Asian Biomedicine Vol. 9 No. 2 April 2015; 237-243 DOI: 10.5372/1905-7415.0902.393

Clinical report

Pituitary stalk interruption syndrome: a case report and review of the literature

Yan Zhang[a], Changbin Quan[b], Weimin Li[c], Liying Chen[a], Xiaoyan Song[a], Jianbin Xu[a], Guangqing Zhu[d]
[a]*Department of Endocrinology and Rheumatism, General Hospital of General Staff Department of the People's Liberation Army, Beijing 100091, China*
[b]*Department of Radiology, General Hospital of General Staff Department of the People's Liberation Army, Beijing 100091, China*
[c]*Department of Hepatobiliary Surgery, General Hospital of General Staff Department of the People's Liberation Army, Beijing 100091, China*
[d]*Department of Oncology, Air Force General Hospital, Beijing 100142, China*

Background: Pituitary stalk interruption syndrome (PSIS) is characterized by a thin or interrupted pituitary stalk, impairing delivery of hypothalamic hormones to the pituitary, and anterior pituitary dysplasia.
Objectives: To summarize the manifestations and treatment of PSIS.
Methods: We performed a meta-analysis of PSIS research published between January 1, 2004 and December 31, 2013. Chinese databases were searched using the terms "pituitary stalk interruption syndrome", "PSIS", and "vertebral handle interrupts syndrome"; and results limited to clinical studies. Age of onset and diagnosis, sex, symptoms at presentation, main clinical signs, magnetic resonance imaging (MRI), laboratory results, and other data were extracted from qualifying studies.
Results: We included 311 cases from 33 papers in the meta-analysis. The male:female ratio was 257:54, and 183 of the 311 patients had a history of abnormal birth or cerebral hypoxia. MRI showed 239 cases of missing and 62 cases of tapered pituitary stalk. Multiple pituitary hormone deficiency was identified in 190 cases, and single growth hormone deficiency in 75. There were significant associations between missing pituitary stalk and multiple pituitary hormone deficiency, and pituitary stalk tapering and single hormone deficiency ($P < 0.01$). The clinical signs included growth retardation and the absence or delay in development of secondary sexual characteristics. Therapy was usually inadequate, with only 12 patients (3.86%) undergoing growth hormone replacement.
Conclusions: Abnormal delivery and cerebral hypoxia may be important causes of PSIS, and MRI is valuable for its diagnosis. Measuring the levels of pituitary hormones is necessary for timely diagnosis and treatment.

Keywords: Abnormal parturition, case report, cerebral anoxia, newborn, pituitary stalk interruption syndrome

Pituitary stalk interruption syndrome (PSIS) is usually characterized by a very thin or interrupted pituitary stalk, complicated by an ectopic posterior pituitary and impaired delivery of hormones secreted by the hypothalamus to the pituitary through the pituitary stalk, which results in dysplasia of the anterior pituitary [1]. PSIS was first described by Fujisawa in 1987 [2]. Chinese researchers used magnetic resonance imaging (MRI) and computed tomography (CT) of the sellar region in studies of primordial dwarfism as early as 1993 [3]. Since Ying Liu et al. first reported PSIS in China in 2004, a growing number of reports have been published, which mainly focus on imaging results of individual cases instead of clinical changes and laboratory test results. Here, we report a case of PSIS with obesity, and review the relevant literature.

Correspondence to: Guangqing Zhu, Department of Oncology, Air Force General Hospital, Beijing 100142, China.
E-mail: guangqingzhu@yeah.net

Case report

A 36-year-old male patient required gastric banding surgery because of weight gain for 14 years. The patient was heavier than average as a child, which did not attract any attention. The patient was diagnosed with small pituitary at 8 years of age because he was shorter than his peers and had learning difficulties. Growth hormone was administered to the

patient twice, but no improvement was observed. At age 21, the patient was 154 cm tall and weighed 68 kg. The patient was diagnosed with dysplasia of the reproductive organs at 22. He had not undergone puberty, as evidenced by his lack of pubic hair, armpit hair, Adam's apple, voice cracking, and nocturnal erections and emissions, the latter indicating immaturity of the penis and testicles. Blood tests showed deficiency in thyroid-stimulating hormone (TSH), growth hormone (GH), luteinizing hormone (LH), and follicle-stimulating hormone (FSH), indicating anterior pituitary hypothyroidism. MRI showed a pituitary with small volume. Bone-age films showed that the patient had the bone age of a 16-year-old, but he refused testosterone undecanoate therapy at this time. At the most recent examination, his body weight had increased to 118 kg (height, 160 cm). The patient had no somnolence, dysosmia, polydipsia, diuresis, headache, dizziness, or visual field defect, and was eligible for gastric banding surgery. He had normal intelligence and was in good mental and physical condition. The patient had dyslipidemia for more than ten years without history of high blood pressure, diabetes, or coronary heart disease. He was single and had no children. His parents were not in a consanguineous marriage, and his brother and two sisters were healthy. The patient was his mother's fourth delivery. He weighed 4 kg at birth and was delivered vaginally, but he suffered oxygen deprivation at birth because of dystocia.

Physical examination findings

Physical examination results were as follows: height, 160 cm (upper body, 84 cm; lower body, 76 cm); arm span, 160 cm; waist, 127 cm; hip circumference, 134 cm; weight, 118 kg; BMI, 47.3 kg/m^2; no facial hair or Adam's apple; developed bilateral breast; sparse pubic hair; penis length, 3 cm; and testicles could not be palpated.

Test results

Laboratory test results were as follows: testosterone, 0.87 (normal range 1.53–7.40) nmol/L; FSH, 0.27 mIU/mL (1.5–12.4); estradiol, 77.18 pmol/L (40.6–288); LH, 1.41 IU/L (1.7–8.6); prolactin, 83.07 μg/L (2.1–17.7); GH <0.05 μg/L (0.06–5.0); insulin-like growth factor-1, 55.8 U; free thyroxine, 7.60 pmol/L (10.42–24.32); TSH, 2.23 mU (l.35–5.50); total thyroxine, 19.9 nmol/L (55.34–160.88); 8.00 AM adrenocorticotropic hormone (ACTH), 5.04 pmol/L [<10.12 (DPC)]; 12.00 PM ACTH, 3.57 pmol/L; 8 AM cortisol, 131.91 nmol/L (198.7–797.5); 12 PM cortisol, 75.02l nmol/L (0–165.7); insulin 0 h, 15.47 mU/L; insulin 2 h, 288.2 mU/L; C peptide 0 h, 3.16 ng/mL; C peptide 2 h, 17.48 ng/L; fibrinogen β chain 0 h, 4.20 mmol/L; PGB 2 h, 7.68 mmol/L; cholesterol, 5.75 mmol/L; triglyceride, 3.00 mmol/L; high density lipoprotein-cholesterol, 1.06 mmol/L; low density lipoprotein-cholesterol, 3.83 mmol/L; glutamate pyruvate transaminase, 56 IU/L; and glutamic oxaloacetic transaminase, 39 IU/L. Other blood and urine test results were normal. The electrocardiogram revealed a sinus rhythm and abnormal T wave. Gonadotropin releasing hormone stimulation test results were as follows: serum LH level was 0.17, 0.17, 0.69, 0.66, 0.64 IU/L 15 min before stimulation and at 0, 30, 60, 120 min, respectively. Serum FSH level was 0.76, 0.51, 1.19, 1.18, 1.63 IU/L 15 min before stimulation and at 0, 30, 60, 120 min, respectively. B ultrasound showed fatty liver disease, but biliary tree, pancreas, spleen, and both kidneys were normal. Right and left testes were 0.98 cm × 1.3 cm × 1.9 cm and 1.1 cm × 1.3 cm × 1.7 cm, respectively, and bilateral testicular volume was reduced with microcalcification. Left adrenal gland showed slight localized thickening without obvious abnormal density on imaging. The pituitary stalk was not evident by MRI scan.

Diagnosis and treatment

Specialists outside of our hospital diagnosed the patient with PSIS, anterior pituitary hypofunction, and dyslipidemia. The patient was prescribed 5 mg of prednisone acetate in tablet form, which he took at 6 AM every day. One month later, the patient began receiving 50 μg of levothyroxine sodium every day, and 259 mg of testosterone undecanoate was injected intramuscularly 3 times per week. The patient made regular visits to an outpatient clinic to monitor his thyroid function, cortisol rhythm, gonadal hormones, blood fat, liver function, and to take other medical tests.

The search terms "pituitary stalk interruption syndrome," "PSIS", and "vertebral handle interrupts syndrome" were used to search the China Hospital Knowledge Database and Wanfang Database, and the results were limited to clinical research published between January 1, 2004 and December 31, 2013. This search returned 40 articles, but 7 of them were from a study of the same population, so we merged the data. In total, 33 articles were identified for further

review (**Table 1**). Age of onset, age at diagnosis, gender, symptoms at presentation, main clinical manifestations, cerebral MRI findings, laboratory results, and other data were abstracted from the articles that met the criteria.

Clinical data

A total of 311 cases of PSIS have been reported, comprising 257 male and 54 female patients. The age at diagnosis ranged from 40 days to 52 years, which was exactly the same as misdiagnosis time. Of the 311 cases, two cases were in infants (40- and 76-days-old) who presented with hypoglycemia and adrenal crisis. Of the cases in adults, two were unusual in that they showed normal growth and development. One 52-year-old patient was characterized by intolerance of cold, reduced sexual function, and normal growth and development; one female patient had diabetes insipidus, typical signs of PSIS on MRI, but normal growth and development, and no pituitary dysfunction. She was diagnosed with central diabetes insipidus. The remaining 307 patients were diagnosed at between 5 and 30 years of age, and they were characterized by growth retardation, missing secondary sex characteristics, and delayed bone age for more than two years. There were 12 cases of diabetes insipidus and one case of obesity.

Table 1. Chinese database search results

No	First author	Published time	Research location	Time of data	Methods	No. of cases
1	Liu et al. [10]	2004/06	Shandong	2003	Case report	1
2	Zhao et al. [11]	2005/06	Nanjing	2003	Case report	1
3	Fang et al. [12]	2007/01	Guangdong	2007	MRI diagnosis	3
4	Tian et al. [13]	2007/09	Shandong	2003–2006	MRI diagnosis	10
5	Cheng et al. [14]	2008/06	Beijing	2005–2007	MRI and clinic	14
6	Li et al. [15]	2008/11	Guangzhou	2007	MRI diagnosis	4
7	Zheng et al. [16]	2009/03	Beijing	2007	Case report	1
8	Tu et al. [17]	2009/06	Anhui	2006–2008	MRI diagnosis	9
9	Xie et al. [18]	2009/10	Jiangxi	2005–2007	MRI and clinic	15/38
10	Rong et al. [19]	2010/06	Hebei	2010	Case report	1
11	Sang et al. [20]	2010/06	Henan	2009	Clinical analysis	8
12	Ren et al. [21]	2010/07	Ningbo, Zhejiang	2009	MRI diagnosis	3
13	Zheng et al. [22]	2010/10	Shandong	2009	Case report	1
14	Quan et al. [23]	2010/09	Hainan	2008	Case report	1
15	Lu et al. [24]	2010/12	Jiangsu	2009	Case report	1
16	Lai et al. [25]	2011/02	Liaoning	2010	Case report	1
17	Pan et al. [26]	2011/02	Beijing	2001–2010	MRI diagnosis	31
18	Lu et al. [27]	2011/02	Guangdong	Unknown	MRI diagnosis	14
19	Zheng et al. [28]	2011/04	Hangzhou, Zhejiang	2009	Nursing care	1
20	Liu et al. [29]	2011/05	Beijing	2005–2010	MRI diagnosis	18
21	Dong et al. [30]	2011/02	Tianjin	2010	Case report	1
22	Zheng et al. [31]	2011/11	Anhui	Unknown	Case report	1
23	Shu et al. [32]	2012/06	Nanchang, Jiangxi	2002–2009	Clinical study	53
24	Wu et al. [33]	2012/01	Zhengzhou, Henan	Unknown	Case report	1
25	Zhang et al. [34]	2012/02	Guangdong	Unknown	Case report	1
26	Chen et al. [35]	2012/02	Beijing	1997–2011	Clinical study	36
27	Wan et al. [36]	2012-4	Xiehe, Beijing	2010/7–2011/3	Clinical analysis	3/1
28	Cao et al. [37]	2012	Guangzhou Military Hospital	2011/8	Case report	1
29	Wang et al. [38]	2012	Hubei	2005/11–2007/3	MRI and clinic	13/1
30	Li et al. [39]	2013/01	Pingdingshan, Henan	2003–2012	MRI diagnosis	8/1
31	Liang et al. [40]	2010/10	Guangdong	Unknown	Clinical study	5/2
32	Wang et al. [41]	2010/04	Hangzhou, Zhejiang	2008/1–2009/6	Clinical analysis	9/4
33	Ye et al. [42]	2008/05	Fudan, Shanghai	2005–2007	Clinical diagnosis	4/1

MRI findings

Among 311 patients, one infant showed ambiguous border between the ectocinerea and the alba, low density signal and cerebral edema in the CT scan of the head, and was diagnosed with PSIS. All the other patients were diagnosed through cerebral MRI, and comprised 239 patients without pituitary stalk, 62 patients with thin pituitary stalk, 215 patients with ectopic posterior pituitary in the optic chiasm and infundibular recess, 34 patients without posterior pituitary, 5 patients without anterior pituitary, and 174 patients with thinning pituitary. Complications included agenesis of corpus callosum (2 cases), right hippocampus smaller than left (1 case), vacuole turcica (7 cases), cerebellar tonsillar hernia into the foramen magnum (2 cases), and hypophysoma (1 case).

Pituitary hormone tests

Results of pituitary function tests were reported for 265 patients, of which 193 (72.8%) displayed lack of multiple pituitary hormones (GH, gonadotropin, TSH, ACTH, etc.). The other 70 patients (26.4%) were deficient in a single growth hormone. Secondary sex characteristics were missing in one patient in which GH level was normal. One patient had diabetes insipidus.

Pituitary stalk damage and pituitary hormone deficiency type

Of 311 patients, 265 exhibited a relationship between pituitary abnormalities and hormone deficiency. Pituitary stalk interruption was evident in 217 patients (81.9%), including 185 (85.3%) with deficiency of multiple pituitary hormones, and 31 patients (14.3%) with deficiency of a single growth hormone. Image examination showed pituitary stalk thinning in 48 patients (18.1%), of whom 8 exhibited a deficiency in multiple pituitary hormones and 39 patients were deficient in a single growth hormone. χ^2 was 85.69 ($P < 0.01$), which revealed a significant difference between the 2 groups.

Pituitary stalk damage type and pituitary change

Among 265 patients, the anterior pituitary was small, thin, or absent in 207, of which 80.7% resulted from pituitary stalk interruption and 19.3% were caused by pituitary stalk thinning. Among 259 patients with posterior pituitary ectopia or absence, 93.4% were because of pituitary stalk interruption and 6.6% were because of pituitary stalk thinning.

Etiological analysis

Among 311 patients, 155 (49.8%) had an explicit history of abnormal parturition, 67 (31.5%) had a history of cerebral anoxia, 48 patients' (22.5%) mothers had experienced pregnancy abnormalities while the patient was in utero, 45 patients (21.6%) were born in a bad parturition environment, and 5 patients had cerebral trauma (1.6%).

Treatment

Only 12 patients (3.86%) out of 311 patients with PSIS accepted growth hormone therapy. After treatment, 8 of them increased body height 1.4–11 cm, including 3 patients who were between 17 and 20 years old at the time of the treatment. Four patients ended therapy early for financial reasons.

Discussion

Since 2004, China has reported a total of 311 patients with PSIS in 33 articles, making PSIS a rare disease with a prevalence of 1/1000–1/10000 [4]. The actual prevalence of PSIS might be underestimated because of the limitations of our understanding of the disease and our methods of diagnosis. Although this disease does not affect survival, it influences the quality of life for the patients and their families. Consequently, it deserves attention from pediatricians, obstetricians, and endocrinologists. Of the 33 articles, 14 described single patients and 3 reported 2–4 cases; only a few reports summarized and analyzed a larger population of patients. Of these, 10 focused on MRI changes. Here, we report cases of adult patients who were diagnosed after puberty. Only 12 patients accepted therapy, and of those, 8 improved their body height, which indicated that even adult patients could benefit from treatment. Earlier diagnosis and treatment could help more patients improve their life condition.

Problems during pregnancy and birth were common. Two-thirds of the 311 patients were born after abnormal pregnancies or parturition, or they had experienced cerebral anoxia inside or outside the uterus. All this strongly suggests that difficulties during childbirth (e.g., breech birth, cesarean section) are a main cause of pituitary stalk interruption. Sheelan and Whitehead discovered that the pituitary was easily fractured [5] during autopsy, and most scholars consider abnormal parturition the main risk factor for PSIS [3, 6]. Therefore, PSIS could be prevented by protecting infants during the perinatal period. In addition, close monitoring of the growth and development of those children who experienced

abnormal parturition, including performing MRI to determine whether the pituitary stalk was injured in patients with suspected PSIS, and employing suitable therapy as early as possible, could avoid or reduce disability.

Because of the large population of China, the number of people with dwarfism is relatively high (180 articles reported 7226 cases of nanism between 2010 and 2012). Laboratory screening of hormone levels is quite common, but researchers often do not realize the importance of pituitary imaging. Japanese researchers [2] reported PSIS in 1980s, but Chinese doctors could not diagnose this disease until 2004 [7] because of the limitations of our imaging equipment. Our review demonstrates that PSIS is not as rare as was thought. In the case we reported, no conclusion could be drawn from the first 2 cerebral MRI and CT scans. The patient was diagnosed with PSIS only after a third MRI scan, which was read by an expert who was called for consultation. This strongly indicates the necessity of a better understanding of this disease by endocrinologists and pediatricians. The sellar region should be closely examined by MRI in patients with multiple pituitary hormone deficiency or single growth hormone deficiency, especially those with a history of abnormal pregnancy and parturition, cerebral anoxia, or both. Our study revealed that the type of damage to the pituitary stalk closely correlates with the hormone deficiency type; for example, patients without pituitary stalks were deficient in multiple pituitary hormones and patients with thin pituitary stalks were deficient in a single growth hormone. The difference between the two groups was significant, a finding that was consistent with what Komreich et al. [8] has reported. In this case, it seems that missing and thinning pituitary stalk are different damage levels. It is also possible that these abnormalities reflect gene deficiencies, and such a possibility deserves further investigation. If the incidence of PSIS could be reduced by protecting infants during the perinatal period, this disease might be renamed as pituitary stalk injury syndrome, which could attract more attention.

Because the pituitary stalk is the only route of blood circulation and the only channel of contact between the hypothalamus and the pituitary, all the patients with PSIS are characterized by anterior pituitary dysplasia. This is because pituitary stalk damage prevents hypothalamus-secreted growth hormone releasing hormone, gonadotropin releasing hormone, thyrotropin releasing hormone, and corticotropin releasing hormone from passing from the pituitary stalk through the supraopticohypophyseal tract to be delivered to the anterior pituitary. The lack of these hormones results in abnormal hormone secretion by the anterior pituitary. In our review, most PSIS patients with anterior pituitary abnormalities had a small, thin, or absent anterior pituitary, and shared clinical features of multiple pituitary hormone deficiency. These observations are consistent with reports in the literature that multiple pituitary hormone deficiency is more common than single growth hormone deficiency [9]. There were 256 cases of abnormal or absent posterior pituitary, but only 12 cases of diabetes insipidus. Some researchers have suggested that pituitary stalk damage interrupts the drainage of vasopressin and antidiuretic hormone from the hypothalamus to the posterior pituitary, but the reason for the low incidence of diabetes insipidus is collateral flow between the hypothalamus and the pituitary [4]. Therefore, although MRI scan did not show a pituitary stalk, the ectopic posterior pituitary was similar to normal; high signal was exhibited on T1-weighted imaging and the minority exhibited normal signal because of antidiuretic hormone particles, which further indicated that the function of the posterior pituitary was normal.

In conclusion, most patients were male, diagnosed late, and inadequately treated. MRI mainly showed missing pituitary stalk and posterior pituitary ectopia, followed by pituitary stalk thinning, and anterior pituitary thinning and absence. MRI scans and a pituitary hormone test are important methods for diagnosis of this disease. For infants who experienced anomalies during pregnancy and perinatally complicated with growth retardation, MRI scans and pituitary hormone tests could facilitate early diagnosis and treatment, and improve prognosis. To control PSIS in the future, we recommend more research into protecting infants from complications during pregnancy and parturition, and closely monitoring those who do suffer these complications for early signs of PSIS.

All of the authors declare that they have no conflicts of interest regarding this article.

References

1. Tauber M, Chevrel J, Diene G, Moulin P, Jouret B, Oliver I, el al. Long-term evolution of endocrine disorders and effect of GH therapy in 35 patients with pituitary stalk interruption syndrome. Horm Res.

2005; 64:266-73.
2. Fujisawa I, Kikuchi K, Nishimura K, Togashi K, Itoh K, Noma S, et al. Transection of the pituitary stalk: development of an ectopic posterior lobe assessed with MR imaging. Radiology. 1987; 165:487-9.
3. Tao XF, Shi ZR, Zhu JM, Cen RG, Ye J. The sella of the 11 patients with primary pituitary dwarfism: a parallel study of MR imaging and computed tomography. Academic Journal of Second Military Medical University. 1993; 14:268-72.
4. Vimpani GV, Vimpani AF, Lidlgard GP, Cameron EH, Farquhar JW. Prevalence of severe growth hormone deficiency. Br Med J. 1977; 2:427-30.
5. Sheehan HL, Whitehead R. The neurohypophysis in post-partum hypopituitarism. J Pathol Bacteriol. 1963; 85:145-69.
6. SloopKW, Walvoord EC, Showalter AD, Pescovitz OH, Rhodes SJ. Molecular analysis of LHX3 and PROP-1 in pituitary hormone deficiency patients with posterior pituitary ectopia. J Clin Endocrinol Metab. 2000; 85: 2701-8.
7. Li YW, Tao XF, Shi ZR, Xiao XS, Ye J, Zhu JM. Correlative statistic study between the MRI signals of sella with HGHD and PGHD. Chin J Radiology. 1997; 16:133-5.
8. Komreich L, Horev G, Lazar L, Schwarz M, Sulkes J, Pertzelan A. MR findings in growth hormone deficiency correlation with severity of hypopituitarism. AJNR. 1998; 19:1495-9.
9. Chen S, Léger J, Garel C, Hassan M, Czemichow P. Growth hormone deficiency with ectopic neurophysis: anatomical variations and relationship between the visibility of the pituitary stalk asserted by magnetic resonance imaging and anterior pituitary function. J Clin Endocrinol Metab. 1999; 84:2408-13.
10. Liu Y, Li CF, Qu CQ, Meng XS, Zheng JY, Hou JW, et al. Pituitary stalk interruption syndrome: a case report. J Clin Neuro. 2004; 17:456. [in Chinese]
11. Zhao M, Wang X, Zhang ZJ. One case of pituitary stalk interruption syndrome. Chin J Endo Metab. 2005; 21:280. [in Chinese]
12. Fang XJ, Xiao P, Zhu RY, Liang CH, Huang B. MRI imaging features and diagnosis of pituitary stalk interruption syndrome. Med World. 2007; 02:16-7. [in Chinese]
13. Tian J, Wang GB, Li GM, Xie BR. MR imaging diagnosis of pituitary stalk interruption syndrome. J Prac Radiol. 2007; 23:1153-5. [in Chinese]
14. Cheng H, Zeng JJ, Li H, Sun GQ. MRI Features of pituitary stalk interruption syndrome: correlation with growth hormone deficiency. J Clin Radiol. 2008; 27:821-4. [in Chinese]
15. Li XR, Ou SX, Jin WS. Imaging diagnosis of pituitary stalk interruption syndrome. J Prac Radiol. 2008; 24: 1457-9. [in Chinese]
16. Zheng KH, Wang SH, Ren AJ, Huang MH, Shi LJ, Guo Y. Pituitary stalk interruption syndrome: a case report. J Naval General Hospital. 2009; 22:57. [in Chinese]
17. Tu WG, Yu YQ, Song B, Wang L, Zhang Y. Low field MR diagnosis of pituitary stalk interruption syndrome. Chinese J Med Imag Tech. 2009; 25:993-5. [in Chinese]
18. Xie LL, Yang Y, Gong LG, Yang L, Zhou B, Wu X, et al. Changes of pituitary magnetic resonance imaging and clinical manifestations in short stature children with growth hormone deficiency. J Applied Clin Pediatr. 2009; 24:1569-70. [in Chinese]
19. Rong ZH, Li Z, Zhao X, Wang XX. Case report of children pituitary stalk interruption syndrome. Clin Focus. 2010; 25:12. [in Chinese]
20. Sang YH, Dong RH. Diagnosis and Treatment of Pituitary Stalk Interruption Syndrome: 8 case reports. J Bethune Military Medical College. 2010; 8:196-7. [in Chinese]
21. Ren DW, Zhang DL. MRI diagnosis of pituitary stalk interruption syndrome. J Chin Clin Med Imag. 2010; 21:499-500. [in Chinese]
22. Zheng KH, Chen XQ, Luo QH, Ding Y. Pituitary stalk interruption syndrome: a case report. Modern J Integrated Trad Chinese Western Med. 2010; 19: 3316-7. [in Chinese]
23. Quan HB, Fu SH, Chen WJ, Li JJ. One case report of Pituitary Stalk Interruption Syndrome. Chongqing Medicine. 2010; 39:1174-5. [in Chinese]
24. Lu SJ. Pituitary stalk interruption syndrome: a case report and literature review. Acta Medicinae Universitatis Scientiae et Technologiae Huazhong. 2010; 39:871-2. [in Chinese]
25. Lia YX. Pituitary stalk interruption syndrome: a case report and literature review. Chin J Misdiagnostics. 2011; 11:989. [in Chinese]
26. Pan QW, Zhu HJ, Gong FY, Li NS, Zhang T, Bai G, et al. Magnetic resonance image of sellar regionin pituitary stalk interruption syndrome in children and adolescents. Acta Academiae Medicinae Sinicae. 33:9-13. [in Chinese]
27. Lu WG, Gao ZH. Clinical and MRI analysis of pituitary stalk interruption syndrome. Chin J CT MRI. 2011; 9:22-4. [in Chinese]
28. Zheng XP, Liu BH, Dai XY, Chen XC. Nursing care of

pituitary stalk interruption syndrome. Chin J Nurs. 2011; 46:406-7. [in Chinese]
29. Liu MY, Feng F, You H, Li ML, Jin ZY. MRI imaging features of pituitary stalk interruption syndrome. Chinese J Med Imag. 2011; 19:383-5. [in Chinese]
30. Dong SH, Guo Z, Wang H. Case reports of pituitary stalk interruption syndrome with optic atrophy. Chin J Strabismus Pediatr Ophthal. 2011; 19:72. [in Chinese]
31. Zheng M, Ye SD, Xiao CC, Ren A. Misdiagnosis reason of pituitary stalk interruption syndrome. Clin Misdiagnosis Mistherapy. 2011; 24:56-7. [in Chinese]
32. Shu T, Xiao XL, Yin JH, Tang XP. Etiological analysis on pituitary stalk interruption syndrome. Maternal Child Health Care China. 2012; 27:853-6. [in Chinese]
33. Wu WX, Li ZZ, Qin GJ, Dong YG, Ren CP. Pituitary stalk interruption syndrome: a case report and literature review. J Zhengzhou Univ (Medical Sciences). 2012; 47:134-6. [in Chinese]
34. Zhang JW, Yu GY. Clinical and MRI features of pituitary stalk interruption syndrome. Acta Academiae Medicinae Wannan. 2012; 31:481-3. [in Chinese]
35. Chen F, Dou JT, Lv CH, Yang GQ, Du J, Guo QH, et al. Clinical characteristics of female idiopathic hypogonadotropic hypogonadism and pituitary stalk interruption syndrome. J Shanxi Medical University. 2012; 43:125-8. [in Chinese]
36. Wan NJ, Song FY, Chen XB, Yan XY. Clinical Analysis of 4 Cases of pituitary stalk interruption syndrome in children. J Applied Clin Pediatr. 2012; 27:588-90. [in Chinese]
37. Cao MP, Wen XY, Chen Y. Pituitary stalk interruption syndrome: a case report. Guangdong Med J. 2012; 33:2220. [in Chinese]
38. Wang GZ. MRI diagnostic value on pituitary stalk interruption syndrome. 2012; 12:447. [in Chinese]
39. Li X, Lu ZX, Chen XH, Ge H, Zhuang GY, Tian CC. Diagnosis value of MRI for pituitary stalk interruption syndrome. Chin Med Herald. 2013; 10:107-9. [in Chinese]
40. Liang ZL, Yao B, Wang LC, Xu Y. Clinical analysis of 7cases of pituitary stalk interruption syndrome. Chin Clin Prac Med. 2010; 4:8-10. [in Chinese]
41. Wang GL, Fu JF, Wang CL, Liang L. Clinical analysis of 13 cases with growth hormone deficiency combined with pituitary stalk interruption. Chin J Pediatr. 2010; 48:305-7. [in Chinese]
42. Ye HY, Li QH, Wu X, Yang YH, Wen J, Lu B, et al. Clinical diagnosis of 5 cases of pituitary stalk interruption syndrome. Chin J Endocrinol Metab. 2008; 24:483-5. [in Chinese]