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COMORBIDITY OF DEPRESSIVE AND DERMATOLOGIC DISORDERS – THERAPEUTIC ASPECTS

Pavo Filaković[1], Anamarija Petek[2], Oliver Koić[1], Ljiljana Radanović-Grgurić[2] & Dunja Degmečić[1]

[1]*School of Medicine Osijek, University of Josip Juraj Strossmayer, Osijek, Croatia*
[2]*Psychiatric Clinic, Clinical Hospital Osijek, Osijek, Croatia*

SUMMARY

Depressive disorders are more common in the population affected with dermatologic disorders. Comorbidity of depression and dermatologic disorders is around 30%. The correlation between depressive and dermatologic disorders still remains unclear. In psychodermatology three disorders are described: a) psychophysiological disorders (both disorders induced and maintained by stressors), b) secondary psychiatric disorders (mental disorder as a result of skin leasions and treatment) and c) primary psychiatric disorders (skin alterations as a result of mental disorders and treatment). In depression and dermatology disorders in which certain precipitating factors are required thereby causing alteration of the patient's immunological identity causing a combination of hereditary features and ones acquired through adaptation occur to cause the disorder to develop. The cytokines are vital in the regulation of the immunology response and are also mediators of non-infective inflammatory processes leading to recurrent hormonal secretion affecting the function of the vegetative and central nervous system leading to so called „sickness behaviour", marked by loss of appetite, anhedonia, anxiety, decrease of concentration and interest along with other changes which generate a picture of depressive disorder. Treatment of depressive and dermatologic disorders is complex and requires an integral therapeutic approach encompassing all aspects of both disorders and their comorbidity. Therefore therapeutic success lies in a team approach to the patient under the auspice of consultative-liason psychiatry by setting the frame for efficient collaboration and bridging the gap between the mental and the physical in everyday clinical practice.

Key words: *depressive disorders - dermatologic disorders – comorbidity - therapeutic aspects*

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INTRODUCTION

Depressive disorders are common in the population affected with dermatologic disorders. Comorbidity of depression and dermatologic disorders is around 30%. Taking into consideration the significant genetic role in the course of inducing depression, still the most common contributing factor to frequency of this psychiatric disorder is modern life stress (World Health Organization 2001, Costa de Silva 2005, Barnow et al. 2002). Depressive disorders are often associated with various somatic disorders. Their prevalence in ambulatory (out-patients) is by far higher than in general population (Runkewitz et al. 2006, Norton et al. 2004). In dermatologic patients frequency of depression is even higher. More precise measurements, for example Mini International Neuropsychiatric Interview Questionnare (MINI), reveal that about one third of depressive patients are clinically depressed. Incidence of depression among dermatologic patients is higher than incidence among general practice patients, where it is 22% (Cohen et al. 2006, Gupta et al. 2005, Fried et al. 2005). The correlation between depressive and dermatologic disorders still remains unclear. In psychodermatology three disorders are described: a) psychophysiological disorders (both disorders induced and maintained by stressors), b) secondary psychiatric disorders (mental disorder as a result of skin lesions and treatment) and c) primary psychiatric disorders (skin alterations as a result of mental disorders and treatment) (Kotrulja & Šitum 2004, Taylor et al. 2006). Therefore, the

therapeutic approach will depend on the interrelation between depressive and dermatologic disorder. On the other hand, the pathogenetic relationship between these two disorders is more deep and complex than answers provided by the above mentioned epidemiology. Irrespective of the nature of their connection, the development of depression and dermatologic diseases in one patient is quite an unfortunate combination in which both of the disorders are aggravated by being in a circulus vitiosus and thereby mutually obstructing the healing process particularly if they are treated individually or if neglected in the therapeutic approach. Before creating an integrative therapeutic approach consideration should be given to the various therapeutic aspects associated with comorbidity and a possible pathogenetic relation between the depressive and dermatologic disorders.

SOME COMMON CHARATERISTICS AMONG DEPRESSION AND DERMATOLOGIC DISORDERS

Certain similarities among features of patients affected with depression and dermatology disorders point to their deeper pathogenetic relation than simply causal connection. At first, most depressions have a recurring course but in the later stages the course becomes chronic. Certain skin illnesses, such as psoriasis, have a similar course. The incidence of severe recurring depressive disorder in general population is around 2-5% while the incidence of psoriasis is 2–3% (Schmitt & Ford 2007). Both disorders have, not only a recurrent course with a tendency to chronicity, but similar provocative factors causing exacerbation and similar are the bimodal distribution of first episode which groups in two life stages – younger and older. In psoriasis as in depression, most accountable factors for occurence in youth are hereditary predisposing factors, and in older age precipitating (provocative) factors (Cristophers & Sterry 1993, Elder et al. 1994). In the later life stage, stress is presumed to be a significant trigger of the first episode and later exacerbations in both depression and psoriasis. Appearance of skin alterations presents additional stress for the ill person thereby closing the circle of skin disorder, stress and depression even when in question is objectively a simple but subjectively hardly acceptable dermatologic state like acne (O´Leary et al. 2004, Wittkowski et al. 2004, Choi & Koo 2003, Stone 2001). Comorbidity of depressive and psychodermatologic disorders (e.g. psoriasis) is interestingly similar. Both disorders are related to increased cardiovascular risk and other manifestations of metabolic syndrome - atherogenic dyslipidemia, abdominal obesity, raised blood pressure, insulin resistance or glucose intolerance and prothrombotic and pro-inflammatory state (Kimball et al. 2008, Christophers 2007). These disorders have one joint connection – an immunological altered state, specifically an elevation in non-infective chronic inflammatory parameters of low intensity. Increased cardiovascular risk in psoriasis can be interpreted by behaviour changes in comorbid depression (sedentary life style, smoking, alcoholism, etc.), (Watson & de Bruin 2006) however exclusion of depressive patients from the sample does not proportionally exclude cardiovascular risk. A common attribute of depression and dermatologic disorders is the inclination towards deterioration in winter and amelioration in the summer season (Posternak & Zimmerman 2002, Braun-Falco 2000). Depressive disorders have a two fold prevalence in women as compared to men. Depressive patients are considerable more often ones affected with allergy, especially pronounced in women, than in the general population. Some authors explain this through specific personality traits, while other point out the genetic background connection of depressive and allergic disorders (Goodwin et al. 2006, Buske-Kirschbaum et al. 2008, Wamboldt et al. 2000).

FROM STRESS TO DEPRESSION AND DERMATOLOGIC DISORDERS

Some individuals predisposed for depression and psoriasis, allergy or acne, will never suffer from it. Besides predisposition, in both types of disorders certain precipitating factors are required thereby causing alteration of the patient's immunological identity causing combination of hereditary features and ones acquired through adaptation lead to the development of the disorder (Holland & Vizi 2002). Immune system cells (β-cells, T-cells, macrophages, neutrophiles, eosinophiles, mastocytes and endothelial cells) communicate through cytokines which control proliferation, differentiation and function of cells and

are involved in inflammation procesess, embrional, neuronal and haematopoetic development of the organism. The cytokines are vital in regulating the immunologic response especially during infection and are also mediators in the non-infective inflammatory processes. Functioning of the immune system is controlled by superposed vegetative and hormonal mechanisms of the central nervous system leading to enhancement or weakening, acceleration or it´s deceleration. The immune system, through cytokines, causes recurrent hormonal secretion affecting the function of the vegetative and central nervous system leading to so called „sickness behaviour" marked by loss of appetite, anhedonia, anxiety, decrease of concentration and come together with other changes which generate a picture of depressive disorder (Vitkovic et al. 2000). The local immune system, for example one in the skin, are controlled by the same higher control mechanisms and their negative influences in the circumstances of chronic stress and other weakening factors (Boranić M & Sabioncello Agabrilovac 2008, Yamakawa et al. 2009). The same stressors can simultaneously or successively initiate systemic or local disturbances leading to the emergence of multiple comorbid chronic states. These states are seemingly coincidentally related or have a reactive causal relationship. Stressors (acute, recurrent or chronic) today are considered to be significant, often critical, negative precipitating factors modulating the immune system by weakening immunity. The system responds to stress throughout the central nervous system. In the acute reaction, causing elevation of catecholamine concentration through the vegetative system and in a prolonged reaction through the hypothalamic-pituitary-adrenal axis increases cortisol level. In recurring and in chronic stress states this second phase dominates (phase of hypercortisolemia) and is responsible for weakening of immunity and promotion of repressed morbidity potentials. Modern-lifestyle stressors bring about a greater manifestation of so called chronic non-infective systemic disorders in comparison to their predisposing potential (Sokal 2007). Some depressive disorders and some chronic dermatologic disorders (e.g. psoriasis) and other pathogenic related states (metabolic syndrome, cardiovascular diseases, diabetes) belong to this category of disorders.

Increasing incidence of depressive and other mentioned disorders in the last few decades are an outcome of excessive exposure to modern life stressors (World Health Organization 2001). Various authors point out the connection of chronic stress or states which implicate the experience of chronic stress (for example depression) and pathological skin lesions - especially psoriasis (Farber & Nell 1993, Koo & Lebwohl 2001, Urpe et al. 2005). This explains such a high correaltion between depression and dermatologic disorders. (Cohen 2006) Depressive individuals are often variously diagnosed from temporary or chronic skin alterations – lichen simplex chronicus, idiopathic pruritus, neurodemitis, nettle rash, atopic dermatitis, psoriasis, alopecia areata etc. (Kretzmer et al. 2008, Konuk et al. 2007, Van Moffaert 1992, Koblentzer 1983). From the other point of view, skin alterations can present a stressful event for a depressed individuals and causally worsen depression or they may even precipitate suicidal behaviour (Picardi et al. 2006).

DEPRESSIVE DERMATOLOGIC DISORDERS AND INFLAMMATORY FACTORS

Depression is a recurrent or chronic affective disorder often accompanied by physical comorbidity – vascular disorders (cardiovascular, cerebrovascular, hypertension, heart attack), diabetes, dyslipidemia, osteoporosis, inflammatory bowel disease, hypothyroidism and malignant or skin diseases (Himmerich et al. 2008). Therefore, depressed patients have an elevated risk of being burdened with a number of physical disorders. Psoriasis is a chronic inflammatory skin disease followed by the same physical comorbidity as found in depression – cardiovascular and cerebrovascular, metabolic syndrome, diabetes, osteoporosis, Chron's disease, hypothyroidism, malignant disease, anxiety and depression (Christophers 2007, Griffiths & Barker 2007). How should we explain such similar comorbidity in two so different and yet so similar comorbid states? Most recent research show that the answer may be found in the fact that depression and psoriasis, including some other chronic skin and somatic diseases, are mediated by disturbances in the immune system leading to activation of various inflammatory factors (Stewart et al. 2009).

Considering the genetic predisposition, in depression and psoriasis, immunoreactivity and maintenance of the chronic inflammatory state are critical to the pathogenesis, especially in patients of older age. These statements are validated through diverse research reports. Depressed individuals have lower indicators of the immune response (reduced mithogen – stimulated proliferation of lymphocytes and reduced activity of killer cells) and elevated systemic inflammatory indicators (elevated IL-6 and C-reactive protein) (Dentino 1999, Kiecolt-Glaser & Glaser 2002, Zorrila et al. 2001, Pike & Irwin 2006, Raison et al. 2006, Miller et al. 2009, Corcos et al. 2002). Interestingly, inflammatory diseases are frequently related to depressed mood, particularly with so called „sickness behaviour". The „Sickness behaviour" is a homeostatic reaction of the organism in to the prevailing inflammation, but in individuals with predisposition to depression it has the proportions of depressive disorder. This process is initiated through pro-inflammatory cytokines (IL-1, IL-6, TNF-α) (Konsman et al. 2002). Wright and associates found how administration of Salmonella typhi vaccine in volunteers leads to significant decrease of mood after 1.5 to 3 hours followed by elevation of interleukine-6 in blood (Wright et al. 2005). The cytokine (inflammatory, macrophage) theory of depression presumes that pro-inflammatory cytokines have a key mediation role in the genesis of depression symptoms. It is based upon findings indicating that the cause of the serotonin deficit lies in pro-inflammatory cytokines by activating neural serotonin transporter. Antidepressants, (SSRI) by deactivating this transporter lead to mood improvement. Additionally, TNF-α stimulates reuptake of serotonin in neural endings causing the effect of depression. (Zhu 2006) Furthermore, immunologic activation with elevated production of pro-inflammatory cytokines activates indolamine-2,3-dioksigenase – an enzyme which disintegrates tryptophan and serotonin thus decreasing availability of serotonin in the central nervous system causing depression (Müller & Schwarz 2007). In comorbid disorders (cardiovascular, atherosclerosis, diabetes, dyslipidemia) accompanying depression elevated levels of pro-inflammatory cytokines are found. However, it is reasonable to assume involvement of these cytokines in pathophysiological processes in the development of these disorders (Broadley et al. 2002). The same are repeatedly found in depressed patients without any comorbidity (Haack et al. 1999, Maes 1999, Dunn 2005). Some research draws attention to the bidirectional nature in the relation of depression and somatic disorders through the mediation of pro-inflammatory cytokines. For example, lesions of coronary artery endothelium resulting in development of coronary heart disease may be the result of decreased serotonin transport activated by pro-inflammatory cytokines and leading to inflammatory changes (Lett et al. 2004). Similar changes of pro-inflammatory cytokines are found in some chronic skin lesions. In chronic systemic inflammatory disease, such as psoriasis, elevated levels of IL-1, IL-6 and TNF-α exist regardless of the comorbid mentioned states. Based on these findings, considerable shifts are to be expected in the treatment of psoriasis with anti-cytokine therapeutic means, especially TNF antagonists (Pietrzak et al. 2008, Schmid-Ott et al. 2009). From the afore mentioned statements it follows that the immunological and inflammatory changes are implemented in the pathophysiology of depression and psoriasis and also in the pathophysiology of numerous chronic skin diseases. Regardless of comorbidity, the presence of inflammatory changes in both disorders confirms the importance of inflammatory processes as vital in the partial development of depressive and chronic skin disorders indicating the mutual comorbidity of these disorders, and thus can worsen prognosis (Himmerich et al. 2008, Kourosh et al. 2008).

THERAPEUTIC ASPECTS OF COMORBIDITY IN DEPRESSIVE AND DERMATOLOGIC DISORDERS

The pharmacotherapeutic approach in comorbidity of dermatologic disorders and depression should be based upon recent pathogenetic understandings of the stress response system involvement and pro-inflammatory mediators in the immunologic response (IL-1, IL-6, TNF-α) of both disorders (Himmerich 2006, Katsambas & Stratigos 2001). Moreover, some research results show, that certain antidepressants (SSRI) have an immunomodulatory, (anti-inflammatory effect) associated with elevation of catecholamines in the brain thereby ensuring their antidepressant effects (Szelényi & Selmeczy 2002, Keshavan 2001). This means that the, usage of antidepressants will not

improve only depressive mood but also have a pharmacologic effect, for example on psoriasis, acne, egzema, hyperhidrosis, rosacea and pruritus of different forms. Antidepressants alleviate adverse effects of pro-inflammatory cytokines on central neurotransmission by reducing their release from activated macrophages thereby facilitating inhibition of the hypothalamic-pituitary-adrenal axis leading to reduction of suprarenal glucocorticoid release. Many antidepressants induce release of endogenous antagonists of pro-inflammatory cytokines (interleucin-1 receptor antagonist, interleucin-10) and some classes of antidepressants act as cycloxygenase inhibitors. The cyclooxygenase inhibitors reduce the concentration of inflammatory prostaglandins in the brain thus moderating the destructive effect of inflammatory changes in neurotransmitter systems. This macrophage theory expands the biogenic-amine hypothesis of depression by including changes in the endocrine and immunologic system involving the importance of stress as one of the alternating factors (Leonard 2001). Presumably there are other disorders within the metabolic syndrome on a subclinical level which might be targets for a therapeutic approach, therefore the treatment of patients with at least two comorbid states and often increased cardiovascular risk, requires skill in the therapeutic adjustment to the overall health condition. It has to be individually adjusted and based on new therapeutic strategies aiming at reducing activity of pro-inflammatory cytokines (Maubach et al. 1999, Dinarello 2004). In dealing with comorbidity of depressive and dermatological disorders physicians will often be faced with „off-label“ psychopharmacotherapy. This does not imply illegal or unethical usage of these drugs especially if it is endorsed by scientific medical evidence or based upon good clinical practice. Such a statement should simply be accepted as there is no sufficiently large interest of pharmaceutic industry for registration of these medications in this indication area (Leonard 2001).

THERAPY OF PRIMARY DEPRESSIVE DISORDERS IN COMORBIDITY WITH SECONDARY DERMATOLOGICAL CHANGES

Primary depressive disorders in comorbidity with dermatological disorders are often detected in dermatological outpatient clinics where patients first seek aid. Confirmation is made by the liason psychiatrist. In treatment of such comorbidity, there is often sufficient treatment of the primary disorder with adequate antidepressant which will also palliate dermatologic disturbances. Recent findings on the biological basis of depression (involvement of the stress response system and of non-infective inflammations of low intensity) evoke hope that new biological medications (pro-inflammatory cytokine antagonists) will significantly enhance treatment of these disorders, especially ones resistant to former treatments.

Secondary dermatologic disorders in depressed patients are pruritus and psychogenic excoriations. Pruritus is associated with many dermatological states and internal system diseases. It is often present in anxiety and depressive disorders. The cause of pruritus extends in a wide range from predominantly psychogenic to somatogenic. Most effective in eliminating this condition is considered to be doxepine, a tricyclic antidepressant with strong antihistaminic activity. Anti-pruritic function is based upon intense peripheral H1 receptor antagonism, more pronounced than in many registered antihistaminics. For this reason it is given in treatment of pruritus refractory to traditional antihistaminics (Koo & Ng 2002).

Psychogenic excoriations are often seen in anxious and depressive disorders (major depressive disorder, general anxiety disorder, obsessive-compulsive disorder, PTSD). There are three main elements involved in the therapeutic approach of depressive patients with psychogenic excoriations: optimization of the patient’s mental state in order to gain a better perspective and healing in combination with psychotherapeutic and pharmacotherapeutic treatment. In today´s therapy of psychogenic excoriations, selective serotonin reuptake inhibitors (SSRI´s), particularly fluoxetine, sertraline and paroxetine (Koo & Ng 2002) are especially effective.

THERAPY OF SECONDARY DEPRESSIVE DISORDERS IN COMORBIDITY WITH DERMATOLOGICAL DISORDERS

Secondary depressive disorders present as the patient’s emotional response to a dermatological condition or are the consequence of treating one. Particularly, such responses are often seen in cystic acne in young people, ichtiosis, psoriasis and

vitiligo. Symptoms in form of feeling worthless, hopeless and suffering overwhelming guilt are also frequent. The patients often report insomnias, concentration disturbances, loss appetite, body weight and energy. Their significant preoccupation with skin lesions leads to substantial distress with social and working dysfunction. The existence of dermatological disorder causes significant emotional suffering. Development of these depressive states is known in patients with vitiligo, alopecia areata or other skin conditions causing discomfort in social communication. Due to equal efficiency in treating light and moderate depression, selective serotonin reuptake inhibitors and tianeptine are the first choice therapy because of a favorable cardiovascular side-effects profile in comparison to tricyclic antidepressants and certain new antidepressants. Selective serotonin reuptake inhibitors are recommended as first line therapy but antidepressants with atypical or dual action mechanism can also be useful (Koo & Ng 2002, Kasper & Oliė 2002, Lee & Koo 2003).

A the same time it is important to point out negative possible side-effects when combining SSRI´s and acetylsalicylic acid, a frequently used drug in cardiovascular patients as protection from cardiovascular incidents, such as gastrointestinal bleeding (Dhondt et al. 2002, Katsambas & Papakonstantinou 2004).

TREATMENT OF DEPRESSIVE DISORDERS IN COMORBIDITY WITH PSYCHOPHYSIOLOGICAL DERMATOLOGICAL DISORDERS

The treatment of comorbidity of psychophysiological dermatological and depressive disorders is particulary demanding and requires a thorough approach based upon principles of consultative-liason psychiatry. The psychophysiological disorders are mostly manifested as chronic dermatosis precipitated or exacerbated by stress. These are psoriasis, atopical dermatitis, acne, rosacea, seborrhoic dermatitis, nettle rash, etc. When a disorder develops it recurrently affect the emotional state and thereafter depression is a frequent accompanying disorder of the mentioned states. According to the macrophage theory, depressive and psychophysiological disorders have the same precipitating and pathophysiological grounds so therapy should utilize effects of pro-inflammatory cytokine antagonists (example – Etanercept – TNF-α antagonist) (Kenreigh 2006). The pro-inflammatory cytokine antagonists should, besides having an anti-inflammatory effect, have an antidepressive mechanism and antidepressants should have an anti-inflammatory mechanism while treating a patient with comorbid psychophysiological dermatologic disorder and depression. The next step in the evolution of new treatment strategies in such comorbid states may be through combining these agents in the therapy of depressive disorders without comorbid dermatosis. These combinations could result in sinergistic effect resolving many cases of treatment resistant depressions due to elevated rates of pro-inflammatory cytokines in depressed patients without comorbidity (Haack et al. 1999, Maes 1999). Some reports confirm clinical efficacy of TNF-α antagonists in the treatment of affective disorders (Soczynska et al. 2009). Among other biological treatment methods, light therapy has proved to be equally useful in therapy of seasonal (winter) depression and in treatment of some skin diseases (psoriasis, vitiligo, neurodermitis) (Eastman et al. 2006, Kaliterna 2006).

IATROGENIC DEPRESSIVE STATES

Simultaneously, patients with depression, psoriasis and other comorbid states are often treated with various drugs carrying a potential depressive effect. In patients burdened with skin disorders iatrogenic depression is not rare. The clinical presentation consists of lethargy, apathy, fatigue, dullness. Sometimes such conditions are called pseudodepression. Medications induced iatrogenic depression belongs to several therapeutic groups: antihypertensives (reserpin, beta – blocators, alfa-methyldopa), antiarrithmycs (digoxin), hormonal preparations (corticosteroides, oral contraceptives), chemotherapeutics (methotrexate, decarbazine, vinblastine, procarbazine, interferone), antiepyleptics (phenytoin, carbamazepine, phenobrabitone), cholesterol decerasing drugs (pravastatin, lovastatin, cholestyramin), antibiotics (cycloserin, dapsone, amphotercine B), histamine H2 receptor blockers (cimetidine, famotidine, raniditine), antipsychotics (phenotiazines, butirophenones, tyoksantens) (Walker & Katona 1997). A depressogenic effect is also assigned to isotretionine – a medication for acne (Lipper 2005). When encountering a patient with psychological problems such as like iatrogenic depression thorough anamnesis should be taken followed by

physical and neurological examination. If the suspicion is confirmed, either elimination of the suspected drug or addition of an antidepressant is possible depending on the relevance of the drug for treating the primary illness. Iatrogenic depressions are, because of multiple somatic disorders, frequently occur in older age, and therefore when a depression syndrome appears it is advisable to consider this possibility (Livingston et al. 2000).

DERMATOLOGICAL SIDE EFFECTS OF MOST FREQUENTLY USED ANTIDEPRESSANTS

Selective serotonine reuptake inhibitors are most often used in therapy of depressive disorders. Although they do not induce serious side-effects as do tricyclic antidepressants or monoaminooxidase inhibitors, skin side-effects are not rare particularly if combined with non-steroid antirheumatics or aspirin used in prevention of cardiovascular disorders. Skin alterations are manifested as: petechiae, echimosis, acneiformn eruptions, leucocytoclastic vasculitis, nettle rash, erythema nodosum, erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis, psoriatic eruptions etc. Reversible serotonin deficiency in trombocytes leading to disorders in blood coagulation is supposed to be the mechanism causing dermatologic disorders. Alopecia, pruritus, dyschromia, hirsurtism, hypertrichosis, photoallergic and phototoxic reaction can occur caused by SSRIs (Krasowska et al. 2007).

PSYCHOTHERAPEUTIC TREATMENT

In the combined treatment of coexisting dermatologic conditions and depression, the psychotherapeutic approach is based on recognizing important psychological factors in the occurence and maintainance of both disorders with a hypothesis of how mood improvement leads to improvement of dermatological disorder (Van Moffaert 1992, Koblentzer 1983, Fried 2002, Vedhara et al. 2007). Psychotherapeutic interventions applicable in psychodermatological practice are: stress management techniques (biofeedback, relaxation training, meditation), cognitive-behavioural therapy, hypnosis, emotional ventilating, individual psychotherapy, psychoeducation and psycho-therapeutic support (Vedhara et al. 2007, Gregurek et al. 2006).

INTEGRATIVE THERAPEUTIC APPROACH IN TREATMENT OF COMORBID DEPRESSIVE AND DERMATOLOGIC DISORDERS

Treatment of depressive and dermatologic disorders is complex and requires an integral therapeutic approach encompassing all aspects of both disorders and their comorbidity. Regardless of the nature of their relationship, the occurrence of skin disorder and depression in an individual requires an integrative approach, because unrecognized concomitant appearance of both disorders can worsen and obstruct the treatment. Therefore, unilateral treatment approaches to these disorders are in advance unsuccessful. The solution lies in a team approach by the psychiatrist, dermatologist and other experts under the auspices of consultative-liason psychiatry setting the frame for efficient collaboration. This interdisciplinary approach should include all risk groups where occurrence of the disorders is frequent (Gregurek et al. 2006, Boguniewicz et al. 2008).

CONCLUSION

Comorbidity of depressive and dermatological disorders is around 30%. Knowledge of the stress response system and of immunological system involvement in disorder pathogenesis enforces development of specific treatment strategies. Complex pathophysiological correlation of these two disorders imposes the necessity for an integral interdisciplinary therapeutic approach. Beside the psychiatrist and the dermatologist, the therapeutic team should include other experts whose collaboration could help in developing treatment of comorbid disorders. The framework for interdisciplinary cooperation lies in consultative-liason psychiatry bridging the gap between mental and physical in everyday clinical practice.

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Correspondence:

Prof. Pavo Filaković, PhD, MD
School of Medicine Osijek
J. Huttlera 4, 31000 Osijek, Croatia
E-mail: filakovic.pavo@kbo.hr pavo.filakovic@mefos.hr