Case Report

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Ayurveda Section

Management of Resistant Anovulation with *Uttara Basti* Therapy: A Case Report

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ABSTRACT

Resistant anovulation represents a challenging subset of ovulatory dysfunction in women with primary infertility, particularly when conventional pharmacological interventions fail. The present case report describes a 29-year-old female presenting with treatment-resistant anovulation in the context of polycystic ovarian morphology who achieved successful ovulation and conception following an integrated treatment protocol combining evidence-based traditional medicine with contemporary reproductive endocrinology principles. The patient presented with a four-year history of infertility despite multiple cycles of conventional ovulation-inducing agents, including clomiphene citrate, letrozole and human Menopausal Gonadotropin (hMG). Implementation of a multimodal management strategy including Ayurvedic therapeutic interventions, metabolic optimisation and hormonal monitoring resulted in spontaneous ovulation during the fourth treatment cycle, culminating in uncomplicated pregnancy and continuation to term. This case illustrates the potential therapeutic value of integrating complementary medical approaches with conventional diagnostic monitoring in the management of resistant ovulatory dysfunction.

Keywords: Ayurvedic management, Female infertility, Ovulation induction, Polycystic ovary syndrome

CASE REPORT

A 29-year-old female, married for five years, presented with primary infertility. She measured 158 cm tall, weighed 68 kg {Body Mass Index (BMI) 27.2 kg/m^2, overweight} and had oligomenorrhea with cycle length 35-90 days after menarche.

Three years prior, polycystic ovarian morphology was diagnosed on transvaginal ultrasound. At baseline transvaginal ultrasound was suggestive of ovarian volumes right 12.8 mL and left 11.4 mL and 10-12 peripheral 2-5 mm follicles on transvaginal ultrasound and endometrial thickness was 6.4 mm. Baseline serum hormone analysis documented elevated Luteinising Hormone:Follicle-Stimulating Hormone (LH:FSH) ratio (3.1) characteristic of Polycystic Ovarian Syndrome (PCOS). Semen analysis revealed normal parameters: volume 4 mL, pH 7.3, sperm count 90 million/mL, morphology 56% normal forms, motility 40% and no pus cells. These findings indicate preserved male fertility potential with no evident seminal pathology [Table/Fig-1]. No hirsutism, acne, or clinical hyperandrogenism was noted on examination, total testosterone was mildly elevated at 0.92 ng/mL (reference range 0.15-0.70 ng/mL), representing isolated biochemical elevation without clinical correlation.

Previous investigations	Details	
LH	18.2 mIU/mL	Semen analysis: pH - 7.3 Volume - 4 mL Appearance - Grayish white Sperm Count - 90 million/mL Normal Morphology -56 % Motility -40 % Pus Cells - Nil
FSH	5.8 mIU/mL	
LH/FSH ratio	3.1	
AMH	6.5 ng/mL	
TSH	3.6 µU/mL	
Prolactin	18 ng/mL	
Testosterone	0.92 ng/mL	
HSG	Bilateral tubal patency	

[Table/Fig-1]: Laboratory investigations (Baseline).
AMH:Anti-Müllerian hormone; TSH:Thyroid-stimulating hormone; HSG:Hysterosalpingogram

Investigations such as Dehydroepiandrosterone Sulfate (DHEAS) levels, etc., were not performed as it is not part of routine Ayurvedic clinical assessment for PCOS-like conditions (*Vandhyatva/Artava Dushti*), where clinical signs such as oligomenorrhea, PCOS morphology, basic hormones and ultrasound suffice for diagnosis and monitoring efficacy.

The patient had undergone multiple cycles of ovulation induction therapy including clomiphene citrate and letrozole across four consecutive cycles and human Menopausal Gonadotropin (hMG) after prolonged gap at various clinical settings the details of which are as below [Table/Fig-2]. During clomiphene citrate followed by letrozole induction, despite adequate follicular development to 14-16 mm diameter, follicles failed to mature to pre-ovulatory size (18-20 mm) or rupture. Ultrasound reports from these cycles documented persistent follicular arrest at 14-16 mm with thin endometrium (<8 mm) and absent follicular rupture, confirming failure of preovulatory maturation. Subsequent induction by hMG therapy (75-150 IU daily) was discontinued after two cycles due to inadequate response and elevated costs, after which the patient approached the present clinician for Ayurvedic treatment.

Regimen	Dose & days	Monitoring	Follicular/ endometrial response	Outcome
Clomiphene citrate 4 consecutive cycles	Cycle 1-2 50 mg OD, days 3-7; Cycle 3-4 100 mg OD, days 3-7; Metformin 500 mg BD daily	TVS on days 10, 12, 14, 16	Max follicle size 14-16 mm; no follicles ≥18 mm; endometrium ≤8 mm; no follicular rupture	No ovulation achieved
Letrozole 2 consecutive cycles	5 mg OD, days 3-7	TVS on days 10, 12, 14, 16	Max follicle size 15 mm; no follicular rupture	No ovulation achieved
hMG 2 cycles (after prolonged gap)	Cycle 1 75 IU on day 3,5,7 Cycle 2 150 IU on day 3,5,7	TVS from day 6, then every 48 hr	<2 follicles >14 mm by day 10	Inadequate ovarian response

[Table/Fig-2]: Previous conventional ovulation induction.
TVS:Transvaginal sonography

The previous gynaecologist where hMG induction was given may have considered inadequate response as fewer than two follicles >14 mm by day 10 despite seven days of maximal stimulation, confirming poor ovarian response. The AMH level of 6.5 ng/mL reported 1.5 years prior by the same gynaecologist indicated potential Ovarian Hyperstimulation Syndrome (OHSS) risk during gonadotropin stimulation, which was appropriately managed with cautious low-dose protocols in that setting. The details of eight cycles of ovulation induction received by patient in 18 months are as follows:

The patient was assessed thoroughly by Ayurvedic parameters, few important findings are summarised in [Table/Fig-3]; where Prakriti refers to the patient's inherent constitutional body-mind type identified as *Vata-Kapha* (included among three bio-energies or functional principles), while Vikriti represents the current imbalance or deviation from this natural constitution manifesting as *Apana Vata Dushti* (disorder of the *Vata* subtype responsible for downward movements including menstruation and reproduction) along with *Artavavaha Srotodushti* (obstruction or dysfunction of the channels carrying menstrual blood/*Artava-vaha Srotas*). The clinical diagnosis of *Vandhyatva* (infertility) was attributed to *Beeja Dushti* (defect in ovum/gamete quality) and *Artava Dushti* (vitiation of menstrual blood or ovulatory dysfunction).

Examination of patient	Ayurvedic assessment
Prakriti (Constitution- innate, natural body-mind type)	*Vata-Kapha*
Vikriti (Imbalance- deviation from natural constitution)	*Apana Vata Dushti, Artavavaha Srotodushti*
Diagnosis	*Vandhyatva* possibly under *Beeja Dushti* and *Artava Dushti*

[Table/Fig-3]: Diagnosis as per Ayurveda.

Treatment objectives: The treatment objectives focused on promoting follicular maturation and ovulation while simultaneously enhancing endometrial receptivity to optimise conditions for conception.

Management Strategy and Clinical Course

The integrated protocol was structured around three objectives:

1. Ayurvedic medicines and therapeutic procedures;
2. Metabolic optimisation through dietary and lifestyle modifications;
3. Transvaginal ultrasonographic monitoring.

1. Ayurvedic intervention plan: The *Shodhana* (purificatory) therapy protocol for *Virechana* [Table/Fig-4], which finishes before next menstrual cycle, consisted of preparatory phases of *Deepana-Pachana* with *Shankha Vati* for five days, *Snehapana* (internal oleation) using escalating doses of *Phala Ghrita* over seven days, followed by *Sarvanga Snehana* (full-body massage) and *Swedana* (therapeutic sudation) with *Sahacharadi Taila* for two days, culminating in *Virechana* purgation using Trivrit Leha 30 g on day 1. This sequential detoxification regimen aimed to optimise digestion, clear metabolic toxins and prepare the reproductive system for subsequent *Uttara Basti* cycles.

Treatment cycles commenced post-*Virechana* with *Uttara basti* [Table/Fig-5], an intrauterine instillation of medicated oil/ghee, starting from day 5 post-menstruation for five days under all aseptic conditions. Before starting *Uttara basti* proper written informed consent was obtained documenting procedure explanation, potential risks (lower abdominal pain, bleeding per vagina or infection) and benefits. Strict aseptic protocol followed in the form of pre-procedure perineal scrub with povidone-iodine, all instruments used were autoclaved. Before every cycle any active vaginal/uterine infection was ruled out clinically on speculum examination. The patient was advised to avoid conception during the treatment cycles, as *Uttara basti* is contraindicated in pregnancy to prevent potential harm to the developing embryo or gravid uterus.

Procedure	Medication	Duration	Dose and frequency	Route
Shodhan Chikitsa (Purificatory Detoxification Therapies)				
Deepana and Pachan (Appetite Stimulation And Digestive Fire Enhancement)	*Shankha Vati*	5 days	2 tabs BD	Orally
Snehapana (Internal Oleation Therapy)	*Phala Ghrita*	7 days	Once daily for 7 days in escalating doses: Day 1: 30 mL, Day 2: 60 mL Day 3: 90 mL, Day 4: 120 mL Day 5: 150 mL, Day 6: 180 mL Day 7: 200 mL	Orally
Sarvang Snehan (full-body oil massage) Swedan (therapeutic sudation/ steam)	*Sahacharadi taila*	2 days	Once a day	Massage
Virechana (purificatory detoxification therapy)	*Trivrit Leha*	1 day	30 gm with lukewarm water	Orally

[Table/Fig-4]: Treatment protocol.

Procedure	Medication	Duration	Dose and frequency	Route
Uttara basti	*Rasona Taila*	Consecutive 5 days post-menstruation	5 mL per day	Intrauterine

[Table/Fig-5]: *Uttar basti*.

Uttara basti was complemented by *Shamana Chikitsa* [Table/Fig-6], a pacification regimen featuring oral medications including *Rajapravartini Vati*, *Ashokarishta*, *Pushpadhanwa Rasa*, *Shatavari* and Ashwagandha administered over three months to regulate menstrual cycles, nourish reproductive tissues, promote follicular development and balance hormonal function.

Procedure	Medication	Duration	Dose and frequency	Route
Shamana	*Rajapravartini Vati*	3 months	2 tab BD	Orally
	Ashokarishta	3 months	10 mL BD	Orally
	Pushpadhanwa Rasa	3 months	2 tab BD	Orally
	Shatavari Churna	3 months	3 gm BD	Orally
	Ashwagandha Churna	3 months	3 gm BD	Orally

[Table/Fig-6]: *Shamana Chikitsa* (Pacification treatment).

2. *Pathya* (Lifestyle and dietary modifications): The patient was advised through comprehensive counseling regarding reduced refined carbohydrate intake, increased fibre consumption, regular physical activity of moderate intensity (30 minutes, five days weekly), stress reduction and sleep optimisation [Table/Fig-7].

Particulars	Details
For mental health	*Pranayamas* (Anulom-Vilom, Bhramari), sleep optimisation
Dietary advice	*Mudga* (green gram), *Yava* (barley), *Shigru* (drumsticks), Cow milk
Yoga practices	*Surya namaskara*, *Katichakrasana*, *Marjaryasana*, *Gomukhasana*, *Baalasana*, *Vajrasana*, *Setubandhasana*, *Bhujangasana*, *Dhanurasana*, *Naukasana*, *Baddha Konasana*

[Table/Fig-7]: Pathya (Lifestyle and Dietary Modifications).

3. Monitoring protocol: Monitoring protocol involved serial transvaginal ultrasound evaluations conducted at 48-hour intervals to assess follicular development, endometrial thickness progression and ovulation confirmation through serial imaging, ensuring treatment efficacy and safety throughout the therapeutic cycles [Table/Fig-8].

Serial transvaginal ultrasound evaluation (at 48-hour intervals)	Commencing from day 8 till ovulation or day 18 of every cycle

[Table/Fig-8]: Monitoring protocol.

Safety Monitoring and Adverse Event Surveillance

The patient was monitored after every setting of *Uttara basti* for adverse events like lower abdominal pain, bleeding P/V or infection. Also the patient was asked to consult if she observes any symptoms like nausea, vaginal discharge and allergic reactions; none of them were observed throughout the treatment. Liver and renal function tests were not performed as per routine Ayurvedic clinical practice, since clinician did not intend to continue these herbo-mineral medications for the long-term. Also, drugs used have established safety profiles of Good Manufacturing Practice (GMP) -certified medications. All Ayurvedic procedures and treatment protocol remained complication-free.

Outcome and Clinical Resolution

By the end of three consecutive treatment cycles, objective parameters showed progressive improvement from baseline, including normalisation of cycle length, step-wise enhancement of endometrial thickness and gradual attainment of pre-ovulatory follicle size across cycles 1-3. The treatment-response can be summarised as, with cycle 1 showing only menstrual regularisation, cycle 2 demonstrating improved folliculogenesis without ovulation and cycle 3 achieved near-optimal follicular and endometrial development with a dominant follicle reaching pre-ovulatory size, but no sonographic evidence of ovulation was observed. During the fourth cycle, transvaginal ultrasound demonstrated follicular development to 20 mm diameter with appropriate endometrial proliferation (9-10 mm). Serial imaging confirmed follicular rupture with disappearance of the follicle and minimal free fluid in the pouch of Douglas, consistent with spontaneous ovulation on cycle day 15 [Table/Fig-9].

Treatment cycle	Cycle length (days)	Dominant follicle (mm)	Endometrial thickness (mm)	Ovulation	Outcome
Baseline	45-90	None	6.4	No	Anovulatory
1	38	None	7	No	No response
2	35	16	8	No	Follicular growth improved no ovulation
3	35	18-20	9	No	Improved no ovulation
4	32	20 mm (on Day 14) (rupture on Day 15)	10	Yes	Conception

[Table/Fig-9]: Outcome and clinical resolution.

Serum beta-hCG measured on day 16 of fourth cycle following ovulation (187 mIU/mL) confirmed pregnancy. Transabdominal ultrasound at seven weeks gestation documented single intrauterine pregnancy with normal embryonic heart rate (155 bpm). Pregnancy progressed uneventfully to full-term (39 weeks gestation) vaginal delivery of a healthy 3.1 kg neonate without maternal complications.

DISCUSSION

Infertility has gained more concern among individuals of reproductive age. Anovulation is responsible for around 25-40% of female infertility [1]. PCOS constitutes the most prevalent endocrine disorder associated with anovulation. While conventional ovulation induction with clomiphene citrate, letrozole and gonadotropins remains first-line management, treatment resistance occurs in approximately 20-25% of PCOS patients [2]. This resistance is frequently associated with chronic hyperinsulinemia, elevated luteinising hormone levels, obesity and disrupted hypothalamic-pituitary-ovarian axis signalling.

Ayurvedic medicine conceptualises reproductive dysfunction through distinct physiological frameworks. The Sushruta Samhita describes conception as dependent on four essential elements: temporal factors (*Ritu*), anatomical structure (*Kshetra*), nutritive medium (*Ambu*) and reproductive gametes (*Beeja*) [3].

In the present case, the integrated use of *Shodhana*, *Uttara Basti* and *Shamana Chikitsa* is consistent with previous Ayurvedic research work on infertility and anovulation. A similar protocol of Panchakarma with *Uttara Basti* and oral formulations (*Pushpadhanwa Rasa*, *Phala Ghrita*) led to conception within three cycles in a primary infertility case reported by Rani V et al., [4]. A systematic review by Bhadargade P et al., highlighted *Uttara Basti* as a key modality in female infertility, particularly in tubal block and anovulatory pathology, with multiple trials demonstrating improved conception rates [5]. In another case, *Virechana* Karma followed by *Shamana* therapies successfully restored ovulation in a PCOS patient with primary infertility, leading to confirmed conception in the post-treatment cycle without serious complications [6], supporting the current protocol's rationale and outcomes.

The present case demonstrates the potential value of integrative therapeutic approaches in managing resistant anovulation. Success may be attributed to multiple synergistic factors. Post-intervention hormone levels were not formally re-evaluated; however, hormonal reset was inferred from clinical signs, including menstrual cycle regularisation and ultrasound findings indicative of improved folliculogenesis and enhanced uterine receptivity. *Uttar Basti* with *Rasona Taila* provides benefits with properties helping to remove obstruction and enhance ovarian function [7].

Shodhana Chikitsa addressed pathophysiological obstruction: *Phala Ghrita* enhanced tissue nourishment through lipophilic carrier mechanisms while promoting hormonal balance [8]; *Trivrit Leha* via *Virechana* normalised Agni and eliminated metabolic toxins [9]; *Uttara Basti* with *Rasona Taila* directly stimulated endometrial receptivity through Lekhana effects, pacified *Vata* dysfunction and improved Hypothalamic-Pituitary-Ovarian (HPO) axis signalling [7].

Shamana Chikitsa provided sustained hormonal optimisation: *Rajapravartini Vati* regulated menstrual induction through *Artava-vaha Srotas* modulation [10]; *Ashokarishta* enhanced endometrial receptivity [11]; *Pushpadhanwa Rasa* rejuvenated reproductive tissues [12]; *Shatavari* promoted follicular maturation through phytoestrogens (*Shatavarins*) regulating HPO axis [13]; *Ashwagandha* (*Withania somnifera*) modulated HPA axis, reducing cortisol-mediated reproductive suppression [14].

Uttara Basti procedure demonstrated acceptable tolerability in this case with no adverse events reported. The patient underwent comprehensive monitoring with no evidence of any procedural complications, consistent with literature on similar protocols. Ayurvedic medications were well-tolerated at prescribed doses without any signs and symptoms of hepato-renal toxicity.

Metabolic optimisation through dietary modification and exercise has been demonstrated in multiple studies to reduce insulin resistance, decrease androgen levels and improve ovulatory function in PCOS patients (independent of substantial weight loss) [15]. Integration of traditional techniques with contemporary diagnostic monitoring created a distinctive clinical approach.

The elevated baseline LH:FSH ratio presented a known barrier to ovulation induction. The 16-day interval from detected ovulation to positive pregnancy serology and uncomplicated pregnancy course

suggest restoration of normal reproductive physiology rather than merely pharmacologically induced ovulation.

Clinical considerations: Single case reports provide low-level evidence and do not permit causal inference. Confounding variables, including patient compliance with lifestyle modifications and passage of time, could have influenced results. Placebo effects and regression to the mean cannot be excluded. Future studies should incorporate comprehensive hormonal assessments (testosterone, DHEAS, LH surge, mid-luteal progesterone, repeat LH:FSH) alongside clinical and ultrasonographic parameters to provide objective verification of endocrine normalisation while following such resistant anovulation treatment protocols.

CONCLUSION(S)

Resistant anovulation in the context of polycystic ovarian morphology represents a complex reproductive challenge. The present case illustrates that integrating traditional therapeutic approaches with evidence-based modern reproductive medicine and systematic clinical monitoring may offer viable management options. Rigorous prospective clinical trials comparing integrated protocols with conventional management are warranted to establish efficacy and elucidate mechanisms. Patient-centered care combining effective elements of both traditional and contemporary medical science offers promise for improving outcomes in resistant ovulatory dysfunction.

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