



A CASE REPORT ON FIBROMYALGIA WITH MODERATE DEPRESSION IN A TERTIARY CARE TEACHING HOSPITAL, TIRUPATHI, ANDHRAPRADESH, INDIA

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ABSTRACT

The association between depression, fibromyalgia suggests that they divide the model of developmental traumatology. This study aimed at presenting a case of depressive disorder and fibromyalgia as well as at discussing causes and consequences of both diagnoses. Here a 36 year old female patient, with fibromyalgia and depressive disorder, current severe episode, without psychotic symptoms, with no somatic symptoms for approximately six months. History of stressing events with loss of affective relationship problems is present. Early life stress may be the causal factor of painful symptoms in depression and fibromyalgia.

Key Words:- Depressive disorder, Fibromyalgia, Duloxetine.

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Quick Response code



DOI:
<http://dx.doi.org/10.21276/ijpt.2020.10.1.2>

Received:12.09.19

Revised:01.10.19

Accepted:16.10.19

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INTRODUCTION

Fibromyalgia (FM) is a rheumatological clinical entity characterized by chronic pain of unknown etiology lasting more than three months, presences of 11 out of 18 hypersensitive points referred as trigger points, and diffuse pain, fatigue, disturbed sleep, poor quality of life .It is more prevalent among females and is associated to insomnia, fatigue and psychological stress (Richard HG

et al., 2012). The prevalence of depression varies from 49% to 80% in female.

Depression worsens social and emotional functionality and the quality of life of female patients.² Exposing developing brain to stress results in hyper functioning amygdala, decreased hypo campus activity with altered negative feedback in glucocorticoid receptors, hypo functioning dopaminergic mesocorticolimbic system and HPA axis hyperactivation.

Traumatology development model explains how early life stress leads to permanent developing brain modifications, with changes in neurotransmitters and hormones modulating the neuronal migration development processes, differentiation, synaptic proliferation which may affect the developing brain (Tsigos C and Chrousos GP, 2002).

This study aimed at observing the association of depressive disorder and to fibromyalgia, in addition to biological stress changes and relationships with depression and fibromyalgia.

CASE REPORT

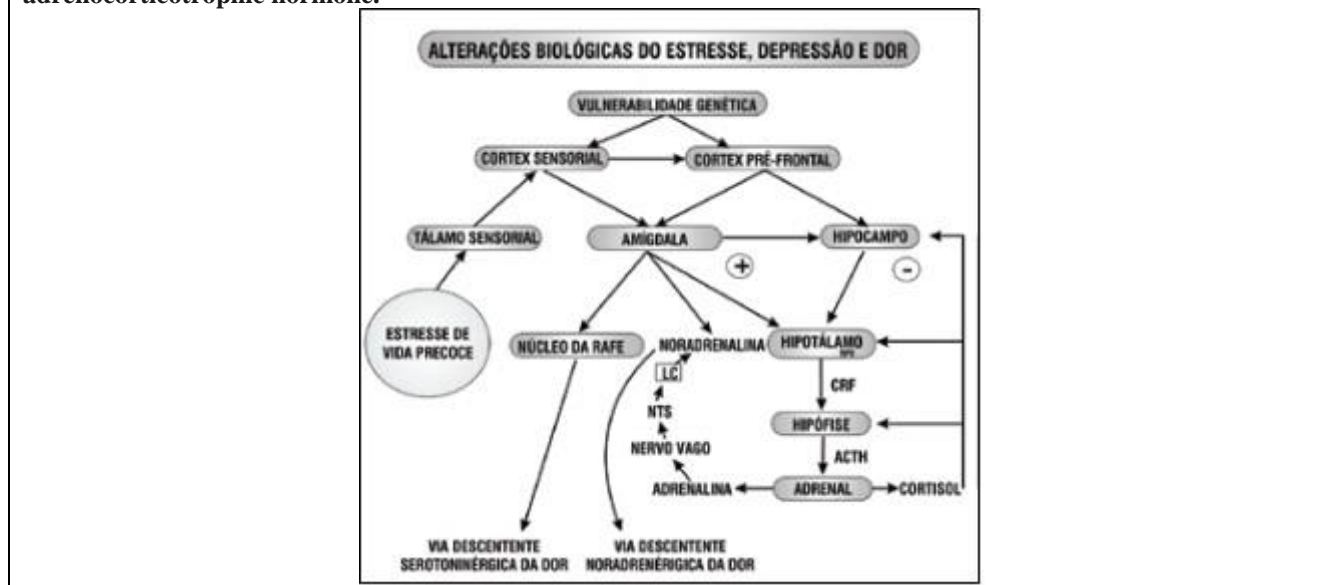
A 36 – year old female visited to the psychiatry department of SVIMS with her aunt. On presentation she complained of multiple joint pains (2-3yr), heaviness of body, and difficulties in performing day to day activities

and occasional sleep disturbances, increased thinking, sad mood. There was no history of other significant illness. Personal history decreased appetite, sleep disturbed. Patient was apparently normal 4 years before pain started as low back ache that gradually multiple joint pains started. On MSE: patient is conscious, co-operated with depressed and low mood.

Table 1. Lab parameters

PARAMETER	OBSERVED VALUE	REFERENCE VALUE
Pulse rate	72beats/min	60-100beats/min
B.P	140/90mmof Hg	120/80mmof Hg
Respiratory rate	20cycles/min	12-20cycles/min
ESR	28mm/1 st hr	1-20mm/1 st hr
Serum alkaline phosphatase	240IU/L	90-120IU/L
Hemoglobin	9gm/dl	12-14gm/dl

Figure 1 – Biologic alterations of stress related to depression and pain. PVN = paraventricular nucleus; LC – locus ceruleus; STN =solitary tract nucleus; CRF = corticotrophic factor; NR = nucleus of raphe; ACTH = adrenocorticotrophic hormone.



DISCUSSION

FM is characterized by significant spontaneous pain. In contrast, many pain conditions are characterized less by spontaneous pain and more by movement-evoked pain. The inability to alleviate pain by quiescence or postural adjustment may contribute to depression. Similarly, the extensive spontaneous pain of FM is accompanied by a large number of symptoms and comorbidities that likely contribute to the probability of reactive depression (Richard HG *et al.*, 2012).

A number of laboratory studies have explored the interaction of depression and evoked pain sensitivity in fibromyalgia. The acute stress response, detailed in numerous reports, involves activation of the hypothalamic-pituitary-adrenal (HPA) axis which is coupled to the autonomic and limbic systems. Activation of the HPA system involves a chain of events. Corticotrophin-releasing hormone (CRH) is secreted by the hypothalamus, which results in pituitary secretion of

ACTH. This CRH effect is synergistically augmented by hypothalamic secretion of arginine vasopressin (AVP). Increased ACTH in turn stimulates adrenal secretion of cortisol. Cortisol is a potent glucocorticoid that activates cytoplasmic receptors throughout the body to ultimately mobilize action and inhibit vegetative processes such as reproduction and growth. The glucocorticoids also provide negative feedback regulation of the HPA axis via multiple pathways acting on the hypothalamus and pituitary. Early life stress may contribute to adult life diseases, such as depressive disorder and FM. Early life stress is related to HPA axis activation leading to changes in the amygdala-hippocampus complex. Hippocampus and prefrontal cortex inhibit HPA axis, while the amygdala activates HPA axis (Sandra OVN *et al.*, 2012). (Fig. 1) These effects of the HPA axis activation are integrated with the locus coeruleus-norepinephrine system (LCNE) that activates brain systems involved in affect and anticipation, precipitation, propagation and

termination of stress-related activity and activation of pain (Tsigos C and Chrousos GP, 2002).

Depression and FM also appear to be linked because drugs with dual serotonergic and noradrenergic actions are used to treat both conditions. The two prime examples are the noradrenergic, serotonergic reuptake inhibitors (SNRIs) Duloxetine and milnacipran, antidepressants approved recently in the US for treatment of FM (Richard HG *et al.*, 2012). A recent secondary analysis of 4 clinical trials of duloxetine in fibromyalgia explored treatment effects in patients who were comorbid with MDD. This analysis demonstrated the relative independence of MDD and fibromyalgia in that the baseline level of one did not affect the treatment efficacy of the other (Marangell LB *et al.*, 2011). Thus the patient was treated with the medication T. Ventab plus 50 mg OD, T. Rejunex plus 1 tab OD, T. Dulane 30 mg OD, T. coricum plus 1tab OD. Ventab plus is combination of desvenlafaxine 50 mg + clonazepam 0.5mg. It is serotonin- nor epinephrine reuptake inhibitor, and is used to treat major symptoms of depression (Robert MB, 2014). Duloxetine inhibits serotonin metabolism cause decrease in proinflammatory cytokine activity and increase in anti inflammatory cytokine as well as sodium ion channel blockade according to medications used in fibromyalgia this mechanism apply to both depression and pain. There is good evidence that increasing synaptic levels of nor epinephrine activates the descending

inhibitory pain system. This is the rationale for the use of SNRIs. Since they are selective and thus have fewer side effects and remain effective over the long term they are the FDA approved for treating pain from fibromyalgia with depression.

CONCLUSION

Depression and FM share both common features and differences at multiple physiological and psychological levels. Depressed mood may be due to internal dysregulation of the HPA system while the persistent pain of FM may be due to known cytokine-mediated HPA activation (Tanriverdi F *et al.*, 2007; Wingenfeld K *et al.*, 2008; Gold PW and Chrousos GP, 2002). Similarly, involvement of systems mediated by serotonin and norepinephrine may be common to both FM and depression, yet the differential effects of NSRI and tricyclic treatments suggest at least distinct differences in the functioning of these systems in these two disorders. Early life stress may be implied as causal factor for painful symptoms in depression and FM.

ACKNOWLEDGEMENT

Nil

CONFLICT OF INTEREST

Nil.

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Cite this article:

Saranya T, Heena kausar A , Robin George, Sunil kumar E. A Case Report On Fibromyalgia With Moderate Depression In A Tertiary Care Teaching Hospital, Tirupathi, Andhrapradesh, India. *International Journal of Pharmacy & Therapeutics*, 11(1), 2020, 5-7. DOI: <http://dx.doi.org/10.21276/ijpt.2020.11.1.2>



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