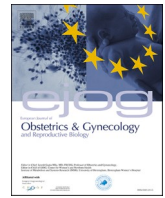




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Full length article

Pelvic pain and generalized persistent pain in people with mayer-rokitansky-küster-hauser syndrome: a cross-sectional survey study

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ABSTRACT

Background: Mayer–Rokitansky–Küster–Hauser (MRKH) syndrome is characterised by congenital underdevelopment of the uterus and vagina. While absence of menstruation might be expected to reduce pelvic pain, the prevalence and nature of pain syndromes in MRKH are not well established.

Aims: To determine the prevalence and severity of self-reported chronic pelvic pain (CPP) and generalised persistent pain (GPP) in a large Australian sample of women with MRKH, and to compare findings with general population estimates.

Materials and methods: A cross-sectional online survey was conducted in collaboration with MRKH Australia. Validated questionnaires were used to assess CPP and GPP, somatic symptom burden, and depressive symptoms. Data on age, self-reported uterine remnant status, and prior diagnosis of fibromyalgia were also collected.

Results: Among 142 participants (median age 32, range 18–71), 34.0% reported no pelvic pain in the preceding three months, while 38.3% reported moderate to severe CPP. Overall, 3.5% reported a diagnosis of fibromyalgia, comparable to global female prevalence, and 55.2% reported depressive symptoms. Younger participants described a higher burden of somatic symptoms. Pelvic pain was strongly associated with increased likelihood of GPP.

Conclusions: Women with MRKH experience moderate to severe CPP and GPP symptoms at rates comparable to those reported in the general female population. This study was designed to estimate the prevalence and severity of pelvic and generalised persistent pain, rather than to determine specific pain aetiologies. Early recognition of pain symptoms and integration of holistic, patient-centred care addressing both somatic and psychosocial contributors are recommended.

Introduction

Mayer-Rokitansky-Küster-Hauser Syndrome (MRKH) [1] is a congenital anomaly affecting 1 in 4000–5000 females [2,3]. MRKH is characterised by underdevelopment of the uterus and/or vagina, with variable phenotypic presentation. In Type 1 MRKH, isolated Müllerian agenesis occurs; Type 2 MRKH is associated with extragenital anomalies, affecting the renal, cardiac, skeletal or auditory systems [4,5]. MRKH presents in adolescence with primary amenorrhoea, normal secondary sexual characteristics, an underdeveloped vagina and 46XX karyotype [4,6]. Some individuals may also have uterine remnants, with or without functional endometrial tissue [4,5].

Pelvic and generalised pain pose significant health priorities, affecting women disproportionately to men [7], likely due to sociocultural, hormonal and psychosocial factors [7]. Painful menstruation is a well-established precursor of chronic pelvic pain (CPP) [8,9], with a recent longitudinal study reporting a 76% increased likelihood of CPP by age 26 among those with adolescent-onset dysmenorrhoea [9]. Furthermore, there are significant overlaps between CPP and generalised persistent pain (GPP) disorders including fibromyalgia, with higher fibromyalgia survey scores associated with more severe CPP symptoms [10,11]. Inflammatory mediator release during menstruation leading to cyclical nociceptor stimulation and central sensitisation is the most well understood mechanism through which dysmenorrhoea contributes to

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CPP and GPP [8,12]. Moreover, in the wider female population, risk factors for CPP include early menarche, heavy and prolonged menstruation and pelvic inflammatory disease, all of which do not affect people with congenitally absent uteri [12–15]. Thus for people born without a uterus who do not experience menstruation, there is a question of whether baseline pain rates might be lower compared to the general population. No previous studies in people with MRKH exist on the prevalence and experience of CPP, central sensitisation or GPP.

Previous traumatic events can also contribute to CPP with an established link between exposure to adverse life events (ALEs) and longer-term alterations in stress reactivity, immunological dysregulation and modification of nociceptive processing [16–18]. Women with MRKH may also experience psychosocial distress related to diagnosis and treatment [19–22]. Studies have examined psychosocial wellbeing, sexual function, and dyspareunia in women with MRKH, who typically undergo neovagina creation using surgical or non-surgical approaches [2,23–25]. However, comparatively little is known about the prevalence of non-sexual CPP and GPP in this population. Existing reports largely focus on selected clinical subgroups, including those with obstructed uterine remnants, rather than broader community-based samples [4–6,26–29].

This study aimed to estimate the prevalence and severity of CPP and GPP in women with MRKH in a large Australian community sample, and to compare these findings with available general population estimates.

Methods

Ethics

Ethical approval was granted by the James Cook University Human Research Ethics Committee (H8839) (Appendix 1).

Questionnaire and procedure

The survey was developed by the research team, with input from MRKH Australia representatives to ensure clarity and acceptability [30]. People aged ≥ 18 with MRKH were invited to participate in an online survey (Appendix 2) through MRKH Australia's newsletter and social media (Fig. 1. Sample size was calculated using Cochran's formula with finite population correction (95% confidence level; 10% margin of error; conservative proportion 0.50). Based on an estimated national MRKH population of approximately 2,000 individuals (derived from ~ 10 million adult women in Australia and prevalence of 1 in 5,000), the minimum required sample size was 92 participants) [31].

Survey completion took 5–10 min. Participants were instructed to complete the survey only once. All responses were manually reviewed for completeness. Given the anonymous design and absence of technical restrictions, duplicate submissions cannot be excluded.

The questionnaire (Appendix 3) utilised Qualtrics software [32], and included questions regarding 6 domains: (1) eligibility to participate (consent; aged > 18 years; no previous survey attempts; MRKH diagnosis), (2) age, (3) self-reported uterine remnant status, (4) pre-existing diagnosis of fibromyalgia, (5) the American College of Rheumatology's validated modified 2010 diagnostic criteria for fibromyalgia (ACRDC)

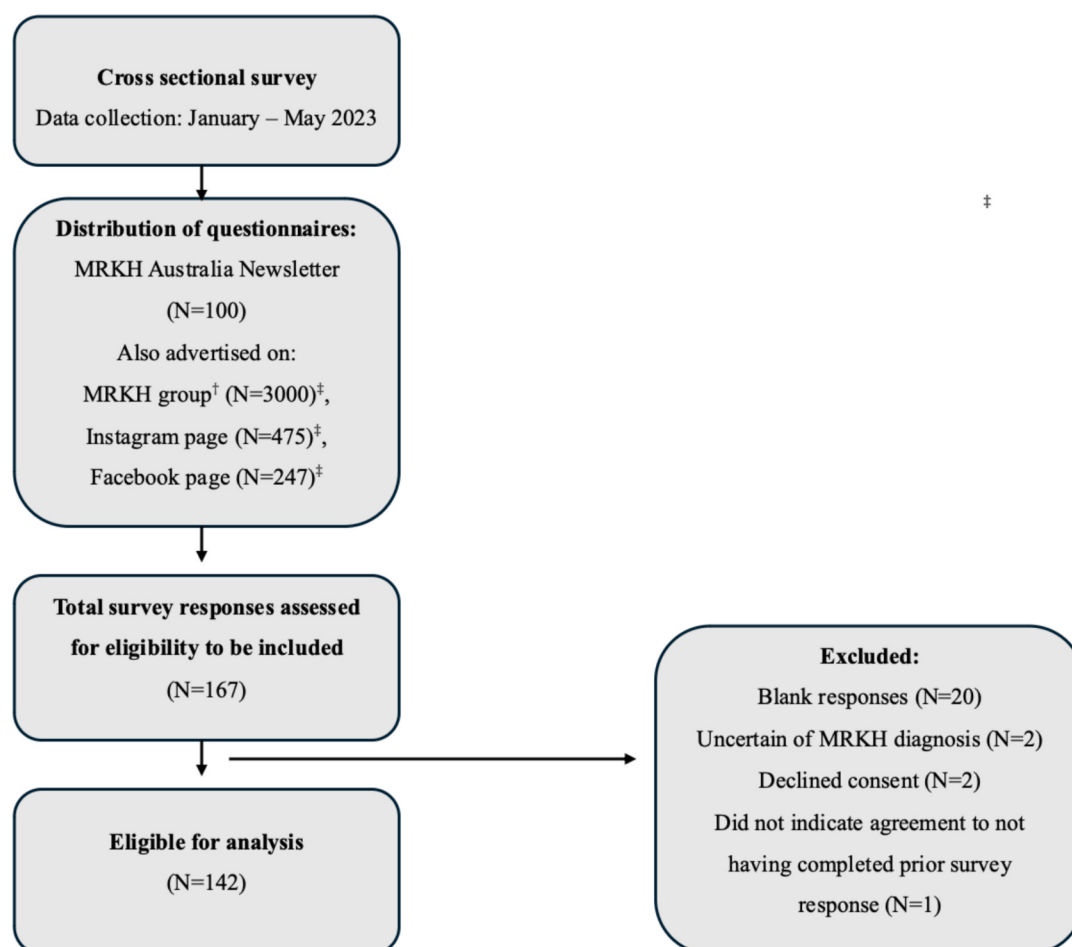


Fig. 1.

[33], and (6) presence and severity of CPP (options: none, slight, mild, moderate, and severe pain). Pelvic pain was defined as recurrent or constant pain in the pelvis over the preceding three months. The lower abdominal pain, headache and depressive symptom items referred to the preceding six months. While a separate item assessed lower abdominal pain, we acknowledge that participants may not clearly distinguish pelvic from abdominal or gastrointestinal pain, and some degree of symptom overlap or misclassification is possible. Recall bias was minimised by using specified timeframes and validated pain scales.

The ACRDC (Appendix 4) was used to measure GPP [30]. This assesses the number and distribution of pain sites utilising the Widespread Pain Index (WPI) and somatic symptom severity through the Symptom Severity Score (SSS). The WPI includes 19 pain sites across the body. The SSS constitutes an assessment of the severity of three factors (fatigue, trouble thinking/concentrating, and waking up tired/unrefreshed) over the past seven days and the presence of lower abdominal pain, depression or headaches during the past six months. A diagnosis of fibromyalgia through these criteria, requires satisfaction of 3 conditions: 1) WPI $\geq 7/19$ and SSS $\geq 5/12$, or WPI 3–6/19 and SSS $\geq 9/12$; 2) that symptoms were present for a similar level for at least 3 months; and 3) that the pain cannot be justified by another diagnosis. Participants who satisfied criteria one and two were deemed to meet the GPP threshold, as the third criterion is not validated for use in epidemiologic studies [33]. The term “generalised persistent pain” was chosen as it is this component of the fibromyalgia syndrome that this study is measuring, as opposed to diagnosing fibromyalgia.

The survey was intentionally brief in order to maximise acceptability for participants recruited through community channels. Accordingly, detailed clinical phenotyping was not performed. The questionnaire did not capture cyclical versus non-cyclical pain, hormonal suppression, prior excision of uterine remnants, neovagina creation method, dyspareunia, or distress related to diagnosis or treatment. Uterine remnant status was based on participant self-report and was not verified against imaging, operative findings, or medical records.

Data analysis

Data was analysed using IBM SPSS Version 29 [34]. Mann-Whitney-U and Pearson Chi-Square tests were used for non-normally distributed continuous variables [35,36], with Fisher's Exact Test being used for categorical data [37]. Spearman Correlation and linear regression were performed to account for the potential impact of age on pain scores and depression. Results were reported with odds ratios; 95% CI, and $P < 0.05$ was considered statistically significant.

Results

Of 167 responses, 142 were eligible for analysis and were included. One entry did not provide CPP data and was excluded from CPP analyses. Fig. 1 depicts the study procedure and exclusion reasons. Of the 117 participants who provided their age, the median was 32 years (range 18–71). Thirty-four (23.9%) participants self-reported having a uterine remnant, 97 (68.3%) reported no remnant, and 11 (7.75%) were unsure.

Prevalence and severity of CPP

Fifty-four (38.3%) participants reported experiencing moderate or severe CPP in the past three months; with 13 (9.22%) reporting severe recurrent or constant pain (Table 1). Forty-eight (34.0%) reported no pelvic pain.

Prevalence of GPP and somatic symptoms

Five participants (3.52%) reported a pre-existing diagnosis of fibromyalgia. The median WPI was 3 (range 0–17), and median SSS was 7

Table 1

Severity of non-sexual pelvic pain in the past 3 months among women with MRKH (N = 141).

Pain severity	N (%) [†]
No pain	48 (34.0%)
Slight to mild pain	39 (27.7%)
Moderate pain	41 (29.1%)
Severe pain	13 (9.22%)
Moderate-severe pain	54 (38.0%)

[†] Percentages may not total 100% due to rounding.

(range 0–12). The commonest pain site was the lower back. Table 2 details somatic symptom data. Those with moderate to severe CPP were significantly more likely to experience GPP (Table 3).

Age, depressive symptoms and pain

There was a significant correlation between increasing severity of CPP and depressive symptoms ($P = 0.002$). Similarly, a higher prevalence of depressive symptoms was noted with an increasing number of pain sites on the WPI ($P = 0.001$).

Whilst the data did not yield a significant association between age and severity of CPP ($r_s = -0.163$, $p = 0.158$) or WPI ($r_s = -0.008$, $p = 0.935$), it did yield a significant association between pain and SSS ($r_s = -0.253$, $p = 0.006$). Young age was associated with somatic symptom severity ($F = 4.997$, $p = 0.027$). There was no significant correlation between age and presence of depressive symptoms ($p = 0.169$).

Discussion

This study examines the prevalence of CPP and GPP in women with MRKH within a large Australian survey sample. Rates of moderate to severe CPP in the study population were comparable to those reported in

Table 2

Generalised persistent pain characteristics among women with MRKH (N = 142).

Variable	N (%)
Meeting generalised persistent pain threshold	40 (28.2)
Symptom severity – fatigue	No problem 20 (14.1)
	Slight or mild problem 58 (40.8)
	Moderate problem 54 (38.0)
	Severe problem 10 (7.04)
Symptom severity – trouble thinking or concentrating	No problem 28 (19.7)
	Slight or mild problem 65 (45.8)
	Moderate problem 42 (29.6)
	Severe problem 7 (4.93)
Symptom severity – waking up tired or unrefreshed	No problem 18 (12.7)
	Slight or mild problem 34 (23.9)
	Moderate problem 54 (38.0)
	Severe problem 36 (25.4)
Pain in the lower abdomen [†]	Present 100 (70.4)
	Absent 42 (29.6)
Depression [‡]	Present 78 (54.9)
	Absent 63 (44.4)
Headaches [†]	Present 118 (83.1)
	Absent 24 (16.9)
Subcategorization of generalized pain type [‡] [n (%)]	Type A 27 (67.5)
	Type B 13 (32.5)

[†] Presence in the preceding six months.

[‡] Type A Criteria = WPI $\geq 7/19$ and SSS $\geq 5/12$ /Type B Criteria = WPI score between 3–6/19 and SSS $\geq 9/12$.

Table 3

Correlations of pelvic pain presence and severity, and meeting the generalized persistent pain threshold (N = 141).

Presence of pelvic pain [n (%)]	Meeting threshold	Not meeting threshold	Totals	Chi Square Statistic Value [X (2)]	P Value	Odds Ratio	95% CI
Pelvic pain present	34 (36.6)	59 (63.4)	93	9.376	0.002	4.130	1.593—10.708
Pelvic pain absent	6 (12.2)	42 (87.5)	48				
Moderate to severe pelvic pain [n (%)]				11.407	0<.001	3.600	1.679—7.718
Moderate to severe pelvic pain	24 (44.4)	30 (55.6)	54				
Moderate to severe pelvic pain absent	16 (18.2)	71 (81.6)	87				
Totals	40	101	141				

the general female Australian population (22%) [38]. Although a significant proportion reported no CPP, the prevalence of moderate to severe CPP and GPP was observed at rates similar to population-based estimates. Comparisons with population-based prevalence estimates should be interpreted cautiously, as differences in study design, sampling strategies, case definitions, and measurement instruments may limit direct comparability. The prevalence of moderate to severe CPP (38.0%) aligns with earlier, smaller retrospective studies in MRKH populations, which have reported rates between 21.2% and 46% [25,39].

In this study, 34.0% reported no CPP over three months. Dysmenorrhoea affects up to 71.7% of adult Australian women and 92% of adolescents [38,40]. Thus, the relatively high proportion of women with MRKH who reported absence of CPP is likely attributable to the absence of menstruation and the associated pro-inflammatory milieu generated during endometrial shedding [41–44]. Nonetheless, moderate to severe CPP was observed in a substantial proportion of participants, underscoring that pelvic pain may occur independently of menstruation. Dyspareunia and sexual function were not included in this study, as these domains require validated psychosexual instruments and have been explored in prior work. [45–47]. Available evidence suggests increased intercourse-related pain and sexual distress in MRKH, along with issues such as vaginismus, however some studies suggest overall reasonable sexual satisfaction. These domains were not included given the brief, community-based survey design focused on pelvic and generalised pain.

We had hypothesised that reduced central sensitisation due to the lack of cyclical inflammatory cytokine release would translate into a lower prevalence of GPP among women with MRKH [41–44], however, a notable proportion of participants reported experiencing GPP. Amongst participants, 3.52% had a diagnosis of fibromyalgia, comparable to global prevalence estimates in women (2.40%–6.80%) [43,48]. Symptom burden from fatigue, depressive symptoms, and widespread pain was also prevalent.

Severe CPP in women with MRKH has previously been reported predominantly in those presenting with obstructed uterine remnants [25,44]. Management of uterine remnants in MRKH is not standardised and is typically individualised, with approaches including hormonal suppression and surgical intervention. We were not able to determine the contribution of uterine remnants to pain, as uterine remnant status was self-reported and not clinically verified, and detailed phenotyping (including functionality, obstruction, or prior excision) was not collected.

Nonetheless, the observed prevalence of CPP suggests that multiple mechanisms may contribute to pain in women with MRKH. Potential contributors include ovarian, gastrointestinal, musculoskeletal, and psychological factors. Given the reliance on self-reported pain location, some symptoms described as pelvic pain may also reflect abdominal or gastrointestinal sources. MRKH-associated renal tract anomalies may increase the risk of urinary tract infections, while vertebral anomalies such as scoliosis (reported in 22.0–42.0%) [45,46] can contribute to musculoskeletal pain. Lower rates of combined oral contraception in this population may also predispose to ovulation pain. In individuals reporting cyclical pain, possible mechanisms described in the literature

include functional uterine remnants or ovulation-related pain [5,44]. Pelvic floor hypertonicity is another well-documented contributor to CPP [48]. The notable prevalence of CPP in this study may also be influenced by vaginal dilator use and the subsequent development of musculoskeletal CPP. While previous studies of dilator therapy have reported favourable outcomes in terms of vaginal length, treatment satisfaction, and sexual pain, they have not specifically focused on CPP [19,49,50].

The prevalence of uterine remnants (24.5%) was consistent with previously reported rates of 10.0–48.0% [4,26]. However, uterine remnant status was self-reported and not confirmed through imaging or medical records. As this was an anonymous survey, we were unable to verify remnant presence, functionality, or prior surgical excision. We did not specifically collect data regarding functional or obstructed remnants, previous excision or endometriosis. Accordingly, remnant status in this study should be interpreted as a broad self-reported characteristic rather than a clinically validated explanatory variable.

The findings of our study align with current literature that GPP is heightened in the context of CPP through central sensitisation mechanisms, particularly during periods of physiological and psychological stress [38]. A key psychological factor in the high rates of CPP and GPP may be the impact of the MRKH diagnosis itself, and any potential negative experiences associated with surgery or vaginal dilator use [12,13].

In this study, 55.2% of participants reported depressive symptoms in the past six months; lower than previously reported rates of 71.7–75.2% [51,52]. The higher median study population age (32 years) compared with those studies (25.78 [51] and 18.83 years) [52] may partly account for this difference. Psychological distress in MRKH has been linked to negative self-evaluation of femininity [53], and to ALEs, both of which are associated with heightened pain perception. While literature supports a relationship between depression and CPP, it remains unclear whether pain precipitates mood dysregulation or pre-existing depressive symptoms amplify pain perception [54–56]. Among participants, younger participants reported a greater burden of somatic symptoms impacting quality of life (QOL). This aligns with prior findings that women more recently diagnosed with MRKH experience higher emotional distress, with consequent effects on QOL and sexual function [51], underscoring the importance of ongoing screening for mood symptoms and QOL in, particularly in the years immediately following diagnosis. However, higher rates of psychological distress are also well described in younger individuals in the general population, including in Australia, and this may partly contribute to the observed age-related differences in this study [57]. Depressive symptoms were assessed using a single self-reported item rather than a validated depression screening instrument. This limits the ability to determine severity, chronicity, or clinical diagnosis, and may result in over- or under-estimation of depressive symptom burden.

This study had several strengths and limitations. To our knowledge, it is the first survey from a large sample of individuals with MRKH using validated pain questionnaires, with active engagement of stakeholder groups [27]. However, the survey did not capture detailed pain characteristics (such as cyclical versus non-cyclical pain) or pain associated with neovaginoplasty. In addition, although lower abdominal pain was

assessed separately, participants may not have clearly distinguished between pelvic and abdominal or gastrointestinal pain, which may have resulted in some misclassification of pain location. The survey was designed to estimate the prevalence and severity of pelvic and generalised persistent pain, rather than to provide detailed clinical phenotyping or determine pain aetiology. We did not collect data on prior diagnosis of endometriosis, previous laparoscopic evaluation, urological or gastrointestinal assessment, connective tissue disorders, trauma history, hormonal suppression, prior remnant excision, neovagina creation method or dyspareunia. These factors may contribute to pelvic pain in some individuals with MRKH and warrant exploration in future studies incorporating detailed clinical phenotyping.

Participants were recruited through MRKH Australia's newsletter and social media platforms, and an accurate response rate could not be determined due to overlap between channels. As such, this represents a convenience sample, and representativeness cannot be formally established. In addition, due to the anonymous survey design and absence of technical safeguards (e.g., IP tracking or unique identifiers), duplicate submissions cannot be definitively excluded. Individuals engaged with support networks may differ from the broader MRKH population in symptom burden, health-seeking behaviour, or healthcare access. These factors should be considered when interpreting the generalisability of the findings. The cross-sectional design also precluded longitudinal assessments of symptom trajectories. Finally, recall bias may influence self-reporting of symptoms.

This study provides the first large-scale characterisation self-reported of pelvic and generalised pain in women with MRKH. While participants were more likely to report an absence of CPP compared with the general female population, the prevalence of self-reported moderate to severe CPP and GPP remained common. The present study cannot determine the specific causes of pain in this population, but the findings indicate that clinically significant pain symptoms are not uncommon and warrant recognition. Importantly, CPP was associated with an increased likelihood of GPP, underscoring the complex interplay between chronic pain and psychological wellbeing. Together, these results emphasise the importance of early recognition of pain symptoms in women with MRKH, with integration of holistic, patient-centred strategies addressing both somatic and psychosocial contributors to pain.

CRedit authorship contribution statement

Riya Gaikawari: Writing – original draft, Project administration, Formal analysis, Data curation, Conceptualization. **Sonia R. Grover:** Writing – review & editing, Supervision, Conceptualization. **Ian Wright:** Writing – review & editing, Supervision, Methodology. **Andrew R. Battle:** Formal analysis. **Natalie Drever:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Conceptualization.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Declaration of generative AI and AI-assisted technologies in the preparation of this manuscript

During the preparation of this work, the authors used generative AI tools to assist with refining language and improving clarity. After using these tools, the authors reviewed and edited all content and take full responsibility for the final manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ejogrb.2026.115159>. Study materials and survey tool.

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