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A Comprehensive Review on Empty Sella Syndrome

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ABSTRACT: This review explores Empty Sella Syndrome (ESS), a clinically significant endocrine and neurological disorder characterized by partial or complete filling of the sella turcica with cerebrospinal fluid, resulting in compression and flattening of the pituitary gland. ESS is increasingly recognized due to advances in magnetic resonance imaging (MRI), which has improved early diagnosis and identification of associated hormonal abnormalities. The condition is classified into primary and secondary types depending on its etiology and may present with neurological, endocrine, reproductive, and visual manifestations. Common clinical features include headache, visual disturbances, hypopituitarism, hypothyroidism, adrenal insufficiency, hyperprolactinemia, infertility, and menstrual irregularities. Recent studies emphasize that ESS should not be considered merely an incidental radiological finding, as many patients develop progressive endocrine dysfunction requiring long-term follow-up. Diagnosis mainly involves MRI evaluation, hormonal assessment, and ophthalmological examination. Current management focuses on individualized hormone replacement therapy, management of intracranial hypertension, and monitoring of complications. Recent advances also highlight the importance of multidisciplinary care involving endocrinologists, neurologists, radiologists, and neurosurgeons. Overall, this review summarizes the classification, pathophysiology, clinical manifestations, diagnosis, treatment, and recent advances in Empty Sella Syndrome while emphasizing the importance of early detection and personalized management strategies.

KEYWORDS: Empty Sella Syndrome, Pituitary gland, Hypopituitarism, MRI, Hormone replacement therapy, Intracranial hypertension

I. INTRODUCTION:

The pituitary gland is a small endocrine organ situated within the sella turcica of the sphenoid bone at the base of the brain. Despite its small size, it plays a major role in regulating several physiological and hormonal activities of the body and is therefore commonly referred to as the “master gland.” The gland is anatomically connected to the hypothalamus through the pituitary stalk and is divided into anterior and posterior lobes. The anterior pituitary secretes hormones such as growth hormone (GH), thyroid-stimulating hormone (TSH), adrenocorticotropic hormone (ACTH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), and prolactin, while the posterior pituitary stores and releases vasopressin and oxytocin.[1] These hormones are essential for maintaining metabolism, growth, stress response, reproduction, lactation, and water balance within the body.[2]

Empty Sella Syndrome (ESS) is a condition characterized by partial or complete herniation of cerebrospinal fluid (CSF) into the sella turcica, resulting in flattening and compression of the pituitary gland against the sellar floor.[3] Radiologically, the sella turcica appears “empty” because the pituitary gland becomes compressed and difficult to visualize clearly on imaging studies. However, the gland is usually still present in a flattened form. ESS was initially considered an incidental radiological finding with minimal clinical significance, but recent studies have shown that the condition may be associated with several endocrine, neurological, ophthalmological, and reproductive abnormalities.[4]

The condition is broadly classified into primary and secondary forms. Primary Empty Sella Syndrome (PES) occurs due to congenital incompetence or absence of the diaphragma sellae,

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which permits herniation of the subarachnoid space and CSF into the sellar cavity. Increased intracranial pressure further contributes to progressive enlargement of the sella turcica and compression of the pituitary gland.[5] Secondary Empty Sella Syndrome (SES), on the other hand, develops due to destruction or shrinkage of pituitary tissue following pituitary surgery, radiation therapy, pituitary tumors, infarction, hemorrhage, trauma, or infections.[6]

The prevalence of ESS has increased significantly with the widespread use of advanced neuroimaging techniques such as magnetic resonance imaging (MRI). MRI is currently considered the gold standard diagnostic modality because it clearly demonstrates the presence of cerebrospinal fluid within the sellar cavity and flattening of the pituitary gland.[4] Studies suggest that ESS is more frequently observed in women, especially obese middle-aged females between 40 and 60 years of age. Obesity, hypertension, multiple pregnancies, and idiopathic intracranial hypertension (IIH) are considered important risk factors associated with the development of ESS.[7] Recent literature also demonstrates a strong association between ESS and elevated intracranial pressure, particularly among obese women presenting with chronic headache and visual disturbances.[8]

Clinically, ESS presents with a broad spectrum of manifestations ranging from asymptomatic incidental findings to severe endocrine dysfunction. Neurological symptoms such as chronic headache, dizziness, fatigue, and cognitive disturbances are commonly reported. Visual symptoms may occur due to compression or prolapse of the optic chiasm and include blurred vision, diplopia, and visual field defects.[9] Endocrine abnormalities are also frequently observed and may involve hypopituitarism, hyperprolactinemia, hypothyroidism, adrenal insufficiency, growth hormone deficiency, gonadal dysfunction, infertility, menstrual irregularities, and sexual dysfunction.[10] In some patients, pituitary hormonal abnormalities may progress gradually over time, emphasizing the importance of long-term follow-up and endocrine monitoring.

The pathophysiology of ESS mainly involves defects in the diaphragma sellae combined with altered cerebrospinal fluid dynamics. Herniation of CSF into the sella turcica exerts pressure on the pituitary gland, causing flattening of the gland and elongation of the pituitary stalk. Compression of the pituitary stalk may interfere with hypothalamic inhibitory control over prolactin secretion, leading to hyperprolactinemia.[11] Similarly, compression of the gland itself may impair secretion of pituitary hormones, resulting in varying degrees of hypopituitarism. In secondary ESS, pituitary tissue loss following surgery, infarction, radiation therapy, or hemorrhage is the major underlying mechanism.[6]

Diagnosis of ESS requires a multidisciplinary approach involving clinical evaluation, radiological imaging, endocrine testing, and ophthalmological assessment. MRI remains the preferred diagnostic modality because it can differentiate between partial and complete ESS and detect associated abnormalities such as pituitary adenomas, optic chiasm prolapse, and pituitary stalk deviation.[4] Hormonal evaluation is essential even in asymptomatic patients because many individuals develop subtle endocrine dysfunction during the course of the disease.[8]

Management of ESS primarily depends on the patient's clinical presentation and hormonal status. Asymptomatic individuals with normal pituitary function usually require only regular follow-up and monitoring. Hormone replacement therapy remains the cornerstone of treatment in patients with endocrine deficiencies. Levothyroxine is used for hypothyroidism, hydrocortisone for adrenal insufficiency, sex hormone replacement for hypogonadism, and desmopressin for diabetes insipidus.[12] Dopamine agonists such as cabergoline and bromocriptine are commonly used in patients with hyperprolactinemia. Surgical intervention is rarely required but may be indicated in cases complicated by cerebrospinal fluid rhinorrhea or severe visual impairment.[13]

Recent advances in the understanding of ESS emphasize that the condition should not be regarded merely as a radiological finding but as a clinically significant endocrine disorder requiring individualized treatment and long-term monitoring. Current research focuses on improved MRI-based diagnosis, routine endocrine screening, personalized hormone replacement therapy, and multidisciplinary management involving endocrinologists, neurologists, ophthalmologists, radiologists, and neurosurgeons.[8] These advances have improved early diagnosis, patient outcomes, and quality of life in individuals affected by Empty Sella Syndrome.

CLASSIFICATION:

Empty Sella Syndrome (ESS) is broadly classified into primary and secondary forms depending on its etiology and pathological mechanism. Based on radiological appearance, it may also be categorized into partial and complete empty sella.[5] [6]

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1. Primary Empty Sella Syndrome (PES)

Primary Empty Sella Syndrome occurs due to congenital incompetence, weakness, or absence of the diaphragma sellae, which permits herniation of the subarachnoid space and cerebrospinal fluid (CSF) into the sellar cavity.[5] As the CSF enters the sella turcica, pressure is exerted on the pituitary gland, causing flattening and compression of the gland against the sellar floor. Increased intracranial pressure further contributes to progressive enlargement of the sella turcica and pituitary compression.[8]

Primary ESS is more commonly observed in obese middle-aged women and is strongly associated with idiopathic intracranial hypertension (IIH), hypertension, and multiple pregnancies.[7] Many patients remain asymptomatic, while others may present with headache, endocrine abnormalities, visual disturbances, and reproductive dysfunction.[9]

2. Secondary Empty Sella Syndrome (SES)

Secondary Empty Sella Syndrome develops as a result of destruction, shrinkage, or regression of pituitary tissue following pathological or therapeutic interventions.[6] Common causes include:

- Pituitary adenoma
- Pituitary surgery
- Radiation therapy
- Traumatic brain injury
- Pituitary infarction
- Sheehan syndrome
- Pituitary apoplexy
- Infections affecting the pituitary gland

In secondary ESS, the sellar cavity becomes filled with CSF after loss of pituitary tissue volume. Unlike primary ESS, the underlying pathology directly damages the pituitary gland, resulting in a higher incidence of hypopituitarism and severe endocrine dysfunction.[3]

3. Partial Empty Sella

Partial Empty Sella refers to incomplete filling of the sella turcica with cerebrospinal fluid. In this condition, less than 50% of the sellar cavity is occupied by CSF, and a significant portion of the pituitary gland remains visible on MRI.[4] Hormonal function may remain normal in many patients, although mild endocrine abnormalities can occur.

MRI findings in partial ESS include:
Mild enlargement of sella turcica
Partial CSF filling
Slight flattening of pituitary tissue
Minimal displacement of pituitary stalk

Partial ESS is often detected incidentally during neuroimaging performed for unrelated neurological symptoms.[9]

4. Complete Empty Sella

Complete Empty Sella occurs when more than 50% of the sella turcica is filled with cerebrospinal fluid, resulting in marked compression and thinning of the pituitary gland.[4] The gland appears as a thin rim of tissue along the sellar floor on MRI examination.

Patients with complete ESS are more likely to develop:
Hypopituitarism
Hyperprolactinemia
Visual disturbances
Chronic headache
Reproductive dysfunction

Advanced MRI imaging helps distinguish complete ESS from pituitary tumors and other sellar abnormalities.[8]

➢ Radiological Classification

Based on MRI appearance, ESS can also be radiologically classified into:

- Partial ESS: Pituitary thickness ≥3 mm with partial CSF filling.
- Complete ESS: Pituitary thickness <2–3 mm with almost total CSF filling of the sellar cavity.[4]

This radiological classification is clinically important because complete ESS is associated with a greater risk of endocrine dysfunction and neurological complications.[8]

EPIDEMIOLOGY AND RISK FACTORS:

Empty Sella Syndrome (ESS) is increasingly diagnosed due to the widespread use of MRI imaging. The condition is more common in women, especially obese middle-aged females between 40 and 60 years of age.[4] [8] Many cases are identified incidentally during neuroimaging for headache or other neurological complaints.[9]

Major risk factors associated with ESS include:
Female gender
Obesity
Multiple pregnancies
Idiopathic intracranial hypertension
Hypertension
Pituitary surgery
Radiation therapy
Traumatic brain injury[3] [6] [7]

Recent studies show a strong association between ESS and elevated intracranial pressure,

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particularly among obese women with chronic headache and visual symptoms.[8]

❖ Clinical Cases Associated with ESS

A 45-year-old obese woman with chronic headache and visual disturbances was diagnosed with Primary Empty Sella Syndrome during MRI evaluation.[8]

A patient developed Secondary Empty Sella Syndrome following pituitary adenoma surgery and later presented with hypopituitarism and hormonal imbalance.[6]

An asymptomatic female undergoing MRI for dizziness was incidentally found to have partial empty sella with normal hormonal function.[9]

PATHOPHYSIOLOGY:

The pathophysiology of Empty Sella Syndrome (ESS) primarily involves abnormalities of the diaphragma sellae and altered cerebrospinal fluid (CSF) dynamics within the sellar region. The diaphragma sellae is a small dural membrane covering the sella turcica and separating the pituitary gland from the subarachnoid space. In primary ESS, congenital weakness, incompetence, or absence of this membrane permits herniation of the subarachnoid space into the sella turcica.[5]

As cerebrospinal fluid enters the sellar cavity, pressure is exerted on the pituitary gland, leading to progressive flattening and compression of pituitary tissue against the sellar floor.[4] Increased intracranial pressure further worsens pituitary compression and contributes to enlargement of the sella turcica.[8] The pituitary stalk may also become elongated and thinned, interfering with hypothalamic–pituitary communication pathways.[11]

Compression of the pituitary stalk disrupts dopaminergic inhibition of prolactin secretion, leading to hyperprolactinemia.[11] Similarly, compression of the anterior pituitary may impair secretion of hormones such as:

ACTH,TSH,GH,LH and FSH

This results in varying degrees of hypopituitarism and endocrine dysfunction.[10]

Recent studies strongly associate ESS with idiopathic intracranial hypertension (IIH), particularly in obese middle-aged women. Elevated intracranial pressure contributes to chronic CSF pulsations into the sellar cavity, causing progressive pituitary remodeling and sellar enlargement.[8]

In secondary ESS, the mechanism differs because pituitary tissue loss occurs following:

- Pituitary surgery
- Radiation therapy
- Pituitary infarction
- Pituitary apoplexy
- Trauma
- Tumor regression

Loss of pituitary tissue creates an empty space within the sella turcica, which subsequently becomes filled with cerebrospinal fluid.[6] Secondary ESS is more frequently associated with severe hypopituitarism because of direct pituitary gland destruction.[3]

Chronic compression and hormonal dysfunction may eventually produce neurological, visual, reproductive, and metabolic complications if left untreated.[4]

SIGNS AND SYMPTOMS:

Clinical manifestations of Empty Sella Syndrome vary widely from asymptomatic incidental findings to severe endocrine and neurological disturbances. Many patients are diagnosed incidentally during MRI performed for unrelated complaints.[9]

➢ Neurological Symptoms

Neurological manifestations are among the most common presentations of ESS and include:
Chronic headache,Dizziness,Fatigue,Weakness

Headache is considered the most frequently reported symptom, particularly in patients with associated intracranial hypertension.[8]

➢ Visual Symptoms

Visual abnormalities occur due to compression or prolapse of the optic chiasm and may include:
Blurredvision,Diplopia,Reduced visual acuity,Visual field defects,Bitemporal hemianopia

Severe visual impairment is uncommon but may occur in advanced cases.[4]

➢ Endocrine Symptoms

Hormonal dysfunction is common in ESS and may involve one or multiple pituitary hormone deficiencies. Common endocrine abnormalities include:

- Hypothyroidism
- Adrenal insufficiency
- Growth hormone deficiency
- Hyperprolactinemia
- Hypogonadism

Patients may experience:

- Cold intolerance
- Weight gain
- Reduced stress tolerance
- Weakness

➢ Reproductive Symptoms

Reproductive manifestations are particularly common in women and include:

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- Amenorrhea
- Menstrual irregularities
- Infertility
- Galactorrhea

Men may develop:

- Erectile dysfunction
- Decreased libido
- Infertility[3]

General Symptoms

Additional symptoms may include:

- Obesity
- Sleep disturbances
- Anxiety
- Depression

Some patients remain completely asymptomatic despite significant radiological findings.[9]

DIAGNOSIS:

Diagnosis of Empty Sella Syndrome (ESS) requires a combination of clinical evaluation, radiological imaging, hormonal assessment, and ophthalmological examination. Many cases are diagnosed incidentally during neuroimaging performed for unrelated neurological complaints such as chronic headache or dizziness.[4]

➢ Clinical Evaluation

Initial diagnosis begins with detailed clinical history and physical examination. Physicians evaluate symptoms related to:

- Endocrine dysfunction
- Neurological abnormalities
- Visual disturbances
- Reproductive disorders

Common presenting complaints include:

Chronic headache
Fatigue
Menstrual irregularities
Infertility
Visual defects
Weakness
Reduced libido[9]

Assessment of obesity, hypertension, and features of idiopathic intracranial hypertension is also important because these conditions are commonly associated with ESS.[7]

➢ Magnetic Resonance Imaging (MRI)

Magnetic Resonance Imaging (MRI) is considered the gold standard investigation for diagnosing Empty Sella Syndrome because it provides detailed visualization of the sellar region and pituitary gland.[4]

Typical MRI findings include:

- Enlargement of the sella turcica
- Partial or complete filling of the sella with cerebrospinal fluid
- Flattened pituitary gland along the sellar floor
- Thin elongated pituitary stalk
- Herniation of suprasellar subarachnoid space into the sella[5]

MRI also helps differentiate:

- Primary and secondary ESS
- Partial and complete ESS
- Pituitary adenoma
- Other sellar abnormalities[8]

➢ Computed Tomography (CT Scan)

Computed tomography may be used when MRI is unavailable or contraindicated. CT scan can demonstrate:

- Enlarged sella turcica
- Sellar bone erosion
- CSF-filled sellar cavity

However, CT is less sensitive than MRI for evaluating soft tissue structures of the pituitary gland.[6]

➢ Hormonal Evaluation

Comprehensive endocrine assessment is essential because many patients with ESS develop pituitary hormone abnormalities.[4] Hormonal investigations include:

Anterior Pituitary Hormone Assessment

- Thyroid-stimulating hormone (TSH)
- Free thyroxine (T4)
- Adrenocorticotropic hormone (ACTH)
- Serum cortisol
- Growth hormone (GH)
- Insulin-like growth factor-1
- Follicle-stimulating hormone (FSH)
- Prolactin[8]

Posterior Pituitary Function

Posterior pituitary involvement may lead to diabetes insipidus, requiring evaluation of:

- Serum osmolality
- Urine osmolality
- Serum sodium levels[11]

Hormonal abnormalities commonly detected in ESS include:

- Hyperprolactinemia
- Hypogonadism
- Growth hormone deficiency
- Adrenal insufficiency[10]

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➢ Ophthalmological Examination

Visual assessment is important in patients presenting with visual symptoms or suspected optic chiasm involvement.[4] Ophthalmological evaluation includes:

- Visual acuity testing
- Visual field examination
- Fundoscopy
- Optic nerve assessment

Visual field defects such as bitemporal hemianopia may occur due to compression or prolapse of the optic chiasm.[9]

➢ Differential Diagnosis

ESS should be differentiated from other sellar and parasellar lesions including:

- Pituitary adenoma
- Rathke cleft cyst
- Craniopharyngioma
- Sellar tumors[5]

Accurate differentiation is essential for selecting appropriate treatment and avoiding unnecessary surgical intervention.

Figure 1:Normal Sagittal MRI of the pituitary gland:

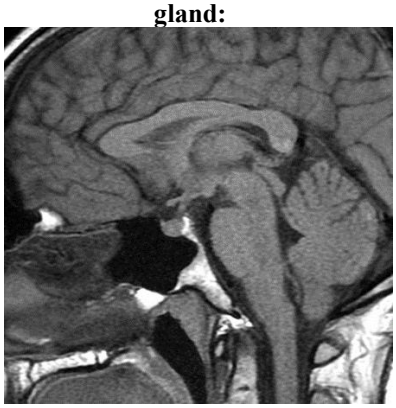
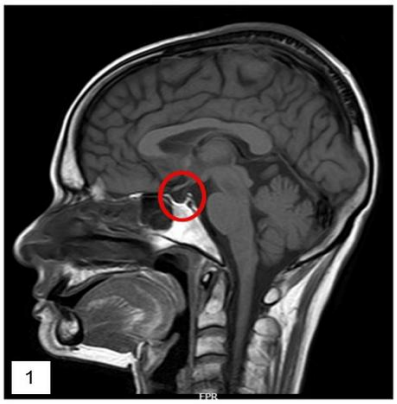


Figure 2: Sagittal MRI Showing Empty Sella Syndrome with shrinken pituitary gland



TREATMENT:

Management of Empty Sella Syndrome depends on the patient's symptoms, hormonal status, and associated complications.[3]

➢ Observation and Follow-Up

Asymptomatic patients with normal pituitary function generally do not require active treatment. However, regular follow-up is essential because hormonal abnormalities may develop later.[4] Follow-up includes:

- Periodic MRI examination
- Hormonal reassessment
- Visual field evaluation

➢ Hormone Replacement Therapy

Hormone replacement therapy remains the cornerstone of ESS management and is individualized according to the deficient endocrine axis.[8]

- Thyroid Hormone Replacement

Levothyroxine is administered in patients with central hypothyroidism to restore normal thyroid hormone levels.[12]

- Glucocorticoid Replacement

Hydrocortisone or prednisolone is used in adrenal insufficiency caused by ACTH deficiency. Early treatment is important because untreated cortisol deficiency may become life-threatening.[3]

- Gonadal Hormone Replacement

Estrogen-progesterone therapy is used in females with hypogonadism.

Testosterone replacement is used in males with androgen deficiency.[10]

- Desmopressin Therapy

Desmopressin is indicated in patients who develop diabetes insipidus due to posterior pituitary involvement.[11]

- Growth Hormone Therapy

Growth hormone replacement may be considered in selected patients after detailed endocrine evaluation.[10]

- Management of Hyperprolactinemia

Hyperprolactinemia is treated using dopamine agonists such as:

Cabergoline,Bromocriptine

These drugs help normalize prolactin levels and improve reproductive symptoms such as amenorrhea, galactorrhea, and infertility.[3]

- Management of Intracranial Hypertension

Patients with associated idiopathic intracranial hypertension may require:

Weight reduction
Lifestyle modification
Acetazolamide therapy

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Blood pressure control[8]

- Surgical Management

Surgery is rarely required but may be indicated in:

Cerebrospinal fluid rhinorrhea
Severe visual disturbances
Progressive neurological deficits

Transsphenoidal surgical repair is commonly performed in such cases.[13]

RECENT ADVANCES:

Recent advances in Empty Sella Syndrome focus on improved diagnostic techniques, endocrine evaluation, and individualized treatment strategies.[4 8]

1. Improved MRI-Based Diagnosis

High-resolution MRI has significantly improved the diagnostic accuracy of ESS. Advanced sellar imaging helps distinguish:

- Partial and complete ESS
- Primary and secondary ESS
- Associated pituitary abnormalities
- Optic chiasm prolapse
- Pituitary stalk deviation[8]

MRI is now considered the gold standard investigation for evaluating sellar pathology.

2. Routine Endocrine Screening

Recent studies recommend routine hormonal assessment even in asymptomatic patients because many individuals gradually develop pituitary dysfunction over time.[4]

Common abnormalities identified through screening include:

- ACTH deficiency
- TSH deficiency
- Gonadal dysfunction
- Growth hormone deficiency
- Hyperprolactinemia

3. Association with Idiopathic Intracranial Hypertension

Current literature strongly links ESS with idiopathic intracranial hypertension, particularly in obese middle-aged women presenting with chronic headache and visual symptoms.[8] This has changed clinical management strategies by emphasizing:

Weight reduction
Intracranial pressure control
Ophthalmological follow-up

4. Personalized Hormone Replacement Therapy

Modern treatment approaches focus on individualized hormone replacement rather than treating ESS itself. Hormonal therapy is tailored according to specific endocrine deficiencies, improving long-term prognosis and quality of life.[3]

5. Multidisciplinary Management

Recent perspectives emphasize multidisciplinary care involving:

Endocrinologists
Neurologists
Ophthalmologists
Neurosurgeons
Radiologists[8]

This approach improves early diagnosis, monitoring, and management of complications.

6. Better Long-Term Follow-Up

Even asymptomatic patients are now advised to undergo:

- Regular MRI monitoring
- Periodic endocrine evaluation
- Visual assessment

Long-term follow-up helps prevent delayed complications and progressive pituitary dysfunction.[4]

II. CONCLUSION:

Empty Sella Syndrome is an important endocrine and neurological disorder characterized by herniation of cerebrospinal fluid into the sella turcica, resulting in compression and flattening of the pituitary gland. Although many patients remain asymptomatic, others develop significant endocrine, neurological, reproductive, and visual abnormalities that may adversely affect quality of life.[3]

MRI remains the gold standard diagnostic modality, while hormonal evaluation plays an essential role in identifying pituitary dysfunction. Hormone replacement therapy remains the cornerstone of management and should be individualized according to the deficient hormonal axis.[8]

Recent advances emphasize that ESS should not be regarded merely as an incidental radiological finding but as a clinically significant disorder requiring multidisciplinary management and long-term follow-up. Early diagnosis, routine endocrine screening, personalized treatment strategies, and regular monitoring significantly improve patient outcomes and reduce complications.[4 8]

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