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New Data of Chugai's Enspryng (Satralizumab) on Risk and Severity of Relapse in Neuromyelitis Optica Spectrum Disorder (NMOSD)

- Enspryng lowered relapse severity in double-blind periods of SAkura Phase III studies (SAkuraSky, SAkuraStar).
- Pooled data from SAkura open-label extension (OLE) studies support continued effect of Enspryng reducing risk of relapse.
- Ongoing data continues to show a favorable safety profile for Enspryng

TOKYO, September 10, 2020 -- Chugai Pharmaceutical Co., Ltd. (TOKYO: 4519) announced the presentation of new Enspryng® (generic name: satralizumab [genetical recombination]) data on reducing relapse severity in the treatment of neuromyelitis optica spectrum disorder (NMOSD), a rare disease of the central nervous system. These data are being presented at MS Virtual 2020, the 8th joint ACTRIMS-ECTRIMS meeting, in addition to longer-term efficacy and safety data on the continued effect of Enspryng on reducing the risk of NMOSD relapse as well as its benefit-risk profile. Enspryng is a pH-dependent binding humanized anti-IL-6 receptor monoclonal antibody created by Chugai.

"Disability in people with NMOSD is known to progressively worsen with each relapse. It is important to reduce both the severity and frequency of relapses in the long term," said Chugai's President and COO, Dr. Osamu Okuda. "With a novel mechanism of action to inhibit IL-6 signaling, which is a key driver in NMOSD, Enspryng shows the possibility to reduce the risk and severity of relapses in the long term. These efficacy data will provide clinical insight for treatment with Enspryng."

In a post-hoc analysis of the Enspryng-treated group, the risk of severe relapse was reduced by 79% compared to placebo (5 of 27 [19%] vs. 12 of 34 [35%]), for patients across the double-blind periods of the SAkura studies (SAkuraSky, SAkuraStar). Preventing relapses, the most severe of which cause cumulative, irreversible neurological damage and disability, is the primary goal for NMOSD disease management. The patients treated with Enspryng were also less likely to require rescue therapy for a relapse compared with placebo (OR 0.46; 95% CI, 0.25–0.86). A relapse was categorized as severe if it resulted in a change of ≥2 points on the Expanded Disability Status Scale.

In a separate pooled analysis, Enspryng reduced the risk of relapse in the combined double-blind period and open-label extension (OLE) by 51% (HR, 0.49; 95% CI, 0.31–0.79) compared to those originally in the placebo group. In aquaporin-4 antibody (AQP4-IgG) seropositive patients, who tend to experience a more severe disease course, Enspryng showed a 66% reduction in the risk of relapse (HR, 0.34; 95% CI, 0.19–0.62) compared to those originally in the placebo group. The median (range) duration of exposure in the Enspryng and placebo groups during the double-blind period was 96.1 weeks (8-224 weeks) and 54.6 weeks (7-219 weeks), respectively, and the median duration (range) of the combined double-blind and OLE periods in the Enspryng group was 131.9 weeks (13-276 weeks).

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

In the double-blind periods, infection rates in the Enspryng-treated and placebo groups in the SAkuraStar study were 99.8 and 162.6 events/100 patient years (PY), respectively, whereas infection rates did not differ between groups in the SAkuraSky study. Serious infection rates were comparable between both groups (Enspryng vs placebo) in each of the studies (SAkuraSky: 2.6 vs 5.0 events/100PY; SAkuraStar: 5.2 vs 9.9 events/100PY). Infection and serious infection rates for Enspryng-treated patients in the combined double-blind and OLE periods were consistent with those for Enspryng-treated patients in the double-blind portion in terms of the nature and rate of adverse events and did not increase over time.

About Enspryng

Enspryng, created by Chugai, 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. In two global phase III clinical studies in Neuromyelitis Optica and NMOSD, the primary endpoint was achieved with satralizumab either as combination therapy with baseline immunosuppressant treatment (SAkuraSky; NCT02028884) or as monotherapy (SAkuraStar; NCT02073279). These studies represent one of the largest clinical trial programs undertaken for this rare disease. Enspryng has been approved in Japan, the U.S., Canada, and Switzerland. Enspryng is designated as an orphan drug in Europe. The application was accepted for review by the European Medicines Agency in 2019.

About neuromyelitis optica spectrum disorder (NMOSD)[1]

NMOSD is an autoimmune disease of the central nervous system characterized by inflammatory lesions in the optic nerves and spinal cord, and cause continual and 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 [2-5]. The inflammatory cytokine IL-6 is now emerging as an important factor in NMOSD pathogenesis [6-10].

Diagnostic criteria introduced in 2006 for neuromyelitis optica 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 was further revised to include a broader spectrum of diseases. The diagnostic term NMOSD is now accepted [11].

Sources

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Contact
For Media
Chugai Pharmaceutical Co., Ltd.
Media Relations Group, Corporate Communications Dept.,
Tomoko Shimizu
Tel: +81-3-3273-0881
E-mail: pr@chugai-pharm.co.jp

For US media
Chugai Pharma USA Inc.
Casey Astringer
Tel: +1-908-516-1350
E-mail: pr@chugai-pharm.com

For European media
Chugai Pharma U.K. Ltd.
Tania Richards
Tel: +44-20-8987-5680
E-mail: pr@chugai.eu

For Taiwanese media
Chugai Pharma Taiwan Ltd.

Susan Chou
Tel: +886-2-2715-2000
E-mail: pr@chugai.com.tw

For Investors
Chugai Pharmaceutical Co., Ltd.
Investor Relations Group, Corporate Communications Dept.,
Toshiya Sasai
Tel: +81-3-3273-0554
E-mail: ir@chugai-pharm.co.jp

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