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Nicole L. Day, Condace L. Floyd, Tracy L. D'Alessandro, William J. Hubbard, and Irshad H. Chaudry


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Abstract

Traumatic brain injury (TBI) is a significant public health problem in the United States. Despite preclinical success of various drugs, to date all clinical trials investigating potential therapeutics have failed. Recently, sex steroid hormones have sparked interest as possible neuroprotective agents after traumatic injury. One of these is 17β-estradiol (E2), the most abundant and potent endogenous vertebrate estrogen. The goal of our study was to investigate the acute potential protective effects of E2 or the specific G protein-coupled estrogen receptor 1 (GPER) agonist G 1 when administered in an intravenous bolus dose 1 hour post-injury in the lateral fluid percussion (LFP) rodent model of TBI. The results of this study show that, when assessed at 24 hours post-injury, E2 or G 1 confers protection in adult male rats subjected to LFP brain injury. Specifically, we found that an acute bolus dose of E2 or G 1 administered intravenously 1 hour post-TBI significantly increases neuronal survival in the ipsilateral CA 2/3 region of the hippocampus and decreases neuronal degeneration and apoptotic cell death in both the ipsilateral cortex and CA 2/3 region of the hippocampus. Finally, these effects were observed to be dose-dependent for E2, with the 5 mg/kg dose generating a more robust level of protection. Our findings further elucidate estrogenic compounds as a clinically relevant pharmacotherapeutic strategy for treatment of secondary injury following TBI, and intriguingly, reveal a novel potential therapeutic target in GPER.

Key words: apoptotic; estrogen; lateral fluid percussion; neuronal degeneration; neuroprotection

Introduction

Traumatic brain injury (TBI) is a significant public health problem in the United States. Annually, approximately 1.7 million TBIs are incurred, 53,000 people die, and 3.2-5.3 million others are living with long-term disabilities as a result. Despite preclinical success, to date all clinical trials investigating potential therapeutics have failed. In addition, the financial burden associated with TBI is estimated at roughly $50 billion a year.13 TBI-induced biochemical (primary injury) and neurochemically-mediated damage (secondary injury) often lead to deficits in cognitive, motor, psychiatric, and physical functioning.14 Secondary injury mechanisms remain targets in the pathophysiology of TBI that could be manipulated by therapeutic interventions for prevention of further cell death and dysfunction. Thus, there is a significant interest need for novel drug therapies that efficaciously target aspects of secondary injury.

Recently, sex steroid hormones have sparked interest as possible therapeutic agents following traumatic injury. One of these is 17β-estradiol (E2), the most abundant and potent endogenous vertebrate estrogen. Our research group has previously reported that E2 administration confers protection in models of spinal cord injury (SCI) and severe blood loss. In prior TBI research, E2 has been shown to reduce cortical contusion volumes, apoptosis, blood-brain barrier permeability, edema, levels of pro-inflammatory cytokines, and intracranial pressure (ICP), as well as to upregulate expression of anti-apoptotic proteins Bcl-2, increase central perfusion pressure (CPP), and improve neurologic scores.12-20 Taken together, these data suggest that E2 is protective and warrant further study as a potential therapeutic for treatment of TBI. E2 signals through the classical estrogen receptors α and β (ER/β) and the recently characterized G protein-coupled estrogen receptor 1 (GPER), which binds E2 and various estrogenic compounds, including the GPER-specific agonist, G 1, and initiates rapid intracellular signaling events.21-26 However, GPER’s role in the CNS has yet to be fully characterized, and its potential contributions to protection in TBI remain uninvestigated. Because GPER binds E2 as well as other sex steroids, it could serve as a novel therapeutic target.
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Immunosuppression regimen and the risk of acute rejection.

Locke JE, James NT, Mannon RB, Mehta SG, Pappas NJ

Author information

Abstract

BACKGROUND: Kidney transplantation (KT) is the treatment of choice for patients with end-stage renal disease. However, acute rejection (AR) remains a significant challenge. Adopting an immunosuppression regimen that minimizes rejection rates while maximizing patient survival is important. This study aimed to evaluate the risk of AR and the impact of different immunosuppression regimens on patient survival.

METHODS: We conducted a retrospective analysis of 516 HIV-positive kidney transplant recipients included in the Registry of Transplant Recipients data from 2003 to 2011.

RESULTS: Compared to HIV-negative patients, HIV-positive transplant recipients had a higher risk of AR (hazard ratio [HR], 1.45; 95% confidence interval [CI], 1.15-1.82; P = 0.001), but these differences were not statistically significant. However, acute rejection rates among patients receiving ATG induction therapy were lower (HR, 0.67; 95% CI, 0.43-0.98). Despite this, HIV-positive patients had a higher risk of graft failure, with a 2.6-fold lower risk of AR (aHR, 0.39; 95% CI, 0.26-0.59) and a 2.2-fold higher risk of graft failure compared to HIV-negative patients.

CONCLUSION: These findings support a role for ATG induction therapy in HIV-positive individuals undergoing KT. However, further research is needed to optimize immunosuppression regimens to minimize AR and improve patient survival in HIV-positive transplant recipients.

PMID: 24162248 [PubMed - indexed for MEDLINE]

MeSH Terms, Substances

MeSH Terms
Adolescent
Adult
Aged
Antilymphocyte Serum/metabolism
Calcineurin/antagonists & inhibitors
Female
Graft Rejection*
Graft Survival
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Tumor necrosis factor alpha inhibitor therapy and cancer risk in chronic immune-mediated diseases


Objective: To compare the incidence of cancer following tumor necrosis factor alpha (TNF-α) inhibitor therapy to that commonly used alternative therapies across multiple immune-mediated diseases. Methods: The Safety Assessment of Biological Therapeutics study used data from four sources: national Medicare and Medicaid databases, Tennessee Medicaid, pharmacy benefits plans for Medicare beneficiaries in New Jersey and Pennsylvania, and Kaiser Permanente Northern California. Propensity score-adjusted hazard ratios and 95% confidence intervals were computed to estimate the relative rates of cancer, comparing those treated with TNFα inhibitors to those treated with alternative disease-modifying therapies. The cancer-finding algorithm had a positive predictive value ranging from 31% for any leukemia to 89% for female breast cancer. Results: We identified 29,955 patients with rheumatoid arthritis (RA) (13,122 person-years), 6,357 patients with inflammatory bowel disease (IBD) (1,583 person-years), 1,098 patients with psoriasis (731 person-years), and 2,468 patients with psoriatic arthritis (618 person-years). The incidence of any solid cancer was not elevated in RA (HR 0.80 [95% CI 0.59-1.08]), inflammatory bowel disease (HR 1.42 [95% CI 0.47-4.26]), psoriasis (HR 0.58 [95% CI 0.30-1.03]), or psoriatic arthritis (HR 0.74 [95% CI 0.20-2.76]) during TNFα inhibitor therapy compared to disease-specific alternative therapy. Among RA patients, the incidence of any of the 10 most common cancers in the US and nonmelanoma skin cancer was not increased with TNFα inhibitor therapy compared to treatment with comparator drugs. Conclusion: Short-term cancer risk was not elevated among patients treated with TNFα inhibitor therapy relative to commonly used therapies for immune-mediated chronic inflammatory diseases in this study. Copyright © 2013 by the American College of Rheumatology.

Indexed keywords:
- EMTree: drug terms: adalimumab, azathioprine, etanercept, hydroxychloroquine, infliximab, leflunomide, mercaptopurine, methotrexate, retinoid derivative, salazosulfapyridine; steroid; tumor necrosis factor alpha inhibitor.
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Mendeley References:
- 38 references

Cited by 18 documents:
- Overview of biologic treatments in the elderly
- Tumor necrosis factor, tumor necrosis factor inhibition, and cancer risk
- Safety of synthetic and biological DMARDs: A systemic literature review informing the 2013 update of the EULAR recommendations for management of rheumatoid arthritis
- Do inflammatory bowel disease therapies cause cancer?
- Cancer risk in immunemediated inflammatory diseases (IMID)
- Infections and malignant complications of TNF inhibitor therapy in IBD
- Medical and the is source for the MoSH terms of this document.

Chemicals and CAS Registry Numbers: adalimumab, 331731-18-1; azathioprine, 448-80-6; etanercept, 185234-69-0; hydroxychloroquine, 118-42-3; 525-31-5; infliximab, 170277-31-3; leflunomide, 75075-12-6; mercaptopurine, 31441-78-8; 50-44-2; 6112-76-1; methotrexate, 15475-56-8; 59-05-2; 7413-34-5; salazosulfapyridine, 599-79-9; Antibodies, Monoclonal; Immunologic Factors; Tumor Necrosis Factor-alpha.
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