,01$); 3-5 years tension-type headache chronicity versus 6-9 tension-type headache chronicity ($p>0,05$); 3-5 years tension - type chronicity versus 10 years or longer tension-type headache chronicity ($p<0,01$); 6-9 years tension-type headache chronicity versus 10 years or longer tension-type headache chronicity ($p>0,05$). Means in self-reported pain sites were as follows: 1-2 years subgroup: 4,7 painful sites; 3-5 years=6,2 painful sites; 6-9 years=6,7 painful sites; 10 years or longer=6,4 painful sites (Kruskal-Wallis statistics ($p<0,0004$): 1-2 years subgroup versus 3-5 years subgroup ($p<0,05$); 1-2 years subgroup versus 6-9 years subgroup ($p<0,01$); 1-2 years subgroup versus 10 years or longer subgroup ($p<0,05$); 3-5 years subgroup versus 6-9 years subgroup ($p>0,05$); 3-5 years subgroup versus 10 years or longer subgroup ($p>0,05$); 6-9 years subgroup versus 10 years or longer subgroup ($p>0,05$). Means in somatization are described as follows: 1-2 years pain chronicity subgroup=8,4; 3-5 years pain chronicity subgroup=10,6; 6-9 years pain chronicity subgroup=12,7; 10 years longer pain chronicity subgroup=12,1). Kruskal-Wallis statistics ($p<0,001$) demonstrated that there was a statistically significant difference in pain sites when some of these subgroups were compared: 1-2 years headache chronicity versus 3-5 years headache chronicity ($p>0,05$); 1-2 years headache chronicity versus 6-9 years hadache chronicity ($p<0,01$); 1-2 years headache chronicity versus 10 years or longer headache chronicity ($p<0,05$); 3-5 years headache chronicity versus 6-9 years headache chronicity ($p>0,05$); 3-5 years headache chronicity versus 10 years or longer headache chronicity ($p>0,05$); 6-9 years headache chronicity versus 10 years or longer headache chronicity ($p>0,05$). **Conclusion:** Subjects with more chronic pain were older, hostility scores decreased in subjects with more chronic tension-type

headache, lower scores in pain sites were found in the less chronic headache subgroups and scores in somatization were higher in the two more chronic headache subgroups.

Keywords: Craniomandibular disorders. Tension-Type Headache. Chronic Pain. Hostility. Pain Sites.

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I. Introduction

Craniomandibular Disorders (CMDs) is a set of terms used in Medicine and Dentistry to describe a well defined set of signs and symptoms of pain and dysfunction occurring in the masticatory muscles, temporomandibular joints (TMJs) and/or adjacent anatomically and functionally related structures in the face, head and neck^[1]. Most common reported signs and symptoms of CMDs include a complaint of pain, abnormalities of jaw movements, tenderness to palpation, a diversity of joint sounds, and headaches of musculoskeletal origin^[2]. Chronic pain is currently recognized as a multidimensional social, economic and psychological problem and as a biopsychosocial disorder associated with biological, psychological and social factors^[3]. Although chronic pain is very complex, this disorder can be better understood on the basis of the influence and/or role of psychological factors^[3]. It has been widely acknowledged that some psychological factors including fear, anxiety, depression, somatization and hostility are directly involved in the experience and clinical manifestations of a chronic pain disorder^[4].

Tension-Type Headache or TTH has been described as a dull, constant, pressure, non pulsatile pain, usually of mild or moderate and sometimes severe intensity and lasting hours or days^[5]. TTH is usually described as a bilateral head pain occurring in the temporal, frontal and occipital regions but sometimes may be described in the whole head. TTH is pain usually radiating in a band – like fashion bilaterally from the occipital/cervical structures to the anterior part of the head and vice-versa which the patient describes as compressing, pressing, band-like or helmet, and or as a tightening sensation around the head^[6].

It has been reported that TTH may transform into chronic headache when influenced by psychological factors including daily stress, anxiety, depression and by analgesic overuse^[7]. Chronic TTH is very difficult to treat since its etiology and interaction of many social and psychological factors are not well understood^[5]. Hundreds of scientific papers have emphasized the fact that chronic headache is associated with a number of psychological and psychiatric disorders including anxiety, somatization, anger-inward and hostility^[3]. These disorders may amplify a pain response which in turn results in more chronic TTH. It has been reported that anger and hostility are implicated in the development and maintenance of chronic pain and that subjects presenting with various chronic pain disorders are characterized by high levels of trait anger and hostility^[6]. It has been accepted that the tendency to suppress anger is a significant determinant factor in chronic pain severity^[8]. Anger, hostility, depression, anxiety and daily life stressful events, constitute important psychological components in TTH subjects^[9]. Somatization is defined as the psychological and unconscious transformation of emotions into the development of somatic/physical problems or complaints. Such disorder constitutes a psychological, economic and social problem for the patient, physician and health institutions^[10]. Somatization is characterized by many physical complaints, including musculoskeletal disorders, depression and pain. These disorders manifest clinically over many years and head pain is a common complaint^[3].

Despite the availability of diagnostic criteria, the many pharmacologic and psychological approaches for headache treatment, and a better understanding of the role of psychological factors, TTH continues to be a clinical challenge more specifically when personality and psychiatric disorders, childhood abuse, negative emotions, stressful life events, the negative effects of chronic drug abuse are observed on clinical and psychological evaluation. Biologic and psychological changes attributed to long-standing headache make the diagnosis and treatment of chronic TTH a potential or real challenge for both the clinician and specialist. Despite the publications of hundreds of studies in the field of headache, there is a paucity of studies about the interrelationship of chronicity, pain sites and hostility in TTH subjects. Thus, this study was designed to test the following hypothesis:

- 1.Higher scores in hostility are observed in CMDs subjects with long-standing TTH.
- 2.There is a positive relationship between more chronic TTH and higher scores in pain sites in CMDs subjects with chronic TTH;
- 3.CMDs subjects with chronic TTH demonstrate higher scores in somatization as compared to CMDs subjects with less chronic TTH;

II. Methods

Patients referred consecutively to the Orofacial Pain Unit School of Dentistry, University of Gurupi are evaluated using a comprehensive and standardized procedure described as follows: Taking the history of the chief complain, palpation of the TMJs and masticatory muscles, use of biomechanical tests to determine the presence of TMJs internal derangements, use of questionnaires to assess main characteristics and type of headache, clinical examination and self-report to evaluate bruxing behavior (BB), type of headache and myofascial pain. Finally, some psychological tests are used to assess depression (BDI), anxiety (TMAS), hostility (Cook-Medley), somatization (Rief and Hiller), dissociation (Bernstein and Putnam) to name a few. Once subjects are completely evaluated, the medical records are stored in a database so as to become available for research of specific variables of interest. Recently, from this database we retrieved 50 medical records of subjects presenting with characteristics of TTH of 1-2 years pain duration, 50 medical records from subjects presenting with TTH of 10 years or longer duration, 45 medical/dental records of TTH subjects with 3-5 years duration and 26 medical records of TTH subjects complaining of TTH of 6-9 years duration. Once these medical records were retrospectively examined, scores in hostility, painful sites and somatization were assessed and compared in each group based on the chronicity of TTH.

Criteria for CMDs: A complain of pain in the masticatory structures usually in the masticatory muscles and TMJs, presence of joint noises based on self-report and clinical examination, difficulty to perform normal movements of the jaw, tenderness to palpation of masticatory muscles and TMJs and headache of musculoskeletal origin.

Criteria for TTH: Pain described as bilateral, occurring in the frontal, temporal, suboccipital and occasionally in the parietal regions. Pain described as dull, aching, constant, sometimes diffuse, tightening, pressure, band-like, nausea occurring only occasionally and absence of vomiting.

Exclusion criteria: Subjects and controls presenting with severe psychiatric or psychological disorders, cognitive difficulties to respond properly to questionnaires, presence of neuromuscular disorders including but not restricted to Parkinson Disease, speech and motor disorders and those subjects that were examined but their information was incomplete and could not be neither stored in the database nor used for any epidemiological or clinical study, were excluded from the investigation.

III. Measures

The Cook-Medley instrument is a classical hostility instrument used to evaluate cynicism, anger, mistrust and aggression. The Cook and Medley^[11] is a 46-item true/false questionnaire derived from the Minnesota Multiphasic Personality Inventory and used widely in epidemiological studies. In this instrument, subjects respond choosing an action/attitude when confronted with a certain personal situation. One of the responses (a or b) is related to an element of hostility. Thirty-two items from the Rief and Hiller^[12] questionnaire were used to assess the presence of physical complaints indicating somatization. This instrument is a self-reported questionnaire in which patients respond to a series of physical complaints selecting "never, rarely, occasionally, frequently and always" to the item. In this study, questions that the patient responded as occurring occasionally, frequently and always were accepted as a positive response to evaluate scores in somatization. Those questions to which the patient reported they occurred rarely, were not accepted as indicating somatization.

IV. Statistical Methods

The Kruskal-Wallis nonparametric statistics was used to evaluate statistical differences when means in pain sites, somatization and hostility were compared in four different subgroups. Significance was accepted if $p < 0,05$.

V. Outcome

This investigation evaluated subgroups of CMDs and TTH subjects with different pain chronicities: 1-2 years ($n=50$); 10 years or longer ($n=50$); 3-5 years ($n=45$) and 6-9 years ($n=26$). Mean age in these subgroups were about 26,8 years ($SD=9,9$, range=14—53), 40 years ($SD=11,2$, range=19—66); 31,3 years ($SD=11,3$, range=14-57); and 32,4 years ($SD=11,4$, range=19-60), respectively. There was a statistically and significant difference when age was compared in these subgroups (Kruskal-Wallis nonparametric test $p < 0,05$) but such difference was observed only when the subgroup 1-2 years of TTH chronicity (mean=26,8) was compared with the subgroup 10 years or longer TTH chronicity (mean=40).

Means in hostility were about 21,7 ($SD=4,8$, range=11-30) in the 1-2 years TTH chronicity subgroup; 21,7 ($SD=4,8$, range=11-30) in the 3-5 years TTH chronicity subgroup; 19,1 ($SD=4,8$, range=10-28) in the 6-9 years TTH chronicity subgroup; and 16,3 ($SD=4,6$, range=7-26) in the 10 years or longer TTH chronicity subgroup. There was a statistically and very significant difference when means in hostility were compared in these different subgroups (Kruskal-Wallis statistics $p < 0,001$): Group 1-2 years TTH chronicity versus group 3-

5 years TTH chronicity ($p>0,05$); group 1-2 years TTH chronicity versus group 6-9 years TTH chronicity ($p>0,05$); group 1-2 years TTH chronicity versus group 10 years or longer TTH chronicity ($p<0,01$); group 3-5 years TTH chronicity versus group 6-9 years TTH chronicity ($p>0,05$); group 3-5 years TTH chronicity versus group 10 years or longer chronicity ($p<0,01$); group 6-9 years TTH chronicity versus group 10 years or longer of chronicity ($p>0,05$).

Means in self-reported pain sites on initial clinical evaluation were as follows: 4,7 painful sites ($SD=1,6$, range=2—7) in the subgroup with 1-2 years TTH chronicity; 6,2 painful sites ($SD=1,6$, range=4-12) in the subgroup with 3-5 years TTH chronicity; 6,7 painful sites ($SD=1,5$, range=5-11) in the subgroup with 6-9 years TTH chronicity, and 6,4 painful sites ($SD=2,1$, range=4-13) in the subgroup with 10 years or longer TTH chronicity. Because Kruskal-Wallis statistics demonstrated that $p<0,0004$, there was a statistically and very significant difference when such means were compared: Group 1-2 years TTH chronicity versus group 3-5 years TTH chronicity ($p<0,05$); group 1-2 years TTH chronicity versus group 6-9 years TTH chronicity ($p<0,01$); group 1-2 years TTH chronicity versus group 10 years or longer TTH chronicity ($p<0,05$); group 3-5 years TTH chronicity versus group 6-9 years TTH chronicity ($p>0,05$); group 3-5 years TTH chronicity versus group 10 years or longer TTH chronicity ($p>0,05$); group 6-9 years TTH chronicity versus group 10 years or longer TTH chronicity ($p>0,05$).

Means in somatization are described as follows: 8,4 ($SD=4,0$, range=1-16) in the 1-2 years TTH chronicity subgroup; 10,6 ($SD=3,8$, range=5-19) in the 3-5 years TTH chronicity; 12,7 ($SD=4,9$, range=5-24) in the 6-9 years TTH chronicity and 12,1 ($SD=4,4$, range=6-24) in the 10 years or longer TTH chronicity. There was a statistically and very significant difference when scores in somatization were compared in these subgroups (Kruskal-Wallis statistics $p<0,001$): 1-2 years TTH chronicity subgroup versus 3-5 years TTH chronicity subgroup ($p>0,05$); 1-2 years TTH chronicity subgroup versus 6-9 years TTH chronicity subgroup ($p<0,01$); 1-2 years TTH chronicity subgroup versus 10 years or longer chronicity subgroup ($p<0,05$); 3-5 years TTH chronicity subgroup versus 6-9 years TTH chronicity subgroup ($p>0,05$); 3-5 years chronicity subgroup versus 10 years or longer chronicity subgroup ($p>0,05$); 6-9 years TTH chronicity subgroup versus 10 years or longer chronicity subgroup ($p>0,05$).

VI. Discussion

1. The main objective of this investigation was to compare scores in hostility, pain sites and somatization in a group of CMDs subjects with “acute TTH” (1-2 years of chronicity) with other subgroups presenting with TTH of longer duration. **Interesting to note is that hostility scores decreased from the less chronic TTH subgroup to the more chronic TTH one.** At first sight, these findings seem contradictory. However and because there is a dynamic interrelation of depression with anger inward and hostility, there are reasons to believe that hostility has a dynamic behavior in populations with different headache chronicities. It may be that there is a point in time in which TTH individuals becomes less hostile but more depressed as more chronic pain, hostility and anger-inward have a significant influence in the development of depression in chronic pain patients. Thus, more chronic TTH is more likely to be associated with signs and symptoms of depression and lower scores in hostility as very chronic TTH subjects become more depressed, hopelessness, worthlessness and more negative about life events. These reactions are probably associated with pain of longer duration, frustration associated with failed treatments, excessive consultations with different doctors, ineffective medication and pessimism about the outcome of treatment. With time, hostility decreases but overt depression increases. Congruent with the aforementioned points of view, Stewart and colleagues^[13] evaluated longitudinal associations among measures of depressive symptoms and /hostility/anger in a cohort of older adults using the Cook-Medley hostility instrument. They found that baseline hostility predicted a 6-year increases in depression concluded that cognitive aspects of hostility or anger may precede future increases in depressive symptoms and also that hostility precipitate and/or maintain symptoms of depression. TTH is basically a myofascial pain type and thus vulnerable to stressful situations. Thus, stress and hostility may be associated with greater adrenergic responses^[14] that when chronic may maintain and/or perpetuate TTH. With time, part of such hostility or anger transforms in depression. Hostility may decrease sleep duration (fewer hours of sleep), which in turn may be one factor triggering and or maintaining fatigue and depression^[15] These assumptions are supported at least in part by one investigation^[16] reporting that more chronic pain is likely to generate depression, withdrawal, irritability and somatic preoccupation. Further, inability to modulate and express intense, unacceptable feelings such as anger may mediate the relationship between anger, hostility and depression^[16]. TTH patients have a significant impairment of anger control and higher level of anxiety suggesting a connection between anger, duration of pain experience and also depression. Even though researchers in one investigation did not specify the chronicity of headache, they found that depression scores were higher in a group presenting with TTH^[17]. Nicholson and associates^[18] examined differences in anger expression in individuals with ($n=171$) and without headache ($n=251$). Researchers used the Cook-Medley instrument as was the case in our study. Although they did not evaluate subgroups by the chronicity of TTH,

they reported that subjects in the headache group had higher levels of anger. They found that anger-in contributed the most to predict headache status and concluded that persons with headache hold their anger-in more frequently than those without headache.

Bag and associates^[19] evaluated 75 subjects with migraine and 55 subjects with TTH and reported that migraine and TTH patients had significantly higher scores on measures of anxiety, depression and hostility. In the current study we report higher, intermediate and lower hostility scores according to the chronicity of TTH. In the current investigation we examined TTH subjects with different chronicities of TTH and we report different groups with different scores in hostility. Emadi and colleagues^[20] looked at psychological variables in subjects with acute and chronic migraine. They reported that variables that had a significant effect on headache chronicity were sex, marital status, headache duration, depression and anger. Their results showed that disability and depression were higher in patients with chronic migraine compared to those suffering from the acute type. In a previous investigation, Molina and associates^[21] evaluated a group of 100 TTH subjects and 54 controls with no headache. Mean in hostility levels in the TTH subgroup was about 18,5 in the total TTH group which was similar to the mean of 21,7 in the subgroups reporting 1-2 years and 3-5 years of TTH chronicity. On the other hand, the mean of 16,8 in their control group was similar to the mean of 16,3 in the 10 years or longer TTH chronicity. The low mean scores in the 10 years or longer chronicity observed in the current investigation^[22] is very likely related to high scores in depression. One investigation asserts that TTH individuals are sensitive, somewhat resentful and rigid. Further, chronic headache suffer from less openness about feelings, greater instability, increased irritability and hostility^[23]. A previous investigation^[24] examined a subgroup of 100 TTH subjects. Although experimental subjects were not separated in subgroups by the chronicity of headache, researchers reported a mean of about 18,5 in hostility levels, an outcome very similar to the outcome (HO=19,7) found in the current investigation.

People with depressive illness often have symptoms of overt or suppressed anger indicating that subgroups of subjects with hostility or anger can be observed. Anger has been thought to play an integral role in depression. Several psychoanalytic theorists and clinicians have suggested that conflict and difficulties in coping with anger play a central role in the onset and persistence of depression. Depression has also been conceptualized as a kind of self directed anger and a propensity toward hatred and hostility has been noticed in patients with depression. In line with these observations, a positive association has been found between the severity of depression, levels of hostility and anger expression (anger directed inward or outward)^[25].

The Freudian theory of inhibited anger as a powerful mechanism central to the pathogenesis of depression has been one of the cornerstone of psychiatric thinking^[26]. The most important aspect of Freudian study in this relationships is the fact that levels of depression varies greatly with the intensity or frequency of inhibited anger. Freud studies in this field also indicate that anger inward is responsible for the levels of depression in any individuals. Further, there are many factors responsible for the individuals' tendency to suppress anger or hostility. Hatch and associates^[27] used a number of standardized psychometric tests to gather psychological data in group of 47 episodic headache free controls. Even though they did not assess groups by the chronicity of TTH, they reported that headache subjects showed significantly higher levels of suppressed anger implying a significant interrelationship between anxiety and depression and depression and anger/hostility in TTH subjects. It may be that anger/hostility is a psychological characteristic of tension-type headache. However high scores can be observed in those who do not conceal hostility or anger and low hostility scores are more frequently found among those who take their anger inward and demonstrate higher scores in depression. In line with these considerations, Bag and Hacıhasanoglu^[19] reported that migraine and TTH subjects in their study showed higher levels of depression, anxiety and hostility. This assumption is reinforced by the fact that high and low levels of depression are observed in TTH subjects. Even though, TTH is frequently co-morbid with anxiety and depression and with reports of repressed anger and resentment^[28], the outcome of the current investigation shows that individuals with low, intermediate and higher levels of hostility can be found in a population of TTH individuals.

In the current study we did not compared a TTH subgroup with a control (No TTH subgroup) one regarding psychological measures. Rather, we compared some psychological measures in less chronic (1-2 years pain chronicity) and more chronic (3-5 years, 6-9 years and 10 years or longer TTH chronicity). Lowest scores in hostility were observed in the subgroup with 10 years or longer TTH. Because in the current investigation only the most chronic TTH subgroup showed the lowest scores in hostility, we may say that anger inward is more frequently observed in those with more chronic TTH in the same way that some investigators believe that more chronic pain corresponds to higher scores in depression. In one investigation^[4] in subjects with migraine and TTH, researchers reported that compared to controls, patients with chronic TTH and those with migraine associated with episodic and chronic tension-type headache showed an increase in scores for depression. Thus, findings in such investigation indicate that TTH of longer duration was related to higher scores in depression and lower scores in hostility or anger inward. Many headache patients tend to turn anger inward toward themselves and become more depressed and helpless.

In the current study, the more chronic TTH subgroups demonstrated **higher scores in pain sites** as compared with the 1-2 years TTH chronicity subgroup. The current literature suggest that there may be a number of clinical and neurophysiological factors implicated in the development of greater number of pain sites in individuals with chronic CMDs or with TTH. These factors include depression, central and peripheral sensitization, failure of the descending inhibitory system and higher scores in somatization. More specifically, many pain sites may contribute with more chronic TTH. Further, TTH is by definition, a form of myofascial pain arising in trigger points in the cervical and neck regions. Thus, as TTH becomes more chronic influenced by the presence of greater number of pain sites and some perpetuating and contributing factors, some latent and/or painful muscle areas becomes increasingly sensitized and transform themselves into new trigger point which in turn further sensitize nervous structures leading to episodes of TTH. Some of these contributing or perpetuating factors include muscle tension, bad posture, stress or mental tension, parafunctional behavior and somatization. These assumptions are in line with one investigation^[5] indicating that TTH represents a painful condition with a greater myofascial component. Thus, we may say that myofascial pain is a strong precipitating factor for TTH.

These considerations are also echoed by one investigation^[28] reporting that muscle tension is a common characteristic of TTH. Anxiety, muscle tension and the presence of multiple painful areas or trigger points are strongly correlated. Because of this “sensitization” of painful muscle areas, frequency, intensity and anatomic areas with pain in TTH may change with time as they become more sensible and more “sensitizing”. In this regard, one investigation^[28] defines TTH as a dynamic condition in which stress is a common and powerful mediator increasing headache sensitivity in the chronic form of TTH^[28]. In the current investigation higher scores in pain sites were observed in those subjects with more chronic headache. As explained previously, many factors **including stress, anxiety, muscle tension, poor neck and posture, anger inward, somatization and presence of cervical trigger points contribute to more chronic TTH**. Somatization may be defined as “a complex psychophysiological phenomenon in which anger is kept suppressed but may manifest in the form of “physical complains”. One investigation^[29] asserts that anger inward mediated by somatization increases the intensity of headache. Intensification or amplification of headache occurs when greater number of pain sites, increased sensitivity, higher scores in depression and somatization predominate. This interrelation is very difficult to analyze as one element in the chain, contributes with the other. For instance, somatization contributes with the transformation of psychological conflicts into bodily symptoms which in turn increase muscle tension. Somatization is a psychiatric disorder thought to facilitate the development of physical complaints including headache and pain in multiple pain sites in other areas of the body. Somatizers are characterized by chronic pain in multiple anatomic sites^[3] even in a restricted anatomic one, for example head, face and neck. A high percentage of chronic pain patients demonstrate elevated somatization scores^[3].

Pain from the pericranial musculature, from facial and temporomandibular joint areas together with pain from sensitive areas with or without myofascial trigger points, may generate prolonged nociceptive afferent stimuli originating from sensible myofascial tissue and contributing to central and peripheral sensitization. Thus, the greater the number of painful areas with pain, mainly those from the facial, temporomandibular joint and cervical structures, the more frequent and intense pain contributing to central and peripheral sensitization, rendering pain in TTH subjects more chronic. In this regard, one study^[5] asserts that lower level segments of the SNC, the dorsal horn and the trigeminal system may be sensitized from peripheral painful areas, contributing to central and peripheral sensitization and prolonged and more intense pain. In the current investigation, scores in somatization increased from the less chronic TTH subgroup to the more chronic TTH ones. Somatization is characterized by the presence of non recognized psychic conflicts which are transformed or translated into physical complaints. Somatizers have an increased sensitivity to pain and there is a direct correlation between more chronic pain and higher scores in somatization. In line with these assumptions in one investigation^[30], researchers did not evaluate TTH patients by chronicity of TTH but reported that a high percentage of the 217 TTH subjects they evaluated met diagnostic criteria for somatization^[30]. Findings in the current study are also reinforced by one investigation^[10] reporting that many patients with recurrent headache have somatization of emotions as a major component of their complaints. The most common psychiatric disorders described in subjects demonstrating signs and symptoms of chronic TTH are anxiety, depression and psychosomatic disorders^[31]. In the current investigation we found one group with low scores, one group with intermediate scores and two groups with higher scores in somatization. Considering that taking anger inward and the development of somatic symptoms are interrelated and that we observed that two subgroups in the current study demonstrated higher scores in somatization, findings in the current investigation are in line with one study^[32] reporting that in patients with a psychosomatic diagnosis, difficulty in switching attention from the emotional material was predicted by the presence of somatic symptoms, depression, childhood trauma and dissociation. There is an association between excessive anger suppression or uncontrolled anger and some aspects of psychosomatic disorders^[32].

Patients who experience emotions intensely but suppress emotional expression suffer the most from the impact of symptoms of disease or ailments^[32]. Negative emotions usually containing elements of anger, anger suppression or hostility constitute significant factors in the progression of chronic diseases including headaches^[33]. Because we found higher scores in somatization in the two more chronic TTH subgroups, this outcome is echoed by one investigation^[3] reporting that somatization disorders are frequently associated with chronic pain.

VII. Conclusion

This investigation demonstrated that means in hostility, in pain sites and somatization scores varies according with the chronicity of TTH headache. Means in pain sites and somatization increased from the “acute” TTH subgroup to those more chronic TTH subgroups. The novelty of this investigation is that we identified a subgroup of CMDs and very chronic TTH subjects presenting with low scores in hostility. Even though we used clinical examination, a questionnaire and two valid tests to assess pain sites, hostility and somatization in four different subgroups, new investigations including longitudinal ones would be extremely useful in this type of research to add more information and thus, reinforce findings in the current study. Further, it would be extremely interesting to evaluate scores in pain sites, hostility and somatization in CMDs subjects with very chronic TTH in order to improve the psychological profile of very chronic TTH subjects as a group.

References

- [1]. Molina OF, Rank R, Ogawa WN, Simiao BR, Rezende JE, Marçal R, Abreu CM. Jutting the jaw forward in different stages of temporomandibular joint internal derangements: A multiple comparison study. *IOSR –JDMS* 2020; 19: 32-38.
- [2]. Yadav U, Ahmed J, Ongole R, Shenoy N, Sujir N, Natarajan S. Influence of psychosocial factors and parafunctional habits in temporomandibular disorders: A cross-sectional study. *Perm J* 2020; 24: 144-48.
- [3]. Manchikanti L, Fellows B, Singh V. Understanding psychological aspects of chronic pain and interventional pain management. *Pain Physician* 2002; 5: 57-82.
- [4]. Perozzo P, Savi L, Castelli L, Valfré W, Giudice R, Gentile S et al. Anger and emotional distress in patients with migraine and tension-type headache. *J Headache Pain* 2005; 6: 392-99.
- [5]. Castells EC, Vázquez E, Gay C. Use of amitriptyline for the treatment of chronic tension-type headache. Review of the literature. *Med Oral Patol Oral Cir Bucal* 2008; 13: 567-72.
- [6]. Molina OF, Peixoto MS, Aquilino RN, Rank R. The modulating effect of bruxism as a form of suppressed hostility on depression in a selected population of tension-type headache and craniomandibular disorders. *Cadernos UNIFOA* 2011; 15: 91-100.
- [7]. Ramírez LM, Sandoval GP, Ballesteros LE. Temporomandibular disorders referred to a craniofacial-cervico-facial clinic. *Med Oral Patol Oral Cir Bucal* 2005; 10: 18-26.
- [8]. Burns JW, Bruel S, Quartana P. Anger management style and hostility among patients with chronic pain. *Psychosomatic Med* 2006; 68: 786-93.
- [9]. Venable V, Carlsson SR, Wilson J. The role of anger and depression in recurrent headache. *The Journal Head Face Pain* 2001; 41: 21-30.
- [10]. Abbas A, Lovas D, Purdy A. Direct diagnosis and management of emotional factors in chronic headache patients. *Cephalalgia* 2008; 1305-14.
- [11]. Cook WW, Medley DM. Proposed hostility and pharisaic-virtue scales for the MMPI. *J App Psych* 1954; 238: 414-18.
- [12]. Rief W, Hiller W, Geissner E, Fichter MM. A two-year follow up of patients with somatoform disorders. *Psychosomatics* 1995; 36: 376-86.
- [13]. Stewart JC, Fitzgerald GJ, Kamarck TW. Hostility now, depression later? Longitudinal associations among emotional risk factors for coronary artery disease. *Ann.Behav.Med* 2010; 39: 258-66.
- [14]. Wong JM, Na B, Regan MC, Whooley MA. Hostility, health behaviors, and risk of recurrent events in patients with stable coronary heart disease: Findings from the heart and soul study. *J Am Heart Assoc* 2013; 2: 1-10.
- [15]. Gastelle BT, Maier KJ. The relations of cynical hostility and depression to sleep. *J Undergrad Res* 2011; 16: 163-67.
- [16]. Suvinen TI, Reade PC. Temporomandibular disorders: A critical review of the nature of pain and assessment. *J Orofac Pain* 1995; 9: 317-39.
- [17]. Chen W, YU S, Zhu J, Chai H, He W, Wang W. Personality characteristics of male sufferers of chronic tension-type headache and cervicogenic headache. *Clin Neurol* 2012; 8: 69-74.
- [18]. Nicholson RA, Gramling SE, Ong JC, Buenevar L. Differences in anger expression between individuals with and without headache after controlling for depression and anxiety. *J Head and Face Pain* 2003; 43: 651-63.
- [19]. Bag B, Hacıhasanoglu R, Tufekci FG. Examination of anxiety, hostility and psychiatric disorders in patients with migraine and tension-type headache. *International J Clin Pract* 2005;59: 515-21.
- [20]. Emadi F, Sharif F, Shaygan M, Sharifi N, Ashjazdeh N. Comparison of pain related and psychological variables between acute and chronic migraine patients, and factors affecting headache chronicity. *IJCBNM* 2019; 7: 192-200.
- [21]. Molina OF, Santos ZC, Sobreiro MA, Peixoto MS, Simião BR. Hostility, depression, and anger-in in headache subgroups with bruxism and craniomandibular disorders. *IOSR-JDMS* 2018; 17: 19-25.
- [22]. Andrasik F, Blanchard EB, Arena JG et. Psychological functioning in headache sufferers. *Psychosom Med* 1982; 44: 171-182.
- [23]. Boyle SW, Church WT, Byrnes E. Migraine headache and anger. *Best Pract Mental Health* 2005; 2005: 47-56.
- [24]. Molina OF, dos Santos J. Hostility in TMD/bruxism patients and controls: A clinical comparison study and preliminary results. *Cranio* 2002; 20: 1-7.
- [25]. Riley WT, Treiber FA, Woods MG. Anger and hostility in depression. *J Nerv Ment Dis* 1989; 177: 668-74.
- [26]. Lutton S. Anger and depression: Theoretical and clinical considerations. *Nordic J Psychiatry* 2007; 61: 246-51.
- [27]. Hatch JP, Schoenfeld LS, Boutros NN, Seleshi E, Moore PJ, Cyr-Provots M. Anger and hostility in tension-type headache. *J Head and Face Pain* 2005; 31: 302-04.

- [28]. Waldie KE, Buckley J, Bull PN, Poulton R. Tension-type headache: A life course review. *J Headache Pain Management* 2015; 1: 1-7.
- [29]. Öz O, Erdem M, Yücel M. Evaluation of anger, anger expression style and mood profile in tension-type headache. *Arch Neuropsychiatry* 2011; 48: 171-74.
- [30]. Puca F, Genco S, Prudenzano MP et al. Group for the study of Psychopathological headaches: Psychiatric comorbidity and psychological disorders in patients with tension-type headache. *Cephalalgia* 1999; 19: 159-164.
- [31]. Battistutta S, Aliverti R, Montico M, Zin R, Carrozzi M. Chronic tension-type headache in adolescent. Clinical and psychological characteristics analyzed through self- and parent-report questionnaires. *J Pediatric Psychol* 2009; 34: 697-706.
- [32]. Güney Z, Sattel H, WithoftM, Henningsen P. Emotion regulation in patients with somatic symptom and related disorders: A systematic review. *PLOS ONE* 2019; 14: 1-29.
- [33]. Majnatic L, Stolnik D. Hostility in chronic disease .CPQ *Neurology and Psychology*. 2018; 2: 1-5.

Table 1: Social and demographic data in subsets of CMDs and TTH subjects with different pain durations.

	1-2 years	10 years/longer	3-5 years	6-9 years
AGE				
Mean	26,8	40	31,3	32,4*
SD	9,9	11,2	11,3	11,4
Range	14-53	19-66	14-57	19-60
GENRE				
Females	47	47	42	24
Males	3	3	3	2
Totals	50	50	45	26

Kruskal-Wallis nonparametric statistics (p<0,0001): 1-2 years versus 3-5 years (p>0,05); 1-2 years versus 6-9 years (p>0,05); 1-2 years versus 10 years or longer (p<0,001); 3-5 years versus 6-9 years (p>0,05).

Table 2: Means, standard deviation and range in hostility, painful sites and somatization in four subgroups of CMDs presenting with different TTH durations.

SUBGROUPS BY CHRONICITY OF TTH (YEARS)

	1-2	3-5	6-9	10/LONGER
	n=50		n=45	
	n=26		n=50	
HOSTILITY				
Mean	21,7	21,7	19,1	16,3*
SD	4,8	4,8	4,8	4,6
Range	11—30	11-30	10—28	7—26
PAIN SITES				
Mean	4,7	6,2	6,7	6,4**
SD	1,6	1,6	1,5	2,1
Range	2—7	4-12	5—11	4—13
SOMATIZATION				
Mean	8,4	10,6	12,7	12,1***
SD	4,0	3,8	4,9	4,4
Range	1—16	5—19	5—24	6—24

*Kruskal-Wallis statistics (p<0,001), a statistically very significant difference: Group 1-2 versus group 3-5 (p>0,05); group 1-2 versus group 6-9 (p>0,05); group 1-2 versus group 10 or longer (p<0,01); group 3-5 versus group 6-9 (p>0,05); group 3-5 versus group 10 or longer (p<0,01); group 6-9 versus group 10 or longer (p>0,05).

**Kruskal-Wallis statistics (p<0,0004), a statistically very significant difference:

Group 1-2 versus group 3-5 (p<0,05); group 1-2 versus group 6-9 (p<0,01); group 1-2 versus group 10 or longer (p<0,05); group 3-5 versus group 6-9 (p>0,05); group 3-5 versus group 10 or longer (p>0,05); group 6-9 versus group 10 or longer (p>0,05).

***Kruskal-Wallis statistics (p<0,001), a very statistically and significant difference: 1-2 years TTH chronicity subgroup versus 3-5 years TTH chronicity subgroup (p>0,05); 1-2 years TTH subgroup versus 6-9 years TTH subgroup (p<0,01); 1-2 years chronicity subgroup versus 10 years or longer TTH chronicity subgroup (p<0,05); 3-5 years TTH chronicity subgroup versus 6-9 years TTH chronicity subgroup (p>0,05); 3-5 years chronicity subgroup versus 10 years or longer chronicity subgroup (p>0,05); 6-9 years TTH chronicity subgroup versus 10 years or longer chronicity subgroup (p>0,05).