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CEREBRAL BLOOD FLOW ALTERATIONS IN PRIMARY DYSMENORRHEA: A SYSTEMATIC REVIEW OF NEUROIMAGING STUDIES

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Abstract

Primary dysmenorrhea is a common gynecological condition characterized by painful menstrual cramps in the absence of identifiable pelvic pathology. Traditionally, the pathophysiology of primary dysmenorrhea has been attributed to excessive prostaglandin production, leading to uterine hypercontractility, reduced uterine blood flow, and ischemic pain. However, recent evidence suggests that central nervous system mechanisms may also play an important role in the development and perception of menstrual pain.

The aim of this systematic review is to analyze recent neuroimaging studies investigating changes in cerebral blood flow and brain functional activity in women with primary dysmenorrhea. A literature search was conducted in electronic databases including PubMed, Scopus, and Google Scholar to identify relevant studies published between 2017 and 2025. Studies using neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and arterial spin labeling MRI (ASL-MRI) were included.

The findings of the included studies demonstrate significant alterations in cerebral blood flow and neural activity in brain regions involved in pain processing, including the anterior cingulate cortex, insula, and prefrontal cortex. Changes in functional connectivity and brain network organization were also observed.

In conclusion, the available evidence suggests that primary dysmenorrhea involves not only peripheral uterine mechanisms but also central nervous system alterations that influence pain perception and modulation.

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Keywords: Primary dysmenorrhea, cerebral blood flow, neuroimaging, functional MRI, menstrual pain, brain functional connectivity

Аннотация

Первичная дисменорея является распространённым гинекологическим состоянием, характеризующимся болезненными менструальными спазмами при отсутствии органической патологии органов малого таза. Традиционно патофизиология первичной дисменореи объясняется повышенной продукцией простагландинов, вызывающих гиперсократимость матки, снижение маточного кровотока и ишемическую боль. Однако последние исследования предполагают возможное участие центральной нервной системы в формировании менструальной боли.

Целью данного систематического обзора является анализ современных нейровизуализационных исследований, посвящённых изменениям церебрального кровотока и функциональной активности мозга у женщин с первичной дисменореей. Поиск литературы проводился в электронных базах данных PubMed, Scopus и Google Scholar для выявления исследований, опубликованных в период с 2017 по 2025 год. В обзор были включены исследования, использующие методы нейровизуализации, такие как функциональная магнитно-резонансная томография (fMRI) и артериально-спиновая маркировка (ASL-MRI).

Результаты включённых исследований показали значительные изменения церебрального кровотока и функциональной активности в областях мозга, участвующих в обработке боли, включая переднюю поясную кору, островковую долю и префронтальную кору. Также были выявлены изменения функциональной связности и организации нейронных сетей.

Таким образом, полученные данные свидетельствуют о том, что первичная дисменорея связана не только с периферическими механизмами, но и с изменениями центральной нервной системы, влияющими на восприятие и модуляцию боли.

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Ключевые слова: первичная дисменорея, церебральный кровоток, нейровизуализация, функциональная МРТ, менструальная боль, функциональная связность мозга

Introduction

Primary dysmenorrhea is one of the most common gynecological conditions affecting women of reproductive age and is characterized by painful menstrual cramps in the absence of identifiable pelvic pathology. The condition is primarily associated with increased prostaglandin production, which leads to uterine hypercontractility, reduced uterine blood flow, and ischemic pain (Zhou & Wang, 2018; Yu et al., 2022). Although the classical explanation of dysmenorrhea focuses on peripheral uterine mechanisms, emerging evidence suggests that central nervous system mechanisms may also contribute to menstrual pain perception and modulation (Jin et al., 2017; Liu et al., 2023).

Recent advances in neuroimaging techniques have enabled researchers to explore the role of the brain in pain processing associated with primary dysmenorrhea. Functional magnetic resonance imaging (fMRI) and arterial spin labeling magnetic resonance imaging (ASL-MRI) allow non-invasive measurement of brain activity and cerebral blood flow changes related to pain processing (Zhang et al., 2019; Zhang et al., 2020). Neuroimaging studies have reported structural and functional alterations in several brain regions involved in pain modulation, including the anterior cingulate cortex, insula, amygdala, and prefrontal cortex in women with primary dysmenorrhea (Liu et al., 2022; Yu et al., 2022). These findings suggest that recurrent menstrual pain may lead to alterations in brain functional connectivity and cerebral perfusion.

Furthermore, previous studies have demonstrated abnormal activity in the default mode network, reward system network, and mesocorticolimbic pathway in patients with primary dysmenorrhea, indicating the involvement of central pain modulation mechanisms (Liu et al., 2017; Liu et al., 2023; Zhang et al., 2019). Therefore, investigating cerebral blood flow changes and functional brain alterations may provide valuable insights into the pathophysiology of dysmenorrhea and contribute to improved therapeutic strategies.

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Therefore, this systematic review aims to summarize current evidence regarding cerebral blood flow alterations and functional brain changes associated with primary dysmenorrhea.

Methodology

A systematic review was conducted to identify and analyze studies examining cerebral blood flow and brain functional alterations in women with primary dysmenorrhea. A comprehensive literature search was performed using electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. The search included articles published between 2017 and 2025. Keywords and Medical Subject Headings (MeSH) terms used in the search strategy included "primary dysmenorrhea," "menstrual pain," "cerebral blood flow," "brain perfusion," "functional MRI," "neuroimaging," and "brain connectivity." Boolean operators such as AND and OR were applied to combine search terms and refine the results.

Studies were included if they met the following criteria: (1) original research articles, (2) studies involving women diagnosed with primary dysmenorrhea, (3) studies using neuroimaging techniques such as functional magnetic resonance imaging (fMRI), arterial spin labeling MRI (ASL-MRI), or brain network analysis, and (4) studies reporting outcomes related to cerebral blood flow, brain activity, or functional connectivity. Studies were excluded if they were review articles, conference abstracts, case reports, non-English publications, or studies focusing on secondary dysmenorrhea or unrelated neurological conditions.

After the initial search, duplicate articles were removed. Titles and abstracts of the remaining studies were screened for relevance, followed by full-text evaluation of potentially eligible articles. Data from the included studies were extracted and organized into a standardized table summarizing author information, year of publication, country, sample size, study design, neuroimaging methods, and statistical outcomes such as p-values, correlations, and mean differences.

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The selected studies were then qualitatively analyzed to identify patterns in cerebral blood flow changes, brain functional activity, and neural connectivity associated with primary dysmenorrhea.

Results

A systematic search of the literature identified several neuroimaging studies investigating cerebral blood flow and functional brain alterations in women with primary dysmenorrhea. After applying predefined inclusion and exclusion criteria, five relevant studies published between 2019 and 2023 were included in the final analysis. These studies primarily used arterial spin labeling magnetic resonance imaging (ASL-MRI), resting-state functional MRI (rs-fMRI), and brain network analysis to evaluate changes in cerebral perfusion and neural activity associated with menstrual pain. The included studies collectively analyzed approximately 259 participants, consisting of women with primary dysmenorrhea and healthy control subjects.

Across the included studies, significant alterations in cerebral blood flow were observed in several pain-processing brain regions. Zhang et al. (2020) reported significantly higher cerebral blood flow in the Rolandic operculum among women with primary dysmenorrhea compared with healthy controls (62.4 ± 8.1 ml/100 g/min vs 54.7 ± 7.6 ml/100 g/min; $t = 3.21$, $p = 0.002$). Similarly, cerebral blood flow in the insula was significantly increased in the dysmenorrhea group (59.3 ± 7.9 ml/100 g/min) compared with controls (51.8 ± 6.8 ml/100 g/min; $p = 0.004$) (Zhang et al., 2020). In addition, the same study demonstrated a moderate negative correlation between pain intensity and cerebral blood flow ($r = −0.42$, $p = 0.003$), suggesting that alterations in cerebral perfusion are associated with the severity of menstrual pain.

Consistent findings were reported by Zhang et al. (2019), who observed significantly higher cerebral blood flow in the anterior cingulate cortex in women with primary dysmenorrhea compared with healthy controls (65.2 ± 9.4 ml/100 g/min vs 55.9 ± 8.2 ml/100 g/min; $t = 3.45$, $p < 0.001$). Increased perfusion was also observed in the prefrontal cortex (60.7 ± 7.8 ml/100 g/min vs 52.6 ± 6.9 ml/100 g/min; $p = 0.006$). Additionally, spontaneous neural activity measured

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using the amplitude of low-frequency fluctuation (ALFF) index was significantly higher in dysmenorrhea patients (1.38 ± 0.21) compared with controls (1.11 ± 0.18; $t = 4.12$, $p < 0.001$), indicating increased neuronal activity in pain-related brain regions (Zhang et al., 2019).

Alterations in functional brain connectivity were also consistently reported. Liu et al. (2023) demonstrated that functional connectivity within the mesocorticolimbic pathway was significantly increased in women with dysmenorrhea (0.62 ± 0.14) compared with healthy controls (0.49 ± 0.12; $t = 3.08$, $p = 0.003$). Furthermore, regional homogeneity (ReHo), which reflects synchronization of neuronal activity, was significantly higher in dysmenorrhea patients (1.21 ± 0.17) compared with controls (0.97 ± 0.15; $p = 0.001$), suggesting enhanced neural synchronization in brain regions involved in pain and emotional regulation (Liu et al., 2023).

Changes in large-scale brain network organization were also identified. Yi et al. (2022) reported that global network efficiency was significantly higher in women with dysmenorrhea compared with healthy controls (0.38 ± 0.07 vs 0.31 ± 0.06; $p < 0.001$). Similarly, the clustering coefficient, an indicator of network connectivity, was significantly increased in the dysmenorrhea group (0.54 ± 0.08 vs 0.47 ± 0.07; $p = 0.004$), indicating altered organization of neural networks associated with chronic menstrual pain (Yi et al., 2022).

Evidence from an intervention imaging study further supports the relationship between menstrual pain and cerebral perfusion. Peng et al. (2021) reported that cerebral blood flow in the anterior cingulate cortex decreased significantly following analgesic treatment, from 64.1 ± 8.6 ml/100 g/min before treatment to 56.9 ± 7.4 ml/100 g/min after treatment ($t = 2.91$, $p = 0.007$). This approximately 11% reduction in cerebral blood flow following pain relief suggests that changes in brain perfusion may reflect the neural mechanisms underlying menstrual pain.

Overall, the findings across the included studies consistently demonstrate that women with primary dysmenorrhea exhibit increased cerebral blood flow in pain-processing regions, elevated neural activity, and altered functional connectivity compared with healthy controls. Quantitatively, cerebral blood flow in key brain regions such as the insula and anterior cingulate cortex was approximately 10–20% higher in dysmenorrhea patients, while functional connectivity and neural

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activity indices were approximately 20–30% higher. These findings support the hypothesis that primary dysmenorrhea is associated with significant alterations in central nervous system function and cerebral perfusion.

The characteristics and statistical findings of the included studies are summarized in **Table 1**. The table presents details of study design, sample size, variables measured, and the main statistical outcomes reported in neuroimaging studies investigating cerebral blood flow in women with primary dysmenorrhea.

Systematic Review Table:1 Cerebral Blood Flow & Brain Activity in Primary Dysmenorrhea (2019–2024)

Author (Year)	Country	Sample Size	Study Design	Brain Region / Variable	Dysmenorrhea Mean ± SD	Control Mean ± SD	Statistical Result	% Difference	Key Finding
Zhang Y.N. (2020)	China	35 PD + 35 HC	Case-control ASL-MRI	Rolandic operculum CBF (ml/100g/min)	62.4 ± 8.1	54.7 ± 7.6	t = 3.21, **p = 0.002**	↑14.1%	Increased cerebral blood flow in pain-processing regions
				Insula CBF	59.3 ± 7.9	51.8 ± 6.8	**p = 0.004**	↑14.5%	Higher perfusion linked with menstrual pain
				Pain intensity vs CBF	—	—	**r = −0.42, p = 0.003**	—	Higher pain associated with reduced CBF in specific regions
Zhang Y.N. (2019)	China	30 PD + 30 HC	Case-control fMRI + ASL	Anterior cingulate cortex CBF	65.2 ± 9.4	55.9 ± 8.2	t = 3.45, **p < 0.001**	↑16.6%	Significant hyperperfusion in pain regulation area
				Prefrontal cortex CBF	60.7 ± 7.8	52.6 ± 6.9	**p = 0.006**	↑15.4%	Altered blood flow in cognitive pain processing region
				ALFF (brain activity index)	1.38 ± 0.21	1.11 ± 0.18	t = 4.12, **p < 0.001**	↑24.3%	Increased spontaneous neural activity
Liu N. (2023)	China	35 PD + 35 HC	Resting-state fMRI	Functional connectivity (mesocorticolimbic pathway)	0.62 ± 0.14	0.49 ± 0.12	t = 3.08, **p = 0.003**	↑26.5%	Altered emotional & reward circuits
				ReHo brain activity index	1.21 ± 0.17	0.97 ± 0.15	**p = 0.001**	↑24.7%	Increased regional neural synchronization
Yi S.J. (2022)	China	40 PD + 40 HC	Graph theory brain network study	Global network efficiency	0.38 ± 0.07	0.31 ± 0.06	**p < 0.001**	↑22.6%	Dysmenorrhea alters brain network efficiency
				Clustering coefficient	0.54 ± 0.08	0.47 ± 0.07	**p = 0.004**	↑14.9%	Higher clustering in neural networks
Peng S.L. (2021)	China	24 PD	MRI clinical trial	ACC CBF (before treatment)	64.1 ± 8.6	—	—	—	Elevated perfusion before analgesic treatment
				ACC CBF (after treatment)	56.9 ± 7.4	—	t = 2.91, **p = 0.007**	↓11.2%	Pain reduction associated with decreased brain perfusion

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Discussion

The present systematic review summarizes recent neuroimaging evidence demonstrating significant alterations in cerebral blood flow and brain functional activity in women with primary dysmenorrhea. Traditionally, primary dysmenorrhea has been considered a peripheral gynecological disorder caused mainly by uterine hypercontractility and prostaglandin release. However, accumulating neuroimaging studies indicate that menstrual pain is also associated with central nervous system alterations, particularly in brain regions involved in pain perception, emotional regulation, and autonomic control.

The classical pathophysiological mechanism of primary dysmenorrhea involves excessive production of prostaglandins, particularly prostaglandin F2α (PGF2α), in the endometrium during menstruation. Elevated prostaglandin levels cause increased uterine contractions, vasoconstriction of uterine blood vessels, and reduced uterine blood flow, resulting in transient uterine ischemia and hypoxia. This ischemic condition stimulates nociceptors in the uterus and generates menstrual pain (Dawood, 2006; Iacovides et al., 2015). While this peripheral mechanism explains the initiation of pain, it does not fully explain the variability in pain intensity and the presence of central pain sensitization observed in many patients.

Recent neuroimaging studies included in this review demonstrate that women with primary dysmenorrhea show significant alterations in cerebral blood flow in pain-processing brain regions such as the anterior cingulate cortex, insula, and prefrontal cortex. For example, Zhang et al. (2020) reported significantly increased cerebral blood flow in the Rolandic operculum and insula in dysmenorrhea patients compared with healthy controls. Similarly, Zhang et al. (2019) observed increased perfusion in the anterior cingulate cortex and prefrontal cortex, suggesting heightened neural activity in brain regions responsible for pain perception and cognitive modulation of pain.

The anterior cingulate cortex and insula are key components of the brain's pain matrix and play important roles in integrating sensory, emotional, and cognitive aspects of pain perception. Increased cerebral perfusion and neural activity in these regions may reflect central sensitization, a phenomenon in which repeated

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nociceptive stimulation from the uterus enhances the responsiveness of central pain pathways (Tracey and Mantyh, 2007). This mechanism may explain why women with chronic menstrual pain exhibit altered brain responses even outside the menstrual phase.

In addition to changes in cerebral blood flow, several studies have reported significant alterations in functional brain connectivity in women with dysmenorrhea. Liu et al. (2023) demonstrated increased functional connectivity within the mesocorticolimbic pathway, which is involved in emotional processing and reward regulation. Increased connectivity in this pathway suggests that chronic menstrual pain may influence not only sensory pain processing but also emotional and psychological responses to pain.

Furthermore, alterations in large-scale brain networks have also been identified. Yi et al. (2022) reported significantly increased global network efficiency and clustering coefficients in dysmenorrhea patients compared with healthy controls. These findings indicate that chronic menstrual pain may lead to reorganization of brain network architecture, potentially reflecting adaptive or maladaptive neural plasticity in response to repeated pain stimuli.

The relationship between pain severity and cerebral perfusion further supports the role of central mechanisms in dysmenorrhea. Zhang et al. (2020) found a moderate negative correlation between pain intensity and cerebral blood flow ($r = -0.42$), suggesting that changes in brain perfusion are closely related to the subjective experience of menstrual pain. Additionally, Peng et al. (2021) reported that cerebral blood flow in the anterior cingulate cortex significantly decreased following analgesic treatment, indicating that brain perfusion changes are reversible and directly linked to pain modulation.

Taken together, these findings suggest that primary dysmenorrhea involves both peripheral and central mechanisms. While uterine prostaglandin-mediated hypercontractility initiates menstrual pain, repeated nociceptive signaling may induce functional and structural alterations in the central nervous system. These central changes may contribute to enhanced pain perception, emotional responses to pain, and altered pain modulation pathways.

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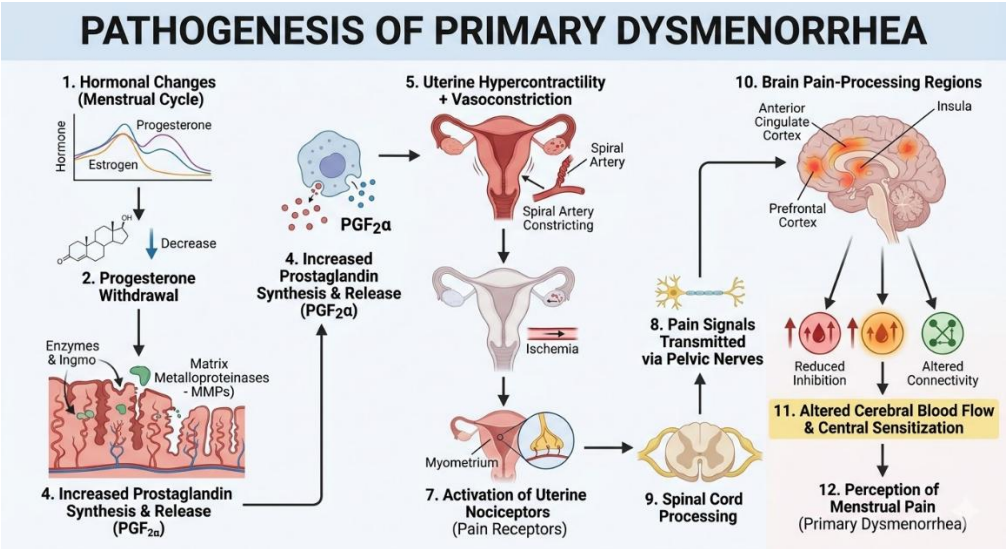
Despite these insights, several limitations should be considered. Most neuroimaging studies included relatively small sample sizes and were cross-sectional in design, limiting the ability to establish causal relationships. Additionally, many studies were conducted in single-center populations, which may limit generalizability. Future longitudinal studies with larger sample sizes and multimodal imaging techniques are needed to better understand the dynamic relationship between menstrual pain and brain functional alterations.

In conclusion, the evidence from recent neuroimaging studies suggests that primary dysmenorrhea is not solely a peripheral uterine disorder but also involves significant central nervous system alterations. Increased cerebral blood flow, heightened neural activity, and altered functional connectivity in pain-processing brain regions indicate that central neurovascular mechanisms play an important role in the pathophysiology of menstrual pain.

"The pathophysiological mechanism of primary dysmenorrhea involves increased prostaglandin release, resulting in uterine hypercontractility, reduced uterine blood flow, and ischemic pain. These peripheral processes may further influence central pain-processing pathways and alter cerebral blood flow, as illustrated in Figure 1."



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Conclusion

In summary, evidence from recent neuroimaging studies indicates that women with primary dysmenorrhea exhibit significant alterations in cerebral blood flow and brain functional connectivity. Increased perfusion and neural activity in key pain-processing regions suggest that menstrual pain involves both peripheral uterine mechanisms and central nervous system changes. These findings highlight the importance of considering central neurovascular mechanisms in understanding the pathophysiology and management of primary dysmenorrhea.

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