Patients with chronic renal failure (CRF) may exhibit various cutaneous abnormalities, including changes in skin colour, pruritus, xerosis, hair, nail and oral changes, metastatic calcinosis, and bullous dermatosis. These changes have a considerable negative effect on the patient’s quality of life. Early recognition of cutaneous signs and prompt initiation of treatment can dramatically alter their course and decrease morbidity.
The cytochrome P450 superfamily (CYP450) in humans is formed by 57 functional monooxygenases critical for the metabolism of numerous endogenous and exogenous compounds. The superfamily is organized into 18 families and 44 subfamilies. CYP nomenclature is based on the identity of amino acids. The most important functions of the CYP450 are related to metabolism of endogenous compounds, detoxification of exogenous xenobiotics and decomposition of the vast majority of currently used drugs. The expression of CYP450 enzymes in the human body is characterized by a marked substrate and tissue specificity, the most important being localized in the liver, but also present in kidney, lung, brain, breast, prostate and in the small intestine. The human cytochrome P450 3A gene family (CYP3A) accounts for the largest portion of CYP450 proteins in human liver and includes 4 genes: CYP3A4, CYP3A5, CYP3A7, CYP3A43. Multiple and complex genetic variations, marked interindividual, interethnic and gender variability have been reported regarding CYP3A isoform expression and activity. Multiple factors may affect CYP3A expression and activity, such as inducers like rifampicin, phenobarbital, 3-methylcholantrene, beta-naphthoflavone, and dexamethasone. The maturation of organ systems, paralleled by ontogeny of drug-metabolizing enzymes during fetal life and in the first months of postnatal life, surely exerts profound effects on drug disposition, probably being the predominant factor accounting for age-associated changes in drug clearance. In fact, drug dosage in the perinatal period represents a continuous challenge for neonatologists. The purpose of this article is to provide a brief review of the pharmacokinetic differences between neonates and adults, showing the peculiarities of liver CYP450-related drug metabolism in the perinatal period and at birth, and to report the toxic mechanisms of liver injury in neonates, due to the most frequently utilized drugs in NICU centers.
Neuropeptides are involved in neurogenic inflammation where there is vasodilation and plasma protein extravasation in response to this stimulus. Nerve growth factor (NGF), identified by Rita Levi Montalcini, is a neurotrophin family compound which is important for survival of nociceptive neurons during their development. Therefore, NGF is an important neuropeptide which mediates the development and functions of the central and peripheral nervous system. It also exerts its proinflammatory action, not only on mast cells but also in B and T cells, neutrophils and eosinophils. Human mast cells can be activated by neuropeptides to release potent mediators of inflammation, and they are found throughout the body, especially near blood vessels, epithelial tissue and nerves. Mast cells generate and release NGF after degranulation and they are involved in iperalgesia, neuroimmune interactions and tissue inflammation. NGF is also a potent degranulation factor for mast cells in vitro and in vivo, promoting differentiation and maturation of these cells and their precursor, acting as a co-factor with interleukin-3. In conclusion, these studies are focused on cross-talk between neuropeptide NGF and inflammatory mast cells.
MANAGEMENT OF NON-MELANOMA SKIN CANCER IN SOLID ORGAN TRANSPLANT RECIPIENTS

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Non-melanoma skin cancer (NMSC) is the most frequent cancer observed in solid organ transplant recipients (SOTR). Early diagnosis, patient education, and modification of immunosuppression are effective measures for reduction of NMSC incidence. Many risk factors have been identified, including age at transplantation, fair skin, type of immunosuppressive drugs, cumulative sun exposure, viral infections, and various genetic markers. Skin self-examination and photoprotection should be encouraged in all transplanted patients. Long-term skin surveillance, early diagnosis and aggressive treatment of any suspicious lesion, reduction of immunosuppressive therapy, and conversion to mammalian target-of-rapamycin (m-TOR) inhibitors can be also effective measures for reduction of NMSC incidence.
Sevelamer hydrochloride is an ionic exchange resin with high affinity for phosphate. This phosphate-binding agent has few serious adverse reactions with the advantage of reducing total and low density lipoprotein (LDL) cholesterol levels. However, it is controversial as to whether sevelamer hydrochloride can modulate the inflammatory response via endotoxin reduction. Therefore, a single-center, open-label, prospective and randomized study was performed to compare the clinical efficacy, safety and anti-inflammatory activity of two sevelamer hydrochloride tablet forms – a branded tablet form, Renagel® (Genzyme manufacturer) and its generic equivalent (EMS manufacturer). Twenty-eight chronic kidney disease volunteer patients at stage 5 (CDK 5D), on chronic low-flux hemodialysis carried out in 4-hour sessions, three times a week, were studied. The serum phosphorus, ionic calcium, total cholesterol and fractions, bicarbonate, blood pH, interleukin (IL)-6, IL-10, IL-1 beta and tumor necrosis factor-alpha (TNF-α) levels were collected prior to dialysis at mid-week. The incidence of gastrointestinal adverse effects were determined at the end of the phosphate-binder washout period as well as at the end of the fourth and eighth weeks of use of both tablet forms. The same magnitude of reduction in serum phosphorus was observed with both sevelamer tablet forms. Only the Renagel® group showed lower total cholesterol and lower LDL cholesterol levels at the fourth and eighth week versus baseline. No significant differences in serum cytokine levels were identified in either drug group. However, the incidence of intestinal obstipation was higher among patients who used the generic equivalent form. In conclusion, Renagel® and its EMS generic equivalent tablet forms have a similar clinical efficacy in reducing phosphorus in CKD 5D patients on low-flux hemodialysis and a similar safety profile.
ROCK ACTIVATION IN LUNG OF IDIOPATHIC PULMONARY FIBROSIS WITH OXIDATIVE STRESS

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The Rho-associated coiled-coil containing protein kinase, (Rho-kinase or ROCK) undergoes activation by oxidative stress. ROCK-II, which is an isoform of ROCK, is activated in a murine model of lung fibrosis. The present study evaluated the level of oxidative stress and activation of ROCK-II in patients with idiopathic pulmonary fibrosis (IPF). The ROCK-II level and the phosphorylation of myosin phosphatase subunit-1 (p-MYPT-1), a hallmark of ROCK activation, were examined by immunohistochemistry of lung tissue sections. The 8-iso prostaglandin-F2α (8-isoPGF2α) level, as a marker of oxidative stress, of exhaled breath condensate was significantly higher in IPF patients than in control patients. In IPF lungs, ROCK-II was predominantly expressed by bronchial epithelial cells, as well as at a lower level by airway smooth muscle cells, vascular smooth muscle cells, and the fibroblasts of fibroblastic foci (FF). In addition, there was moderate p-MYPT-1 expression in these cells of IPF lungs. In control lungs, ROCK-II was expressed by these cells. p-MYPT-1 was weakly expressed by the bronchial epithelial cells. In conclusion, ROCK-II was activated in various lung cells of IPF patients along with oxidative stress detected by 8-isoPGF2α elevation. The ROCK pathway may play a role in the development of IPF via oxidative stress.
Breast cancer is estimated to be the most common malignancy affecting women in Iraq. The cancer antigen CA 15-3 has been used as a possible serum marker of occult and recurrent breast carcinoma, either alone or in combination with other tumor markers such as HER2/neu, that has evolved as a major classifier of invasive breast cancer and target of therapy for the disease. ELISA, used to evaluate serum levels of CA15-3 and immuno-histochemistry staining technique, was used to establish the HER2/neu status. The results of this study indicate an increased level of CA15-3 in breast cancer patients (29.02±1.79 IU/ml) as compared to both women with benign tumor and healthy controls (13.78±1.24 and 8.92±0.48 IU/ml, respectively), and that this increase is associated to advanced stages. Patients with HER2/neu positive malignancies show elevated serum CA15-3 (37.09±2.55 IU/ml), as well as patients who developed recurrence (40.75±2.11 IU/ml). Our data study suggests that higher levels of CA 15-3 would be a reliable prognostic marker as they were directly related to advanced stages and recurrence. In addition persistent elevation of CA 15-3 was associated to HER2/neu positivity in breast cancer patients.
Class IA phosphatidyl inositol-3 kinases (PI3-K) are important targets in cancer therapy and are essential to immune responses, particularly through costimulation by CD28 and ICOS. Thus, small PI3-K inhibitors are likely candidates to immune intervention. PIK-75 is an efficient inhibitor of the PI3-K p110α catalytic subunits that suppresses tumor growth, and its effects on immune and autoimmune responses should be studied. Here, we describe the effect of PIK-75 on different immune parameters \textit{in vitro} and \textit{in vivo}. PIK-75 at concentrations commonly used \textit{in vitro} (≥0.1 μM) inhibited T and B cell activation by Concanavalin A and LPS, respectively, and survival of non-stimulated spleen cells. In naive CD4\(^+\) T lymphocytes, PIK-75 induced apoptosis of resting or activated cells that was prevented by caspase inhibitors. At low nanomolar concentrations (≤10 nM), PIK-75 inhibited naive CD4\(^+\) T cell proliferation, and IL-2 and IFN-γ production induced by anti-CD3 plus anti-CD28. In activated CD4\(^+\) T blasts costimulated by ICOS, PIK-75 (≤10 nM) inhibited IFN-γ, IL-17A, or IL-21 secretion. Furthermore, PIK-75 (20 mg/kg p.o.) suppressed clinical symptoms in ongoing experimental autoimmune encephalomyelitis (EAE) and inhibited MOG-specific responses \textit{in vitro}. Thus, PIK-75 is an efficient suppressor of EAE, modulating lymphocyte function and survival.
IMMUNOMODULATORY AND PROTECTIVE PROPERTIES OF TACROLIMUS IN EXPERIMENTAL SCORPION ENVENOMATION

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Involvement of imbalance between pro- and anti-inflammatory events has been reported in the developed pathogenesis after scorpion envenomation. The immunosuppressive and anti-inflammatory properties of tacrolimus (FK-506) have been investigated: i) to better understand evolution of signaling pathways which are involved in the immune system ii) to reduce observed clinical signs while keeping a balance between pro- and anti-inflammatory cytokines. Naval Medical Research Institute (NMRI) mice received tacrolimus (1 mg/kg every 12 hours per os) for 21 days before envenomation with a sublethal dose (10 µg/20 g body weight) of Androctonus australis hector venom (Aah). Cell migration, pulmonary edema, exudation, Myeloperoxydase (MPO), Eosinophil peroxidase (EPO), C-reactive protein (CRP), C3, Creatine phosphokinase (CPK), aspartate aminotransferase (ASAT), alanine aminotransferase (ALAT), and hyperglycemia were analyzed 30 min, 3 and 24 hours after injection of Aah venom. Histological analysis of lung parenchyma was undertaken 24 hours after envenomation. Aah lethality was evaluated on mice with or without pretreatment with tacrolimus. (Fab')2 fragments (40 mg/kg) were also used as specific treatment in all protocols. Tacrolimus significantly inhibited cell migration, pulmonary edema, exudation, CRP and hyperglycemia. It also decreased MPO and EPO activities and prevented tissue damage in lung tissue, balancing seric parameter levels (CPK, ASAT and ALAT). The pretreated animals seemed to be protected by this macrolide against the venom lethality. These findings suggest that the overactivation of the immune system is one of the causes involved in the aggravation of the pathophysiological effects induced after envenomation. The obtained results showed that the use of F(ab')2 fragments as specific treatment cannot reduce the induced inflammatory response.
Angiotensin II type 2 receptors are believed to counter the effects of the angiotensin type 1 receptors and there is no data relating to the co-localisation of either receptor in human diseased arteries. We sought to determine whether AT₂R counter the effects of AT₁R and immunolocalise both receptors to cells in human diseased arteries. Human radial arteries (RA, n=11) were placed in organ bath chambers and preincubated with the AT₂R antagonist PD123319 for twenty minutes before an angiotensin II dose response curve. Immunohistochemistry was performed to identify receptors and pathology was quantified by image analysis software. We observed both receptors in human arteries. Angiogenic blood vessels within occluded arteries expressed both receptors. PD123319 impaired angiotensin II mediated vasoconstriction by 20% (n=5, p<0.05), however in other arteries, PD123319 exacerbated angiotensin II-mediated vasoconstriction by 60% (n=6, p<0.01), respectively. We conclude that inhibition of AT₂R can enhance or reduce angiotensin II-mediated vasoconstriction. These data indicate that the role of AT₂R in human diseased arteries is divergent although the AT₂R-mediated vasorelaxation prevails.
Occult hepatitis C virus (HCV) infection is a new entity that should be considered when diagnosing patients with abnormal liver functions of unknown origin. This work was carried out to evaluate T-helper 1/T-helper 2 (Th1/Th2) cytokine profiles in patients with occult HCV infection versus chronic hepatitis C (CHC) infection, also to investigate any association between these cytokines and liver histological features in both groups. Serum levels of Th1 cytokines (IL-2, IFN-γ) and Th2 (IL-4 and IL-10) were measured in 35 patients with occult HCV infection compared to 50 patients with chronic hepatitis C infection and 30 healthy controls. We have found that Th1 cytokines were significantly increased in patients with CHC infection than in both occult HCV infection and control groups (p<0.001). On the other hand, serum IL-4 levels were higher in occult HCV infection than in CHC and control groups (p<0.001). Furthermore, serum IL-10 levels were higher in both patient groups vs control group (p<0.001), with no significant difference between CHC and occult HCV groups. Finally, only serum IL-10 levels were significantly higher among patients with high activity (A2-A3) than those with low activity (A0-A1) in both CHC and occult HCV groups (p=0.038, p=0.025, respectively). Patients with occult HCV infection exhibited a distinct immunoregulatory cytokine pattern that is shifted towards the Th2 arm.
Osteopontin (OPN) is an extracellular matrix protein implicated in bone remodeling, but it presents also pro-inflammatory and pro-fibrotic properties. OPN expression also occurs upon exposure of cells to classical mediators of acute inflammation such as tumor necrosis growth factor alpha (TNF-α) and interleukin-1 beta (IL-1β), as well as fibrogenic cytokines such as transforming growth factor beta (TGF-β), although a detailed understanding of these regulatory pathways is still unknown. Plasma OPN levels in both limited and diffuse systemic sclerosis patients (lSSc and dSSc) were statistically higher compared to those of control subjects. Immunohistology demonstrated that high TGF-β levels, alpha smooth muscle actin (αSMA) levels and consequently high OPN levels were found in the affected skin of sclerodermic patients (lSSc and dSSc) compared to levels found in healthy skin. In order to better understand how OPN interferes with the fibrotic process, healthy skin fibroblasts were treated for 24 and 48 hours with bleomycin and with endothelin-1 (ET-1) plus TGF-β in order to induce the fibrogenesis. After 48 hours of stimulation, healthy treated fibroblasts showed statistically increased αSMA levels (index of differentiation into myofibroblasts) and simultaneously statistically increased OPN levels compared to healthy untreated ones. This study demonstrates that OPN levels increase simultaneously with the increasing of αSMA levels, therefore it is reasonable to hypothesize that OPN interferes in the pathogenesis of Systemic Sclerosis in the early stage of fibroblast differentiation process.
LETTER TO THE EDITOR

MULTIPLE CYTOKINE-PRODUCING TESTICULAR MALIGNANT LYMPHOMA WITH CLINICAL SYMPTOMS RESEMBLING INFECTIOUS SIGNS

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We present the case of a 64-year-old male with painful swelling of the bilateral testes and epididymides, high fever, leukocytosis, and an elevated C-reactive protein (CRP) level. This is the first case report of testicular diffuse large B-cell lymphoma, not otherwise specified (DLBCL, NOS) immunostained for multiple cytokines and their receptors, which clearly demonstrates that tumor cells express multiple cytokines [interleukin-6 (IL-6) and granulocyte colony-stimulating factor (G-CSF)] and their receptors [IL-6 receptor (IL-6R) and G-CSF receptor (G-CSFR)]. The clinical course showed that the reduction in tumor size was accompanied by a corresponding improvement in clinical symptoms and peripheral blood findings. Such clinical investigation may lead clinicians to misdiagnose inflammatory disease rather than neoplastic disease. Recognizing this paraneoplastic phenomenon associated with some cases of testicular DLBCL, NOS is important. In addition, this case suggests that the growth of tumor cells may be promoted through autocrine mechanisms of IL-6 and G-CSF, which are produced by tumor cells. The possibility that these cytokines can be produced by tumor cells and can accelerate tumor proliferation should be considered to be a cause of severe clinical symptoms, an aggressive clinical course, and an indication of the necessity of treatment. Certain cytokines may be used as tumor markers in some cases of DLBCL, NOS.
Hymenoptera venom anaphylaxis after bee or wasp sting is a common problem that affects about 1.2% to 3.5% of the general population. Venom-specific immunotherapy (VIT) is an established mode of treatment for immunoglobulin (Ig) E–mediated Hymenoptera venom allergy. However, VIT may often be associated with immediate anaphylaxis which can lead to treatment withdrawal. Several cases published in recent years suggest that omalizumab, used as add-on therapy may be able to prevent anaphylaxis during VIT. We report the case of a 30-year-old woman, suffering from mild persistent asthma, who had a history of severe anaphylactic reactions after yellow jacket sting, and after eating peanuts, contact with guinea pig hair, and i.v. administration of dexamethasone natrium phosphate. Initial specific immunotherapy had to be stopped due to severe anaphylaxis (hypotension, dyspnea, and angioedema). The immunotherapy was reintroduced accompanied by the anti-immunoglobulin (Ig) E monoclonal antibody omalizumab. Subcutaneous omalizumab 150 mg was initiated 4 weeks after the anaphylaxis incident and 1 day before the resumption of VIT. Rush treatment was uneventful, and the usual cumulative dose of 111.1 μg was successfully reached. The combination of omalizumab and VIT is a valid option of therapy for these patients and could reduce asthma and food allergy symptoms.
Infection with HIV may lead to the development of cardiomyopathy as improved antiretroviral regimens continue to prolong patient life. However, advanced therapeutic options, such as heart transplant, have until recently been precluded to HIV-positive persons. A favorable long-term outcome has been obtained after kidney or liver transplant in HIV-positive recipients fulfilling strict virological and clinical criteria. We recently reported the first heart transplant in a HIV-infected patient carried out in our center. In this article, we detail the major challenges we faced with the management of antiretroviral and immunosuppressive treatments over the first 3 years post-transplant. The patient had developed dilated cardiomyopathy while on antiretroviral treatment with zidovudine, lamivudine and efavirenz. He was in WHO Stage 1 of HIV infection and had normal CD4+ count and persistently undetectable HIV-RNA. In spite of cardiac resynchronization therapy and maximal drug therapy, the patient progressed to end stage heart failure, requiring heart transplant. He was placed on a standard immune suppressive protocol including cyclosporine A and everolimus. Despite its potential pharmacokinetic interaction with efavirenz, everolimus was chosen to reduce the long-term risk of opportunistic neoplasia. Plasma levels of both drugs were monitored and remained within the target range, although high doses of everolimus were needed. There were no infectious, neoplastic or metabolic complications during a 3-year follow-up. In summary, our experience supports previous data showing that cardiac transplantation should not be denied to carefully selected HIV patients. Careful management of drug interactions and adverse events is mandatory.
Allergic reactions associated to the use of macrolides are uncommon; in particular only two cases of anaphylaxis with erithromycin and clarithromycin have been reported to date. The aim of this study was to investigate macrolide-induced anaphylaxis. Between December 2007 and December 2011, 136 consecutive children were referred to the Allergy Unit of A. Meyer Children’s Hospital because of a past history of reactions to macrolides. Allergy work-ups were carried out according to the European Network for Drug Allergy protocol. Anaphylaxis was diagnosed according to the clinical criteria proposed by Sampson et al. and graded according to Brown SGA et al. Sixty-six out of 136 patients completed the allergologic work-up and among them we investigated three cases of anaphylaxis due to azithromycin which included one child with anaphylaxis to both clarithromycin and azithromycin. In two of the children with anaphylaxis, the diagnosis was only confirmed with the skin prick test, the third was positive to the Intradermal Test. The azithromycin allergy shows a surprisingly high sensitivity to the in-vivo tests. Moreover, this study shows that cross-reactivity may occur between different macrolidic molecules; it has even been suggested that macrolide allergies are unlikely to be class allergies.
LETTER TO THE EDITOR

POMPHOLYX OF THE HANDS AFTER INTRAVENOUS IMMUNOGLOBULIN THERAPY FOR CLINICALLY ISOLATED SYNDROME: A PAEDIATRIC CASE

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Pompholyx is a common eruption of small vesicles on the palms, soles, and/or lateral aspects of the fingers. It has a multifactorial etiology, including genetic determinants, allergy to metals, and id reaction; rarely it is a drug-related side effect. We report a paediatric case of pompholyx of the hands related to the intravenous immunoglobulin (IVIG) therapy for Clinically Isolated Syndrome (CIS). A 10-year-old boy, received an IVIG therapy (Venital®, Kedrion Spa, Italy) at a dose of 400 mg/kg daily for five days. The fifth day of IVIG infusion, a symmetrical vesicular eruption appeared on the palms of the hands and on lateral aspects of the fingers. The lesions improved with application of topical steroids in few days. The mechanism of induction of pompholyx by IVIG therapy is unknown. A review of the Literature suggests the hypothesis that dyshidrotic eczematous reactions may be related not only to the type of IVIG, to the dose and the rates of infusion, but also to an allergic response to excipients and preservatives contained in the drug, probably elicited by an underlying neurological disease in some cases.
The prevalence of nickel hyper-sensitivity varies widely in different countries, nevertheless it is the leading cause of contact dermatitis. The presence of nickel in the diet (mainly plant foods) in some nickel-sensitive subjects can provoke/aggravate eczema and systemic contact dermatitis as well as cause extra-cutaneous symptoms (respiratory, gastrointestinal, neurological). These symptoms, correlated to the ingestion of nickel-containing foods and beverages, in nickel patch test positive individuals, defines the so called “systemic nickel allergy syndrome (SNAS)”, a condition successfully treated by oral desensitization. Although numerous studies have investigated the prevalence of contact nickel allergy or addressed the relationship between nickel intake and onset of systemic symptoms, to our knowledge no epidemiological studies have attempted to estimate the prevalence of SNAS. Therefore, we decided to evaluate consecutive patients (1,696), afferent to four allergy units in Sicily, a region of southern Italy, from October 2010 to March 2011. SNAS was confirmed in 98 patients (5.78%) of the 1,696 studied, suggesting that this clinical entity may be an emergent allergological condition rather than an occasional finding. The most common symptoms complained of in our population were cutaneous (51 patients), gastrointestinal (87 patients) and other systemic clinical manifestations (37 patients). Furthermore, 16 out of the 98 SNAS patients (16.3%) presented IgE-mediated food allergy with a statistically significant association ($\chi^2=16.950; P<0.0001$), therefore suggesting underlying cross-facilitating pathways. These findings need confirmation on wider populations but may help allergists to suspect, during common clinical practice, that cutaneous and extra-cutaneous symptoms may be referred to nickel intake and deserve specific in-depth investigation.
LETTER TO THE EDITOR

THE GROWTH OF STREPTOCOCCUS MUTANS IN DIFFERENT MILKS FOR INFANT FEEDING

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After birth various bacterial species, mainly streptococci, colonize the oral cavity and are frequently isolated from carious lesions in children. Previous studies suggest that an earlier colonization of a child’s mouth by cariogenic organisms might be related to a higher risk of caries. The aim of this study is to assess the influence of different milks for infant feeding on the growth of Streptococcus mutans (SM) colonies. Three human milk samples from three different mothers and five infant formulas were tested. To prepare the bacterial inoculum, SM was grown in Brain Heart Infusion broth for 18 hours at 37°C in microaerophilic atmosphere. The growth of SM was determined immediately after the inoculation of milks (T₀) and after 24 hours (T₂₄) of incubation. After 24 hours of incubation (ΔT= CFUs/ml at T₂₄ – CFUs/ml at T₀) the bacterial growth changes were different among milks. Among the complementary milks tested, ΔT of formulas supplemented with Lactobacillus reuteri and with Bifidobacterium lactis was lower than those of non-supplemented formula. In conclusion, on the basis of the reduced SM growth in milks supplemented with probiotics, we may speculate that these formulas have a preventive effect on the development of caries in children.
LETTER TO THE EDITOR

SACCHAROMYCES CEREVISIAE FUNGEMIA, A POSSIBLE CONSEQUENCE OF THE TREATMENT OF CLOSTRIDIUM DIFFICILE COLITIS WITH A PROBIOTICUM

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The yeast Saccharomyces boulardii is a biotherapeutic agent used for the prevention and treatment of several gastrointestinal diseases, such as diarrhoea caused by Clostridium difficile, in addition to the antibiotic therapy. In this study we report a case of Saccharomyces cerevisiae fungemia in a patient with Clostridium difficile-associated diarrhoea (CDAD) treated orally with S. boulardii in association with vancomycin. The identification of the S. cerevisiae was confirmed by molecular technique. Fungemia is a rare, but a serious complication to treatment with probiotics. We believe it is important to remind the clinicians of this risk when prescribing probiotics, especially to immunocompromised patients.