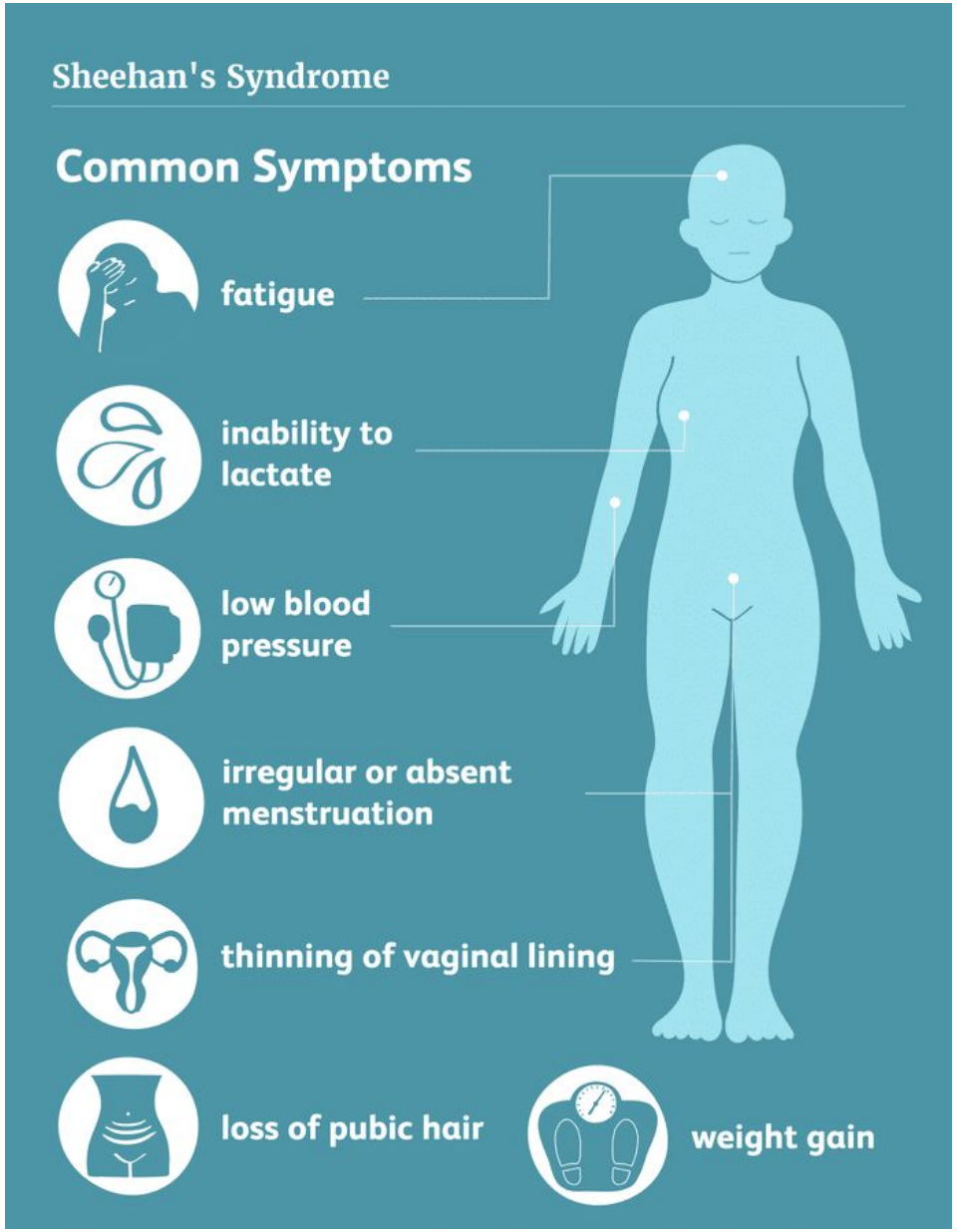


СИНДРОМ ШИХАНА

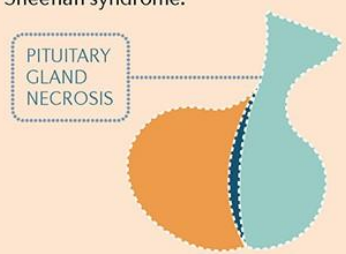
Катамадзе Натия Зурабиевна

For the Primer, visit doi:10.1038/nrdp.2016.92

Sheehan syndrome is a condition characterized by hypopituitarism due to necrosis of the pituitary gland, which is secondary to massive postpartum haemorrhage (PPH).

EPIDEMIOLOGY

In the developed world, Sheehan syndrome is rare. The prevalence of hypopituitarism is ~40 per 1,000,000 individuals. The most common causes (~60%) of hypopituitarism are tumours (mainly in the pituitary gland) and their treatment; 6% of all hypopituitarism cases are caused by Sheehan syndrome. Prevalence of the condition is much higher in the developing world, owing to less-advanced obstetrical care. In India, ~3% of the women ≥20 years of age who have given birth are estimated to have Sheehan syndrome.



Slow deterioration of pituitary function is often observed many years after the initial insult. Autoimmunity due to antigen exposure by the necrotized tissue might play a part.

Genetics
Small sella turcica size
Autoimmunity
Severe PPH
Coagulation abnormalities

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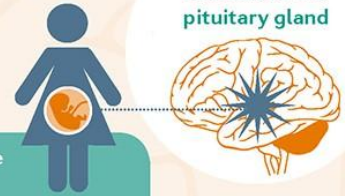

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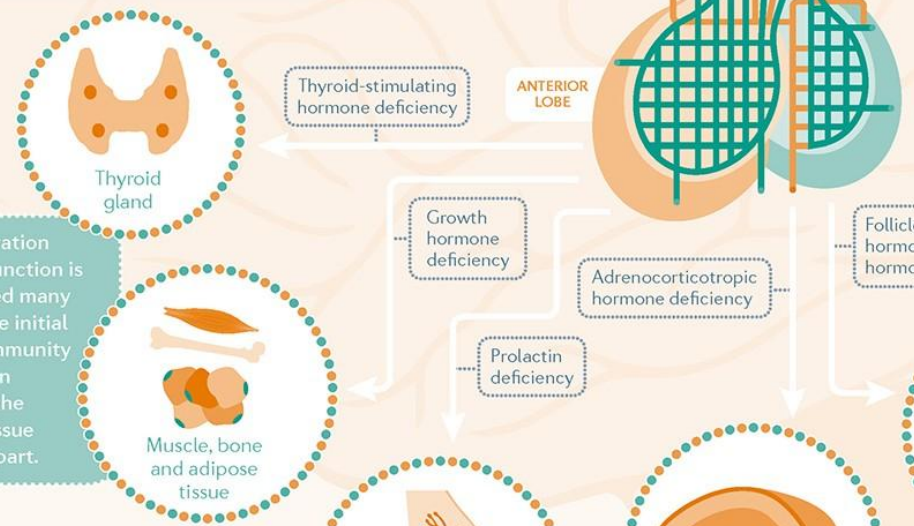

The anterior lobe, which mainly receives blood supply from the venous system instead of arterial supply, is most susceptible to ischaemia

DIAGNOSIS

Diagnostic criteria vary but usually involve a history of PPH, absence of postpartum lactation, failure to resume regular menstruation, varying degrees of anterior pituitary hormone insufficiencies and an empty sella turcica on imaging. Symptoms are caused by a decrease or absence of at least one pituitary hormone, and vary from nonspecific symptoms (such as fatigue) to severe adrenal crisis. Laboratory tests (hormone and electrolyte levels and blood cell counts), hormone stimulation tests and imaging of the pituitary gland are used to confirm diagnosis.

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MANAGEMENT

Hormone replacement therapy is the only available management option, although it does not improve pituitary function or halt the progression of pituitary gland necrosis. In accordance with the location of hormone-secreting cells relative to the vasculature, the secretion of growth hormone and prolactin is most commonly affected, followed by follicle-stimulating hormone and luteinizing hormone. Severe pituitary gland necrosis also affects the secretion of thyroid-stimulating hormone and adrenocorticotropic hormone. Although rare, involvement of the posterior lobe of the pituitary gland can result in arginine vasopressin deficiency, with diabetes insipidus as a consequence.

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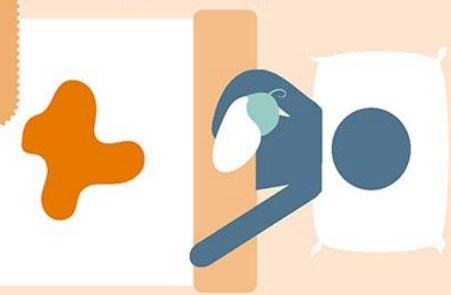

Written by Liesbet Lieben; designed by Laura Marshall

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MECHANISMS

Postpartum necrosis of the pituitary gland

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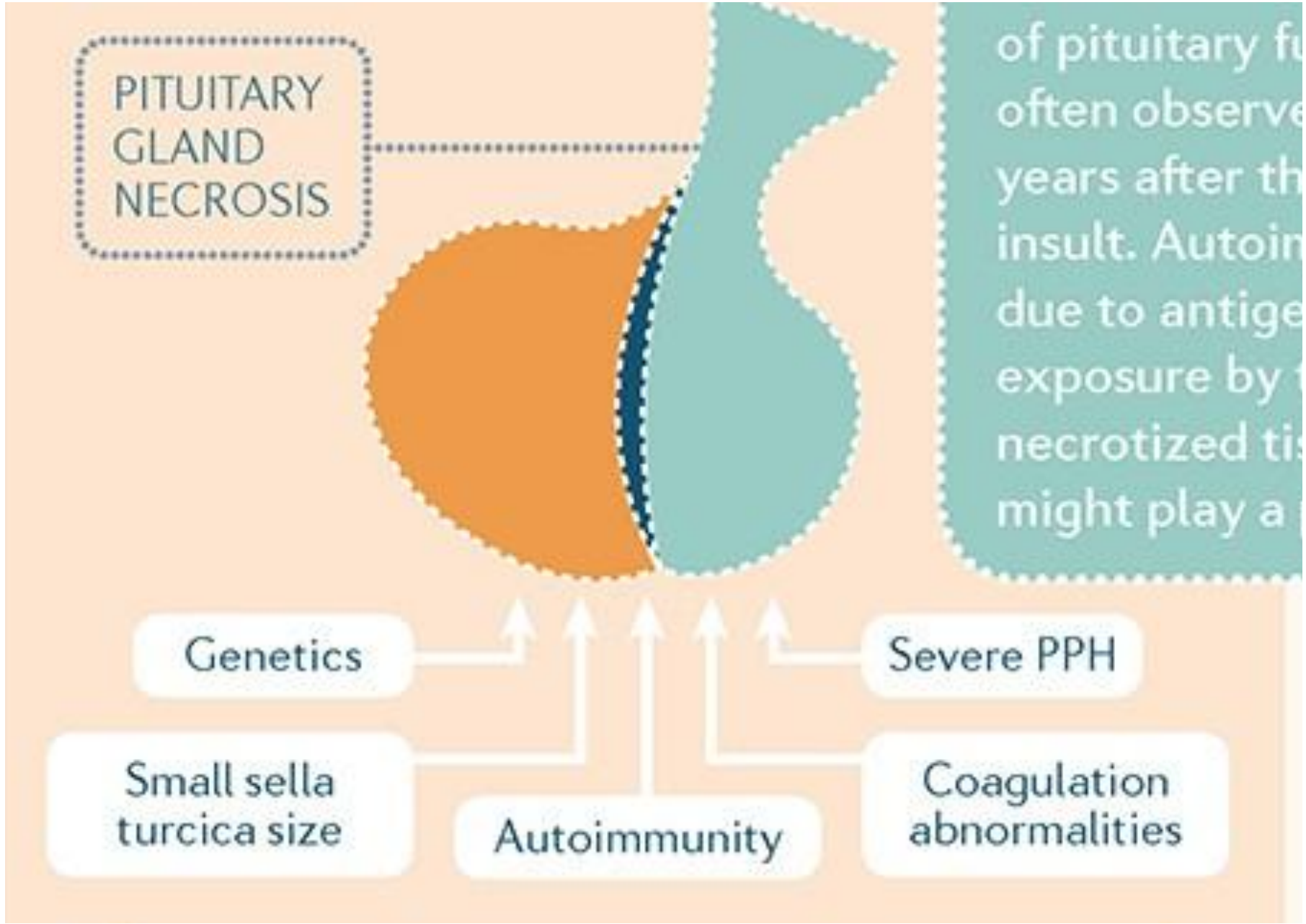
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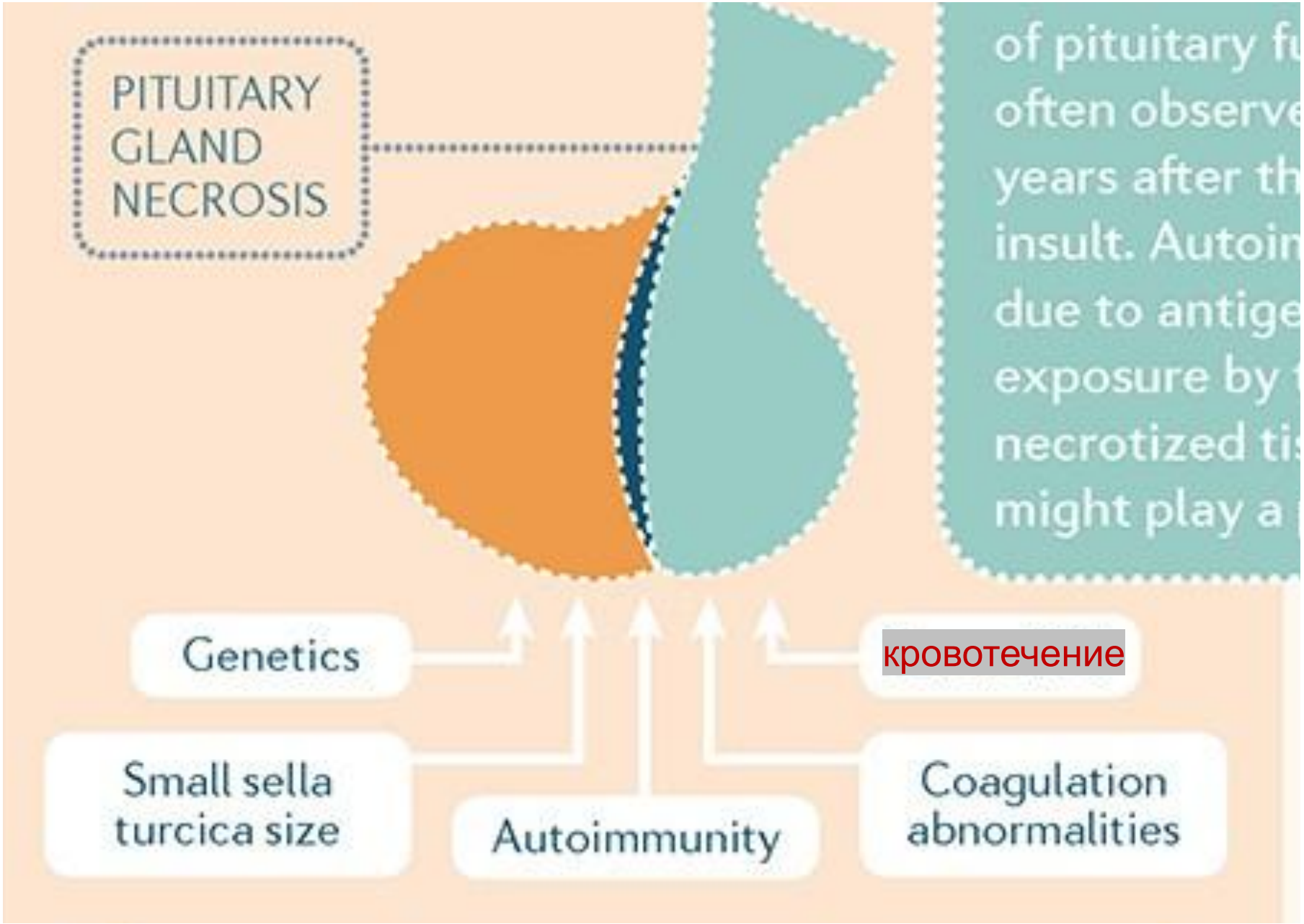
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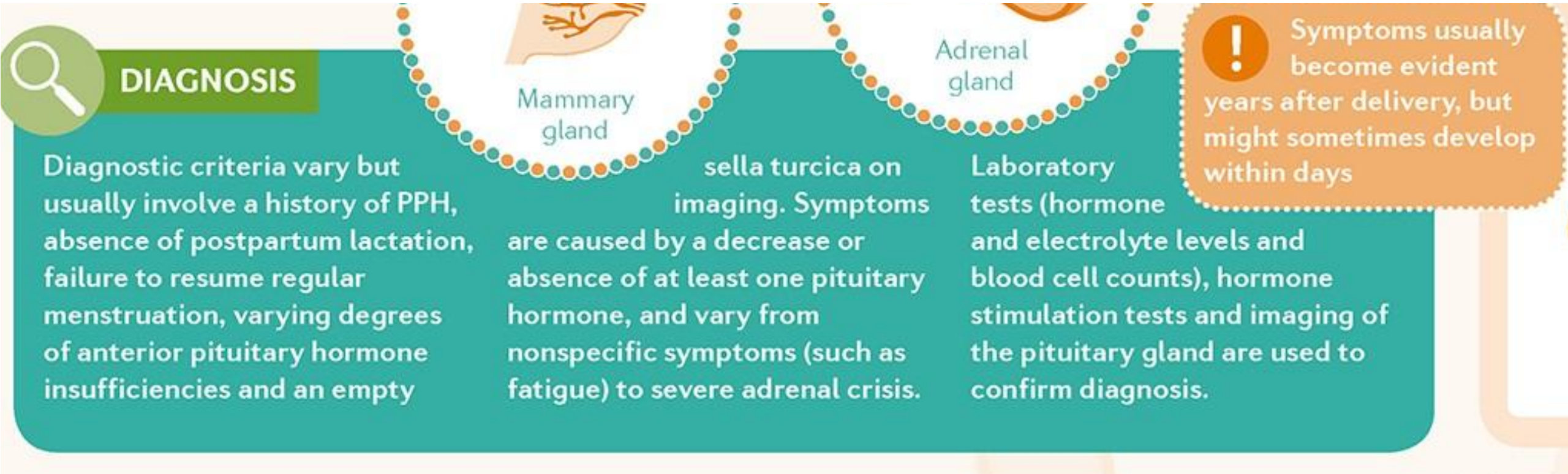

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Adrenal gland

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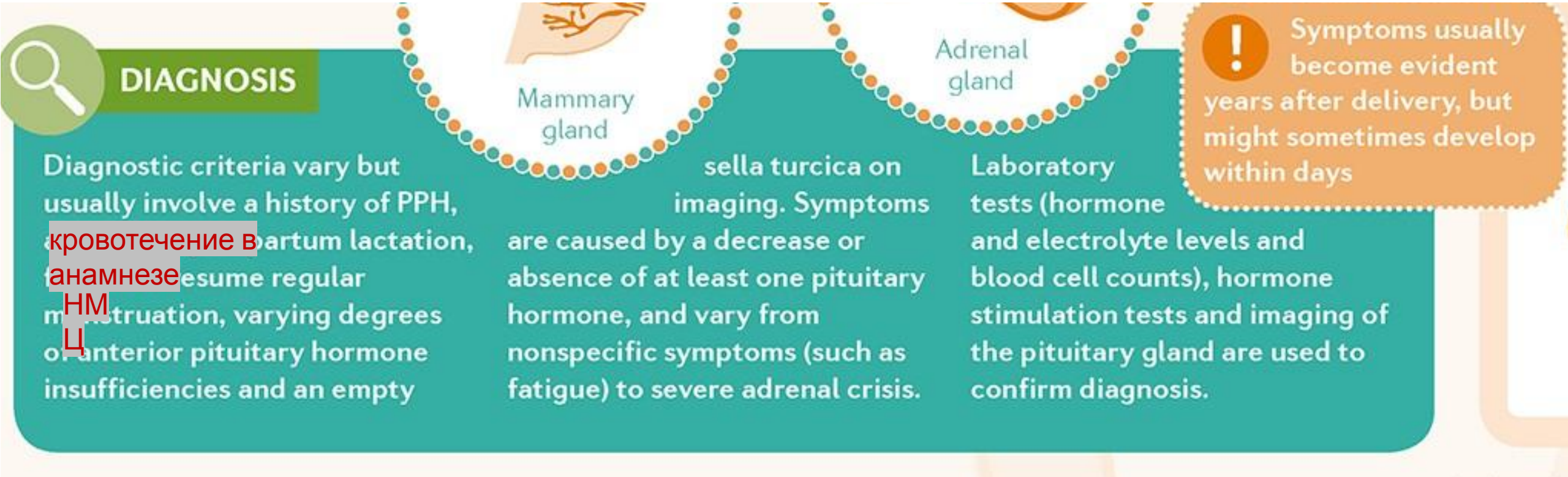

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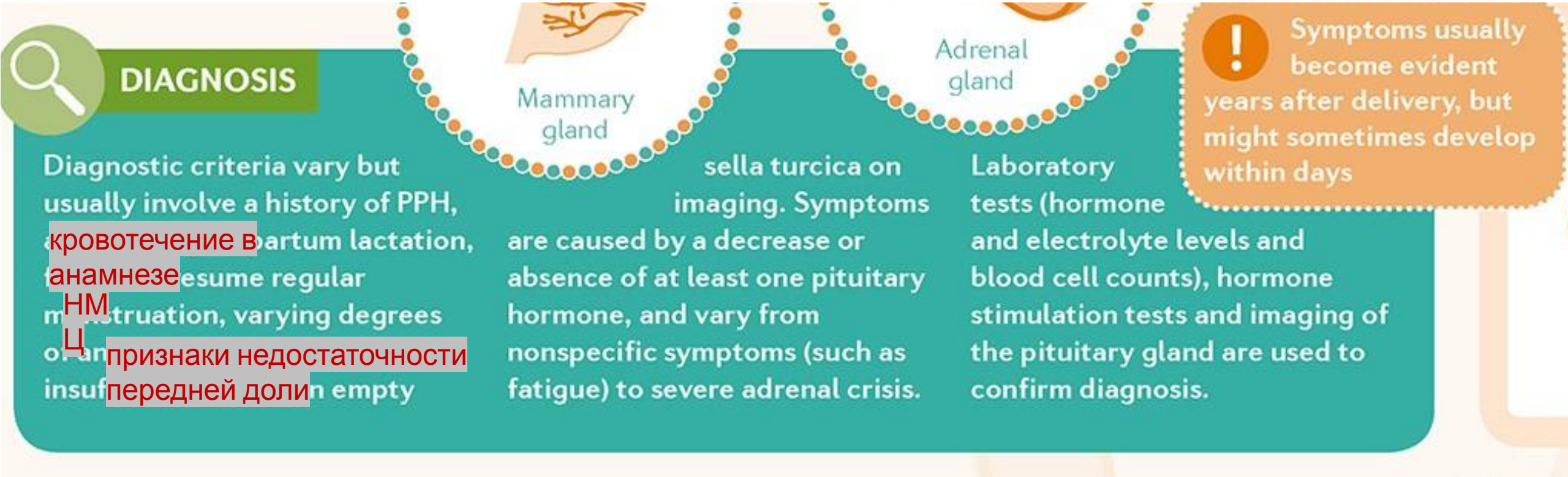

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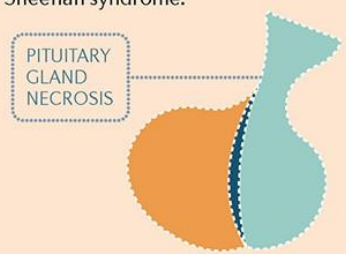

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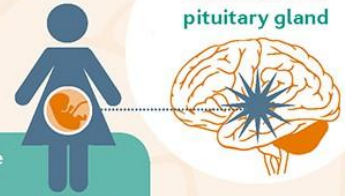

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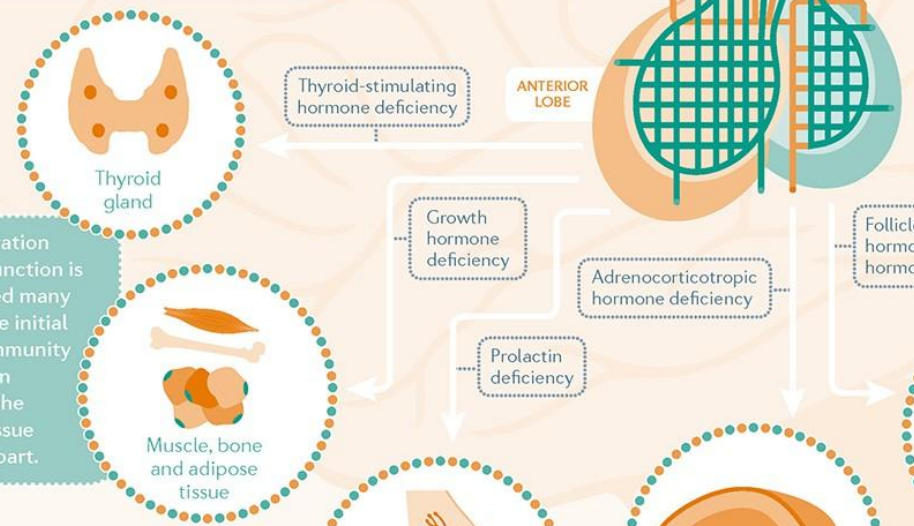

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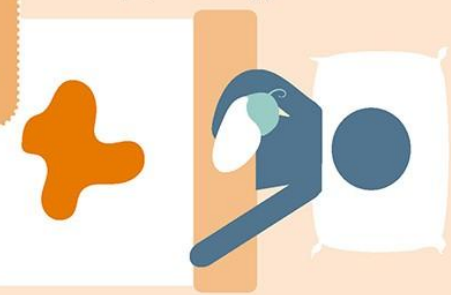

Written by Liesbet Lieben; designed by Laura Marshall

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Clinical Study | H Diri and others | Sheehan's syndrome | 171:3 | 311–318

Extensive investigation of 114 patients with Sheehan's syndrome: a continuing disorder

Halit Diri[1], Fatih Tanriverdi[1], Zuleyha Karaca[1], Serkan Senol[2], Kursad Unluhizarci[1], Ahmet Candan Durak[2], Hulusi Atmaca[3] and **Fahrettin Kelestimur[1]**

Departments of [1]Endocrinology and [2]Radiology, Erciyes University Medical School, 38039 Kayseri, Turkey and [3]Department of Endocrinology, Ondokuz Mayis University Medical School, Samsun, Turkey

Correspondence should be addressed to F Kelestimur
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Abstract

Objective: Sheehan's syndrome (SS) is a well-known cause of hypopituitarism resulting from *postpartum* pituitary necrosis. Because of its rarity in Western society, its diagnosis is often overlooked. We aimed to investigate the clinical, laboratory, and radiological aspects of SS in a large number of patients.
Study design: A retrospective assessment of the medical records of 114 patients with SS was conducted. In addition, sella turcica volumes of 29 healthy women were compared with those of patients by magnetic resonance imaging examinations.
Results: The mean period of diagnostic delay was 19.7 years in patients with SS. It was found that 52.6% of patients had nonspecific complaints, 30.7% had complaints related to adrenal insufficiency, and 9.6% had complaints related to hypogonadism when diagnosed. At the time of diagnosis, 55.3% of the patients had panhypopituitarism, while 44.7% had partial hypopituitarism. The number of deficient hormones was found to be increased over the years. None of the patients whose basal prolactin was below 4.0 ng/ml had adequate prolactin responses to TRH test, while all patients whose basal prolactin was above 7.8 ng/ml had adequate responses. Mean sella volume was found to be significantly lower in the SS group (340.5 ± 214 mm^3) than that in the healthy group (602.5 ± 192 mm^3).
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Results

The mean age of 114 patients previously diagnosed with SS between 1985 and 2013 was 63.2±12.5 years, and age at diagnosis was 52.1±12.7 years. When the past histories of patients with SS were analyzed, mean age at the last delivery was 32.4±6.5 years and thereby the period of diagnostic delay was 19.7±10.2 years. After diagnosis, the follow-up time was 7.4±6.9 years. Nine (7.9%) cases had died and 55 (48.2%) were alive. The other 50 (43.9) patients did not attend follow-up visits in the last 3 years, and therefore it was not known whether they were still alive.

Table 3 Deficient hormones of anterior pituitary gland in patients with SS.

Deficient hormones	Number (%) of patients
All hormones	63 (55.3)
FSH–LH + GH + ACTH + TSH	17 (14.9)
FSH–LH + GH + TSH + PRL	12 (10.5)
FSH–LH + GH + TSH	11 (9.6)
FSH–LH + GH + PRL	5 (4.4)
FSH–LH + GH	4 (3.5)
FSH–LH + GH + ACTH + PRL	1 (0.9)
FSH–LH + GH + ACTH	1 (0.9)
Total	114 (100)

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Deficient hormones
All hormones
FSH–LH + GH + ACTH + TS
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FSH–LH + GH
FSH–LH + GH + ACTH + PR
FSH–LH + GH + ACTH
Total

Table 4 Progression of hormonal deficiency in ten patients with SS.

Patient nos	Intact hormones at date of diagnosis	After a period of (years)	Intact hormones in last clinical visit
21	ACTH	18	None
31	ACTH + TSH + PRL	15	ACTH + PRL
35	ACTH	22	None
60	ACTH	6	None
64	ACTH + PRL	9	None
66	PRL + TSH	12	None
81	PRL	22	None
90	ACTH	11	None
95	ACTH	10	None
109	ACTH + TSH	8	TSH

Clinical Report of 28 Patients with Sheehan's Syndrome

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Abstract. The aim of the present study was to determine the clinical and hormonal characteristics with Sheehan's syndrome in 28 cases that we had diagnosed and followed in the last 20 years. Twenty-eight patients with Sheehan's syndrome, diagnosed and followed at our University Endocrinology Clinic in the last 20 years were reported in the study. Medical history, physical examination, routine laboratory examinations, pituitary hormone analysis, CT and/or MRI scan of the sella of the patients were reviewed. All patients had a history of massive hemorrhage at delivery and physical signs of Sheehan's syndrome. Twenty-six of them lacked postpartum milk production, followed by failure of resumption of menses. There were 9 subjects with disturbances in consciousness associated with hyponatremia on admittance. All 28 patients had secondary hypothyroidism, adrenal cortex failure, hypogonadotrophic hypogonadism and growth hormone deficiency. Diabetes insipidus has not been found in any patient. Empty sellae were revealed in 8 patients by CT and/or MRI scan. Sheehan's syndrome is still encountered in clinical practice occasionally. If not diagnosed early, it could cause increased morbidity and mortality. The most important clues for diagnosis of Sheehan's syndrome are lack of lactation and failure of menstrual resumption after a delivery complicated with severe hemorrhage.

Key words: Sheehan's syndrome, Pituitary failure, Hyponatremia, Empty sellae

(*Endocrine Journal* **50**: 297–301, 2003)

Table 1. The characteristics of the patients with Sheehan's syndrome.

Patients	Age at diagnosis (year)	Duration of illness (year)	Total number of pregnancy	Lactation	Serum sodium (mEq/L)	Consciousness on admittance	Drugs of substitution	CT and/or MRI scan of sellae
1	45	8	3	–	N	N	L, P, HR	N
2	60	17	8	–	110	Coma	L, P	ES
3	70	20	6	–	N	N	L, P	N
4	60	22	3	–	132	Confusion	L, P	N
5	30	7	1	–	128	N	L, P, HR	N
6	34	6	2	–	N	N	L, P, HR	N
7	64	9	2	–	N	N	L, P	N
8	35	8	1	–	N	N	L, P, HR	ES
9	51	19	1	–	110	Stupor	L, P, HR	ES
10	48	10	2	–	N	N	L, P, HR	N
11	38	9	5	–	N	N	L, P, HR	N
12	50	15	4	–	N	N	L, P, HR	N
13	35	6	4	–	N	N	L, P, HR	N
14	60	18	9	–	N	N	L, P, HR	N
15	35	6	1	–	N	N	L, P, HR	N
16	39	8	1	+	N	N	L, P, HR	N
17	45	12	1	–	N	N	L, P, HR	N
18	50	17	3	–	N	N	L, P, HR	N
19	46	20	1	–	N	N	L, P, HR	N
20	45	14	1	–	N	N	L, P, HR	N
21	43	15	1	+	N	N	L, P, HR	N
22	58	10	4	–	126	Coma	L, P, HR	ES
23	67	30	7	–	119	Coma	L, P	ES
24	47	20	5	–	101	Confusion	L, P, HR	ES
25	57	20	3	–	N	Confusion	L, P	ES
26	49	19	5	–	N	N	L, P, HR	ES
27	44	12	6	–	110	Coma	L, P, HR	N
28	45	13	4	–	114	Coma	L, P, HR	N

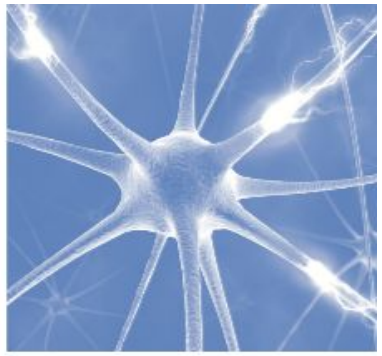
+ Present, – Absent, N: Normal, ES: Empty Sellae, L: Levothyroxine (100–150 ug/day), P: Prednisolon (7.5–10 mg/day), HR: Hormone replacement (estrogen plus progesterone)

Secondary hypothyroidism, secondary adrenal failure, hypogonadotropic hypogonadism and growth hormone deficiency were present in all patients. Serum hormone results (means ± SD) were found as follows: TSH: 1.32 ± 0.85 mIU/ml (R: 0.37 to 4.2, normal range [N]: 0.47–5.01), FT_3: 2.09 ± 1.7 pg/dl (R: 0.17–2.70, N: 2.3–4.2), FT_4: 0.43 ± 0.28 ng/dl (R: 0.11–0.80, N: 0.89–1.80), FSH: 5.01 ± 3.21 mIU/ml (R: 0.4–11, normal range for menopause 40–200), LH: 2.07 ± 1.95 mIU/ml (R: 0.08–7.3, N for menopause 40–200), GH: 0.23 ± 0.25 ng/ml (R: 0–0.9, N: >7 after insulin induced hypoglycemia), prolactin: 2.38 ± 1.53 ng/ml (R: 0.1–4.5, N: 2.7–26), ACTH: 17.15 ± 11.03 pg/nl (R: 0.1–43.5, N: 10–60), cortisol: 2.96 ± 3.37 µg/dl (R: 0.1–11.4, N: >20 after insulin induced hypoglycemia). Individual value of each hormone is presented (see Fig. 1). Diabetes insipidus was not observed in any patient.

CT and/or MRI scan of sella of 28 subjects revealed 20 normal and 8 empty sellae.

КЛИНИЧЕСКИЕ СЛУЧАИ

Синдром Шихана у пациентки с патологией щитовидной железы



Волкова А.Р.,
Остроухова Е.Н.,
Дора С.В.,
Власова К.А.,
Семикова Г.В.

Федеральное государственное бюджетное образовательное учреждение высшего образования «Первый Санкт-Петербургский государственный медицинский университет им. акад. И.П. Павлова» Министерства здравоохранения Российской Федерации, 197022, г. Санкт-Петербург, Российская Федерация

Пациентка П., 34 года, обратилась на прием 14.08.2019 с жалобами на выраженную слабость, пониженный аппетит, тошноту, сердцебиение, снижение массы тела на 1,5 кг за последнюю неделю. Описанные жалобы появились в конце мая 2019 г. и постепенно прогрессировали.

Пациентка П., 34 года

ТТГ крови – 5,6 мМЕ/л (норма 0,2–2,5),
св.T_4–12,1 пмоль/л (норма 12–22).
Был поставлен диагноз: «субклинический гипотиреоз АИТ?».
Назначены L-тироксин (50 мкг/сут), йодомарин (200 мкг/сут)

Пациентка П., 34 года

ТТГ 1,8 мМЕ/л.

ТТГ крови – 5,6 мМЕ/л (норма 0,2–2,5),
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Пациентка П., 34 года

ТТГ 1,8 мМЕ/л.

срочные роды

12.10.2018 (6 неделя)	22.11.2018 (12 недель)	24.05.2019 (39 неделя)	С июня по август 2019 г.	21.07.2019	11.08.2019	14.08.2019

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Через 40 мин после родов - нарушение гемодинамики с затруднением дыхания, слабостью, массивное кровотечение, гемоглобина до 59 г/л (до родов гемоглобин составлял 116 г/л). Пациентка переведена в реанимационное отделение областной больницы – экстренное переливание крови. Выписана из стационара в удовлетворительном состоянии на 7-й день, гемоглобин составил 88 г/л.

Пациентка П., 34 года

ТТГ 1,8 мМЕ/л.

срочные роды

12.10.2018 (6 неделя)

22.11.2018 (12 недель)

24.05.2019 (39 неделя)

С июня по август 2019 г.

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11.08.2019

14.08.2019

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МРТ головного мозга и гипофиза от 21.07.2019 – размеры гипофиза в норме, имеет однородный сигнал

Пациентка П., 34 года

ТТГ 1,8 мМЕ/л.

срочные роды

ТТГ крови – 5,6 мМЕ/л (норма 0,2–2,5), св.T_4–12,1 пмоль/л (норма 12–22). Был поставлен диагноз: «субклинический гипотиреоз АИТ?». Назначены L-тироксин (50 мкг/сут), йодомарин (200 мкг/сут)

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МРТ головного мозга и гипофиза от 21.07.2019 – размеры гипофиза в норме, имеет однородный сигнал

ТТГ крови – 0,0025 мМЕ/л, св. Т4–28,67 пмоль/л (норма 9–19,05), св. Т3–4,24 пмоль/л (норма 4,0–7,4 пмоль/л), гемоглобин – 118 г/л. Обратилась к эндокринологу, поставлен диагноз диффузного токсического зоба,

Пациентка П., 34 года

ТТГ 1,8 мМЕ/л.

срочные роды

Рекомендован прием тирозола (по 10 мг 3 раза в день) (не принимала)

12.10.2018 (6 неделя)	22.11.2018 (12 недель)	24.05.2019 (39 неделя)	С июня по август 2019 г.	21.07.2019	11.08.2019	14.08.2019
ТТГ крови – 5,6 мМЕ/л (норма 0,2–2,5), св.T_4–12,1 пмоль/л (норма 12–22). Был поставлен диагноз: «субклинический гипотиреоз АИТ?». Назначены L-тироксин (50 мкг/сут), йодомарин (200 мкг/сут)		Через 40 мин после родов - нарушение гемодинамики с затруднением дыхания, слабостью, массивное кровотечение, гемоглобина до 59 г/л (до родов гемоглобин составлял 116 г/л). Пациентка переведена в реанимационное отделение областной больницы – экстренное переливание крови. Выписана из стационара в удовлетворительном состоянии на 7-й день, гемоглобин составил 88 г/л.		МРТ головного мозга и гипофиза от 21.07.2019 – размеры гипофиза в норме, имеет однородный сигнал	ТТГ крови – 0,0025 мМЕ/л, св. Т4–28,67 пмоль/л (норма 9–19,05), св. Т3–4,24 пмоль/л (норма 4,0–7,4 пмоль/л), гемоглобин – 118 г/л. Обратилась к эндокринологу, поставлен диагноз диффузного токсического зоба,	

14.08.2019 обратилась на прием к эндокринологу в ПСПбГМУ им. акад. И.П. Павлова.

Объективный осмотр: общее состояние удовлетворительное, кожные покровы бледные, поредение волос в паховой и подмышечной областях, рост – 161 см, масса тела – 52,8 кг, индекс массы тела – 20,4 кг/м2, пульс – 82 в минуту, удовлетворительных свойств, артериальное давление на левой руке – 90/50 мм рт.ст. Тоны сердца ясные, чистые.

Щитовидная железа не увеличена, сосудистый шум над железой не выслушивается, узловые образования пальпаторно не определяются. Глазные симптомы отрицательны.

Клинически не складывалось впечатление в пользу тиреотоксикоза.

Рекомендовано дообследование: определение в крови кортизола, адренокортитропного гормона (АКТГ), анализ крови на антитела к тиреопероксидазе (ТПО), антитела к рецептору тиреотропного гормона (ТТГ).

16.08.2019: кортизол – 1,8 нмоль/л (норма 171–536), АКТГ – 5,1 пг/мл (норма 0–46), антитела к рецептору ТТГ – 0,75 МЕ/л (норма 0–1,75), антитела к ТПО – 7,7 Ед/мл (норма 0–34). Поставлен диагноз: «послеродовый тиреодит, эутиреоз? Вторичная надпочечниковая недостаточность. Синдром Шихана».

16.08.2019: кортизол – 1,8 нмоль/л (норма 171–536), АКТГ – 5,1 пг/мл (норма 0–46), антитела к рецептору ТТГ – 0,75 МЕ/л (норма 0–1,75), антитела к ТПО – 7,7 Ед/мл (норма 0–34). Поставлен диагноз: «послеродовый тиреодит, эутиреоз? Вторичная надпочечниковая недостаточность. Синдром Шихана».

16.08.2019: кортеф (50 мг/сут: 30 мг – утром, 20 мг – 16.00). Рекомендовано дообследование: ФСГ, ЛГ, эстрадиол, пролактин, соматотропный гормон (СТГ).

16.08.2019: кортизол – 1,8 нмоль/л (норма 171–536), АКТГ – 5,1 пг/мл (норма 0–46), антитела к рецептору ТТГ – 0,75 МЕ/л (норма 0–1,75), антитела к ТПО – 7,7 Ед/мл (норма 0–34). Поставлен диагноз: «послеродовый тиреодит, эутиреоз? Вторичная надпочечниковая недостаточность. Синдром Шихана».

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На 2-й день приема глюкокортикоидов пациентка отметила улучшение самочувствия: уменьшилась слабость, появился аппетит.

16.08.2019: кортизол – 1,8 нмоль/л (норма 171–536), АКТГ – 5,1 пг/мл (норма 0–46), антитела к рецептору ТТГ – 0,75 МЕ/л (норма 0–1,75), антитела к ТПО – 7,7 Ед/мл (норма 0–34). Поставлен диагноз: «послеродовый тиреодит, эутиреоз? Вторичная надпочечниковая недостаточность. Синдром Шихана».

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28.08.2019: ФСГ – 0,85 мМЕ/л (норма 3,5–12,5), ЛГ – 0,41 мМЕ/л (норма 1,69–15,0), эстрадиол – 12 пмоль/л (90–860 норма), пролактин – 121 мкг/л (норма 130–540), св.Т3–3,3 пмоль/л (норма 2,6–5,7), св.Т4–8,25 пмоль/л (норма 9–19), ТТГ – 0,07 мМЕ/л (норма 0,3–3,4).

16.08.2019: кортизол – 1,8 нмоль/л (норма 171–536), АКТГ – 5,1 пг/мл (норма 0–46), антитела к рецептору ТТГ – 0,75 МЕ/л (норма 0–1,75), антитела к ТПО – 7,7 Ед/мл (норма 0–34). Поставлен диагноз: «послеродовый тиреодит, эутиреоз? Вторичная надпочечниковая недостаточность. Синдром Шихана».

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29.08.2019: состояние удовлетворительное, кожные покровы обычного цвета и влажности, рост – 161 см, масса тела – 54,1 кг (увеличение на 1,3 кг), ИМТ – 20,9 кг/м2, артериальное давление – 110/80 мм рт.ст., пульс – 78 в минуту, ритмичный

Диагноз: недостаточность передней доли гипофиза. Синдром Шихана. Вторичная недостаточность коры надпочечников. Вторичный гипотиреоз. Вторичный гипогонадизм.

кортеф – 10 мг/сут (по 1 таблетке утром и по 1/2 таблетки в 16.00)

эутирокс (L-тироксин) – 25 мкг 1 раз в день, утром, натощак, за 40 мин до завтрака; фемостон 2/10.

15.09.2019: кортизол в суточной моче – 267,96 мкг/сут (норма 58–403), св.Т3–3,3 пмоль/л (норма 2,6–5,7), св.Т4–14,5 пмоль/л (норма 9–19). Рекомендовано дальнейшее наблюдение у эндокринолога, гинеколога.