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Chugai's Enspryng Launched in Taiwan as First Indication for Neuromyelitis Optica Spectrum Disorder (NMOSD)

TOKYO, October 2, 2023 -- Chugai Pharmaceutical Co., Ltd. (TOKYO: 4519) announced that Chugai Pharma Taiwan Ltd., a wholly-owned subsidiary of Chugai, has launched Enspryng®, a drug created by Chugai on October 1 for "the treatment of neuromyelitis optica spectrum disorder (NMOSD) in adult and adolescent over 12 years old patients who are anti-aquaporin-4 (AQP4) antibody positive" as the first indication in Taiwan. Enspryng was designated as an orphan drug Ministry of Health and Welfare (MoHW) on September 14, 2022 and approved orphan drug license by Taiwan Food and Drug Administration (TFDA) on July 28, 2023.

<Reference>
Chugai's Enspryng Approved in Taiwan as First Approved Medicine for Neuromyelitis Optica Spectrum Disorder (NMOSD) (Press release issued on December 9, 2020)
https://www.chugai-pharm.co.jp/english/news/detail/20201209170000_784.html

About Enspryng

Enspryng, designed by Chugai, a member of Roche group, is a pH-dependent binding humanized anti-IL-6 receptor antibody, which was the first product developed by applying our proprietary recycling antibody® technology. The drug is designed to prevent relapse of neuromyelitis optica spectrum disorder (NMOSD) by inhibiting the cytokine IL-6 which is a key driver in NMOSD. Enspryng has been approved for the treatment of NMOSD in more than 85 countries including Japan, the U.S. the EU.

About neuromyelitis optica spectrum disorder (NMOSD)

NMOSD is an autoimmune disease of the central nervous system characterized by inflammatory lesions in the optic nerves and spinal cord, and causes a significant decrease in quality of life due to permanent neurological disability. Patients with NMOSD frequently experience a relapsing disease course with repeated attacks leading to accumulating neurological damage and disability. Symptoms may include visual impairment, motor disability, pain leading to decreased quality of life. In some cases, attacks of NMOSD result in death. Aquaporin-4 antibodies (AQP4-IgG), pathogenic antibodies, are detected in around 70-80% of NMOSD people. AQP4-IgG is known to target and damage a specific central nervous cell type called astrocytes, resulting in inflammatory demyelinating lesions of the optic nerve(s), spinal cord and brain.[1-4] The inflammatory cytokine IL-6 is now emerging as an important factor in NMOSD pathogenesis.[5-9] Diagnostic criteria introduced in 2006 for NMO were characterized by inflammation of the optic nerve (optic neuritis) and the spinal cord (myelitis). These were revised in 2007 with the definition of NMOSD, proposed for diseases with either optic neuritis or myelitis. In 2015, the definition of NMOSD further revised to include a broader spectrum of diseases. The diagnostic term NMOSD is now accepted.[10]

CHUGAI PHARMACEUTICAL CO., LTD. Corporate Communications Dept.
1-1, Nihonbashi-Muromachi 2-chome, Chuo-ku Tokyo, 103-8324 Japan
Media Relations Group TEL:+81-(0)3-3273-0881 E-mail:pr@chugai-pharm.co.jp
Investor Relations Group TEL:+81-(0)3-3273-0554 E-mail:ir@chugai-pharm.co.jp

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