The Early Effect Onset of SSRI to the Non-Organic-Pain in the Mouth and Face Area

Tsunemasa OHKUBO[1)], Toshihiro ANDO[2)], Meiho NAKAYAMA[3)] and Hideaki KATO[4)]

1） Japanese Red Cross Takayama Hospital, Department of Dentistry and Oral Surgery, Takayama Japan
2） Japanese Red Cross Takayama Hospital, Department of psychological medicine, Takayama Japan
3） Good Sleep Center, Nagoya City University Hospital, Nagoya Japan
4） Suda mental hospital, Takayama Japan

These contents obtain the approval of Japanese Red Cross Takayama Hospital Ethics Committee and it is considering sufficiently about the ethical models such as the protection of the privacy.
There is not a state of the conflict of interests (COI).
【 key words 】Glossodynia, Escitalopram, Endogenic opioid system

Ⅰ Introduction

As for the glossodynia which is the typical affection of the non- organic-pain of the maxilla-facial area, the cause isn't clear yet. In the ambulatory-practice, it is one of the affections most anxiously in the diagnosis and at the medical treatment. At meals, the tongue pain often reduces and it often disappears but daily, it appears something like the inflammatory diseases. Specifically, it experiences a lot of examples to occur more often and to complain to the woman of the convex experience which is in the cancer age of them as the cancer phobia, too. Therefore, it thinks that it places a glossodynia as so-called non organic chronic pain disease.

Ⅱ Materials and Methods

From 2013 in 3 years and half-year of the pasts, we treated with Escitalopram of SSRI for 50 examples (45 female and 5 male) of the glossodynia which complained of the non-organic-pain. The evaluation used Visual Analogue Scale (VAS).

Ⅲ Result

Becoming VAS0 was 29 examples in 40 examples to the 4th week. The half example did a remission rate within two weeks. Specifically, it did 2 examples by the remission within 1 week. Finally, we get the remission rate at 75 %.

Ⅳ Discussion

Usually, the effect manifestation is recognized that it does the activation of the descending pain modulation system in SSRI or SNRI by the non-organic-pain patient. We get the half example did a remission rate within two weeks. This result supports the common view to show an effect comparatively in short time at low dosage when using SSRI or SNRI to the chronic pain.

While, the research by fMRI and PET became accomplished and it found the thing that the involvement of the system which secretes an endogenous-opioid peptide by the activation of the endogenic dopamine system is concerned. It reasoned by what action mechanism a dolor arrest was accomplished by prescribing Escitalopram which is SSRI at this article.

Generally, in case of pain added to the human body, by that phasic dopamine which presents the transient firing of burst style like from the ventrotegmental area (VTA) of the midbrain is slipped in quantities, peptide of the endogenous-opioids such as the β-endorphin is produced in the nucleus accumbens (NAc) and the ventral pallidum (VP) and a pain is restrained [1)]. This

is the operation of the A10 nerve which is stretching an axis cylinder in NAc, or VP and at the mesolimbic dopamine system from VTA and is for peptide of the endogenous-opioids such as the β-endorphin to be secreted by the production through the mesolimbic dopamine system and for a pain to be restrained through the descending pain modulation system.

By fMRI (functional magnetic resonance imaging) Hanba is reporting that the cerebral activity of NAc declines compared with 16 chronic lumbar backache patient and 16 able-bodied people, adding a heat stimulation repeatedly[2]. By the report which used adding PET in the pain stimulation a μ-opioid was secreted of the nucleuses such as the NAc, the amygdaloid body, the anterior cingulate gyrus, the frontal cortex, the thalamus is reported[3]

Actually, We knew that the dopamine which was confiscated from these as the main system of " the reward circuit " which relates to the higher brain dysfunction such as the emotion and the reward, the learning and the exercise in the past concerned pain restraint deeply[4,5].
Serotonin concentration is supposed to decline with the chronic stress and the fatigue like a mind and body. Also, the patient who complains of the chronic pain is often exposed to the chronic stress and the fatigue and there are not even among them few persons who present a depressed state.

In the depressive state, it falls into a state of the serotonin lack at both of the dendrite and the synapse area and the serotonin receptor behind the presynapse and the synapse presents a state of the up-regulation. When prescribing SSRI, by the obstruction of the serotonin transporter, in the early stages of the prescribing, the serotonin in the somatodendritic area increases. Therefore, the increased serotonin restrains the isolation of the serotonin when it combines with the $5HT_{1A}$ receptor which is an autoreceptor on the somatodendritic of the presynapse and another $5HT_{2A}$ receptors of synapses on the dopamine nerve cell aren't activated. For glutamicacid in the brain stem through the $5HT_{2A}$ receptor not to be released, inhibitory GABA, too, isn't released and prompts for the dopamine in the downstream corpus striatum. The opioid peptide of stimulating an accumbens nucleus by the dopamine which was liberated in this way through the mesolimbic dopamine system as phasic dopamine by the ventrotegmental area such as β-endorphin (Fig.1). It forwards the production of opioid peptide and a chronic pain is restrained through the μ opioid-receptor of the descending pain modulation system[6].

Ⅴ Conclusion

We reported because we prescribed Escitalopram which is SSRI to 40 cases of glossodynia by the stress and supposed a theoretical background to the effect. As a result, the rise of the dopamine concentration is supposed to be gotten mainly in the A10 nerve when prescribing Escitalopram for the non-organic-pain patient by it, and it supposes that the dopamine neural circuit which covers a limbic cortex and a basal ganglia, a midbrain and so on is made activation when prescribing and endogenous-opioid peptide is supposed to be secreted by it and it supposed a pain control to be attempted through the descending pain modulation system.

Ⅵ Reference

1) Shin-ichi Konno : Dopamine System and pain, Clinical Orthopaedic Surgery 46:343-346,2011
2) Michiko Hanba : Chronic pain and Brain, Practice of Pain Management 1:32-36,2010
3) Zubieta JK, Bueller JA et al: Placebo effects mediated by endogenous opioid activity on μ-opioid reception. J Neurosci 25:7754-

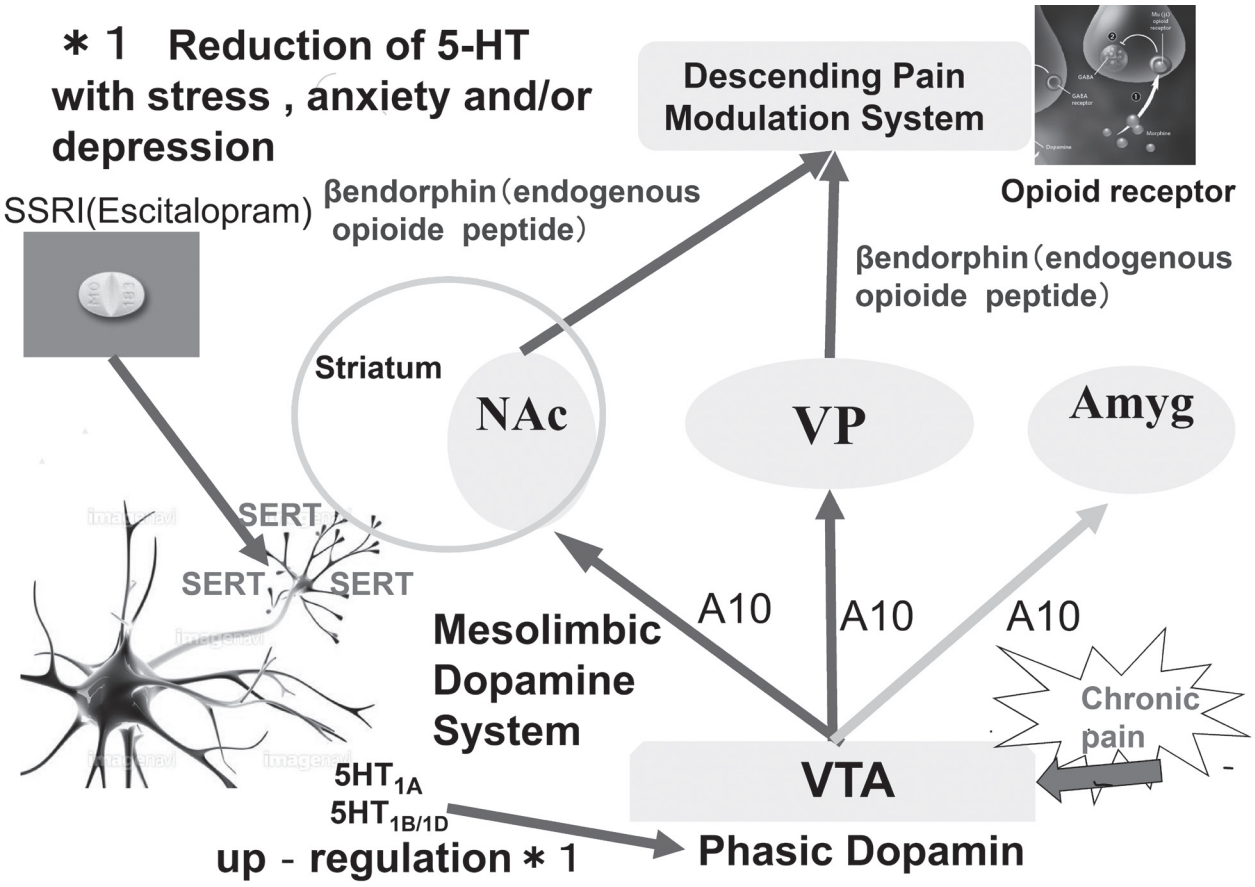

Fig.1: The chronic pain disappearance in short time by Phasic Dopamine increase

7762,2005

4）Wood PB: Mesolimbic dopaminergic mechanisms and pain control. Pain 120:230-234.2006

5）Leknes S, Tracey I: A common neurobiology for pain and pleasere. Nature Reviews Neurosci 9: 314-320.2008

6）Tsunemasa Ohkubo, Meiho Nakayama et.al: The influence over the dopamine by SSRI, JRC Takayama Hosp 40:33-37.2016