Cutaneous adverse drug reaction after continuous subcutaneous apomorphine infusion.

Related Articles

Cutaneous adverse drug reaction after continuous subcutaneous apomorphine infusion.

J Eur Acad Dermatol Venereol. 2020 Jan 28;:

Authors: Calvão J, Cardoso JC, Moreira F, Januário C, Gonçalo M

Abstract

A 48-year-old woman with a 12-year history of juvenile onset Parkinson’s Disease (PD) with severe motor complications (fluctuations and dyskinesia) and severe impact on her daily activity (baseline Hoehn & Yahr stage 2; Schwab & England of 30%; MDS-UPDRS part III of 61 in off motor condition) despite levodopa equivalent daily dose of 1200mg, started continuous subcutaneous (s.c.) apomorphine (APO) infusion (APO-go® gradually increased in one week to 3mg/h during daytime), with no immediate complications and a very good motor response within a week.

PMID: 31991500 [PubMed — as supplied by publisher]

Clonal selection in therapy related myelodysplastic syndromes and acute myeloid leukemia under azacitidine treatment.

Related Articles

Clonal selection in therapy related myelodysplastic syndromes and acute myeloid leukemia under azacitidine treatment.

Eur J Haematol. 2020 Jan 28;:


Abstract

INTRODUCTION: Therapy related myelodysplastic syndrome and acute myeloid leukemia (t-MDS/AML) are defined as complications of previous cytotoxic therapy. Azacitidine (AZA), a hypomethylating agent, has showed activity in t-MDS/AML.

OBJECTIVES: We evaluated the clonal dynamics of AZA treated t-MDS/AML.

METHODS: We collected bone marrow samples, at diagnosis and during treatment, from AZA-treated t-MDS/AML patients. NGS on 19 myeloid genes was performed and candidate mutations with a variant allele frequency >5 % were selected.

RESULTS: 7 t-AML and 12 t-MDS were included with median age of 71 (56–82) years old, median number of AZA cycles of 6 (1–15) and median overall survival (OS) of 14 (3–29) months. We observed correlation between AZA response and clonal selection. Decrease of TP53 mutated clone was correlated with response to AZA, confirming AZA efficacy in this subgroup. In some patients, emergence of mutations was correlated with progression or relapse without impact on OS. Clones with mutations in genes for DNA methylation regulation frequently occurred with other mutations and remained stable during AZA treatment, independent of AZA response.

CONCLUSION: We confirmed the molecular complexity of t-MNs and that the follow-up of clonal selection during AZA treatment could be useful to define treatment combination.

PMID: 31990086 [PubMed — as supplied by publisher]

Comprehensive genetic structure analysis of Han population from Dalian City revealed by 20 Y-STRs.

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Comprehensive genetic structure analysis of Han population from Dalian City revealed by 20 Y-STRs.

Mol Genet Genomic Med. 2020 Jan 27;e1149


Abstract

BACKGROUND: Dalian is a city formed in the 1880s in Liaoning
Sulodexide for the Symptoms and Signs of Chronic Venous Disease: A Systematic Review and Meta-analysis.

Related Articles

Sulodexide for the Symptoms and Signs of Chronic Venous Disease: A Systematic Review and Meta-analysis.

Adv Ther. 2020 Jan 27;

Authors: Bignamini AA, Matuška J

Abstract

INTRODUCTION: Chronic venous disease (CVD) is a common condition associated with valvular dysfunction, venous hypertension and endothelial inflammation. Sulodexide facilitates the healing of venous ulcers and is frequently used in patients with CVD without ulcer. This review assessed the efficacy and safety of sulodexide for treatment of signs and symptoms of lower extremity CVD.

METHODS: We searched MEDLINE, EMBASE, CINAHL and AMED as well as the Cochrane Central Register of Controlled Trials and the World Health Organisation (WHO) International Clinical Trials Registry Platform Search Portal. We also manually searched potentially relevant journals, conference proceedings and journal supplements. Any study monitoring any effect of sulodexide in patients with CVD at any stage of the disease, classified or non-classified, was considered. Treatment effects were estimated using standardised mean differences (SMDs), mean differences (MDs) and risk ratios (RRs), as appropriate. We calculated 95% confidence intervals (CIs) and heterogeneity (Q, tau and I2).

RESULTS: The search found 64 studies, but only 23 provided data on 7153 participants (mean age 55 years; 68% female). The 13 studies providing extractable quantitative information included 1901 participants (mean age 55.2 years; 65% female). Sulodexide decreased the intensity of pain, cramps, heaviness, oedema and total symptom score and reduced inflammatory mediators in patients with CVD. The risk of adverse events (AEs) was not different between sulodexide and placebo or heparan sulphate (RR 1.31, 95% CI 0.74−2.32; I2 = 0%; 270 participants). The overall risk of AEs with sulodexide was low: 3% (95% CI 1−4%) estimated from 3656 participants.

CONCLUSION: Sulodexide was found to have a beneficial venoactive effect on the major signs and symptoms of CVD such as pain, cramps, heaviness and oedema without increasing the risk of AEs. It is also likely to exert a systemic effect on the course of CVD by interfering with inflammatory chemokines.

PMID: 31989486 [PubMed — as supplied by publisher]

Impact of clinical features, cytogenetics, genetic mutations, and methylation dynamics of CDKN2B and DLC-1 promoters on treatment response to azacitidine.

Related Articles

Impact of clinical features, cytogenetics, genetic mutations, and methylation dynamics of CDKN2B and DLC-1 promoters on treatment response to azacitidine.

Ann Hematol. 2020 Jan 28;


Abstract

Azacitidine (AZA) is a DNA hypomethylation agent administered in myeloid neoplasms; however, there is still a lack of established predictors of response. We studied 113 patients with myelodysplastic syndromes (n = 85) or acute myeloid leukemia (n = 28) who received AZA to assess the predictive value on response of clinical features, cytogenetics, genetic markers. Overall, 46 patients (41%) responded to AZA. Platelet doubling after the first AZA cycle was associated with a better response (68% vs. 32% responders, P = 0.041). Co-occurrence of chromosome 7 abnormalities and 17p deletion was associated with a worse response (P = 0.039). Pre-treatment genetic mutations were detected in 98 patients (87%) and methylation of CDKN2B and
DLC-1 promoters were detected in 50 (44%) and 37 patients (33%), respectively. Patients with SF3B1 mutations showed a better response to AZA (68% vs. 35% responders, P = 0.008). In contrast, subjects with mutations in transcription factors (RUNX1, SETBP1, NPM1) showed a worse response (20% vs. 47% responders, P = 0.014). DLC-1 methylation pre-treatment was associated with poor clinical features and its reduction post-treatment resulted in a better response to AZA in MDS patients (P = 0.037). In conclusion, we have identified several predictors of response to AZA that could help select the best candidates for this treatment.

PMID: 31989250 [PubMed — as supplied by publisher]

Moral Disengagement Strategies in Sex Offenders.

Related Articles

Moral Disengagement Strategies in Sex Offenders.
Psychiatr Psychol Law. 2017;24(3):470–480

Authors: Petruccelli I, Simonelli C, Barbaranelli C, Grilli S, Tripodi MF, D’Urso G

Abstract
Sexual abuse is a heterogeneous phenomenon. The literature on sexual offenders considers risk factors in the individual and familial history as well as precursors such as cognitive distortions, defence mechanisms and moral disengagement (MD) mechanisms. This study investigates the MD in sex offenders and non-sex offenders in a sample of 362 males comprising a control group of 268 non-offenders, a group of 42 detained sex offenders and a group of 52 detained non-sex offenders. Participants were administered a semi-structured interview and the Moral Disengagement Scale (MDS). The results show a significant difference between the jailed participants (non-sex offenders and sex offenders) and controls; offenders were found to generally display overall higher levels of MD. Among the jailed participants, sex offenders seem to make more use of MD mechanisms than non-sex offenders.

PMID: 31983968 [PubMed]