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Case Report

Importance of medical imaging in ovarian hyperstimulation syndrome post suction and evacuation of H-mole: a rare case report and literature review

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ABSTRACT

Hydatidiform mole (H-mole) is a gestational trophoblastic disorder caused by abnormal fertilization, often presenting with elevated β-HCG levels and abnormal uterine findings. A rare complication is spontaneous Ovarian Hyperstimulation Syndrome (OHSS), usually associated with fertility treatments but occasionally triggered by high endogenous β-HCG levels. We report a case of a 17-year-old primi-gravida who developed OHSS following suction and evacuation of a complete H-mole. Despite of initial management, she presented later with ovarian enlargement, lung nodules, and rising β-HCG, indicating high-risk gestational trophoblastic neoplasia (GTN) for which imaging played a key role in identifying complications. She responded well to EMA-CO chemotherapy with normalization of β-HCG. This case emphasizes the importance of post-evacuation monitoring and the critical role of imaging in diagnosing and managing rare but serious complications like spontaneous OHSS.

Keywords: Ovarian hyperstimulation syndrome, Hydatidiformmole, Gestational trophoblastic disease/neoplasia, β-HCG, Suction and evacuation, Dilatation and evacuation, Retained product of conception

INTRODUCTION

Hydatidiform mole (H-mole**)** is a benign yet potentially malignant gestational trophoblastic disorder caused by abnormal fertilization. Diagnosis is based on ultrasound and elevated β-HCG. Complete moles (46XX/XY) carry a 15–20% risk of persistence, while partial moles (69XXX/XXY) have a 1–5% risk. Risk factors include maternal age extremes, poor nutrition, and previous molar pregnancy. Complete and partial moles are the most common GTD types, characterized by abnormal trophoblastic growth and embryonic development. Risk increases significantly in women <21 or >40 years and with prior molar history.[5] Common symptoms include amenorrhea, vaginal bleeding, high β-HCG, and passage of grape-like vesicles.[1] Hydatidiform moles, though rare, have high metastatic potential. D and E or D and C is the treatment of choice.[9]

Ovarian hyperstimulation syndrome (OHSS) is typically iatrogenic, resulting from ovarian stimulation drugs. OHSS is usually linked to fertility treatment, it has been reported in natural pregnancies—especially those involving HM.[8] Severe OHSS occurs in ~1% of

gonadotropin cycles.[3] It ranges from mild (5–10%) to severe (0.1–0.5%). It may occur spontaneously in the presence of high β-HCG levels. Risk factors of OHSS include young age, low BMI, PCOS, high estrogen, past OHSS episodes and high level of TSH which stimulate ovaries.[2,3] The pathophysiology of OHSS is not fully understood, literature describes that high levels of β-HCG leads to the release of vasoactive substances by the ovaries, such as interleukins and tumor. As a result vasodilatation occurs promoting mesothelialhyperpermeability with fluid leakage into the extravascular space. This process results in significant clinical consequences, including hemoconcentration, hypovolumia, worsened renal function, and hypercoagulability. Recent studies linked a mutation in the FSH receptor gene, which increases its basal activity and sensitivity to β-HCG, with the development of OHSS.[2-4] Spontaneous OHSS is a recognized complication of H-Mole due to high β-HCG levels. Risk may increase post D and E and clinicians must watch for ovarian cysts and manage complications promptly.[10] We reported extremely rare cases of OHSS in spontaneous pregnancies, especially with post S and E of invasive moles.

CASE REPORT

A 17yearsyoung female patient, married for an year, primi-gravida, was admitted to our hospital with complaints of amenorrhea for 3 months, dull and non- radiating abdominal pain for 1 month, PV bleeding with passage of clots for 5-6 days for 1 month, nausea and vomiting for 1 day.

On examination her vitals were normal. Her chest, CVS and abdominal examination were normal. On P/S examination: cervix was healthy without active bleeding, and on PV examination: uterus was 14–16-week size with B/L fornix free.

Patient symptoms were aggravated for 1 month prior to admission. There were no medical or surgical significant past history. A pelvic USG scan performed which showed bulky uterus with increased parenchymal echotexture, endometrial cavity showed mixed echogenic lesion of size measuring 83x65mm with multiple cystic focus and with minimal flow on colour-doppler within endometrial cavity, findings were consisted with H-mole. On day of admission β-HCG was 273600 mIU/ml. On 2nd day of admission suction and evaluation under USG guidance was done, 500ml of POC was obtained and sent for HPE. Patient was discharged on 3rd day of admission with antibiotic coverage and with other supportive treatments. She was advised to follow-up in 1 week with β-HCG and HPE report. On follow-up, HPE report showed complete hydatiform mole (Figure 1A, B) and β-HCG was 6, 68,339 mI U/ml.

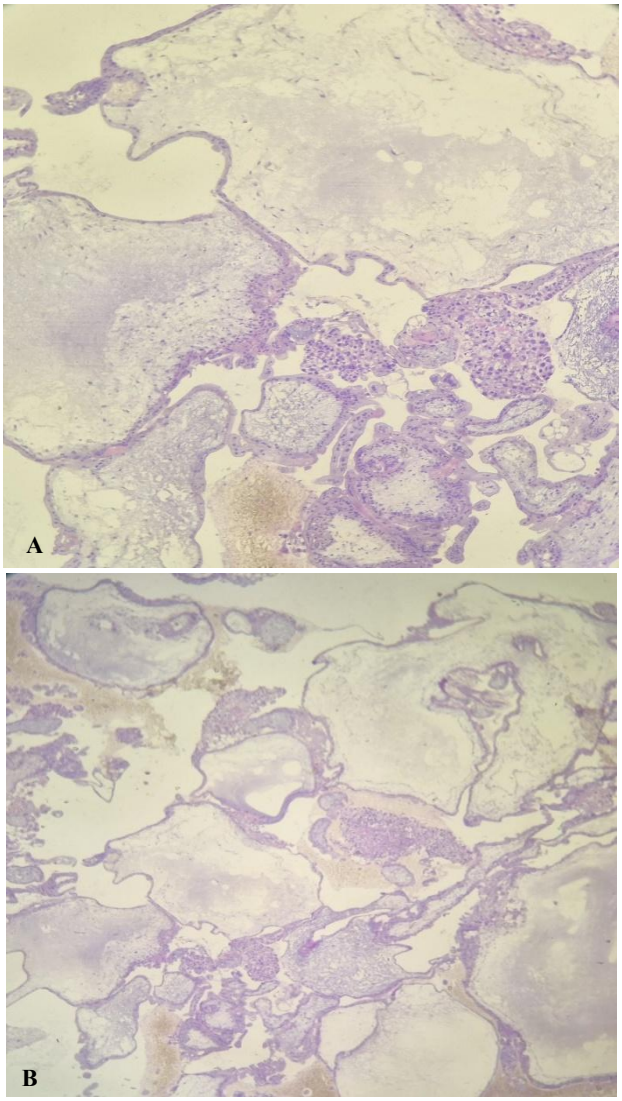

Figure 1: (A) Complete hydatidiform mole. (at low power magnification, 10x) well- developed complete mole shows large hydropic villi with cistern formation and circumferential trophoblastic proliferation and (B) complete hydatidiform mole (at low power magnification, 4x). Diffuse enlargement of chorionic villi with cistern formation and lined by attenuated trophoblasts.

After 35 days of discharge, she was again admitted with complaints of nausea and vomiting for 4 days, non-bilious vomitus mixed with food particles without blood for 4-5 episodes per day and anorexia for 4 days. β- HCG was 273600 mIU/ml. On repeat USG on day of admission there was bulky uterus with heterogeneous component within endometrial cavity, endometrium was 11.0 mm thick. B/L adnexa show enlarged multiseptated and multi loculated ovaries, right measuring 102×54×91 mm and left measuring 112×56×91 mm. Patient advised for CECT abdomen and pelvis.

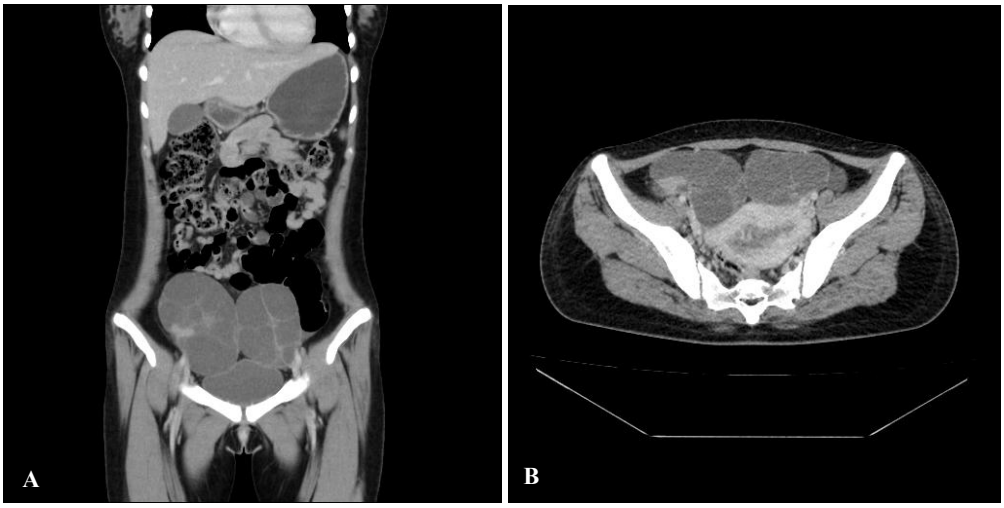

Figure 2: (A) Multicystic-multisepated spoke wheel appearance of B/L enlarged ovary and (B) B/L enlarged multicystic-multiseptated ovary with bulky uterus and hypodense endometrial component (Heterogeneously enhancing tiny cystic areas & endometrial fluid).

On 4th day of 2nd admission, USG was followed by CECT chest and abdomen on same day which showed few small hyper-attenuating nodule in B/L lungs likely metastasis, B/L minimal pleural effusion, Bulky uterus with ill-defined heterogeneous area with multiple cystic spaces and an enhancing component (53×59×36 mm), also extending into the myometrium of anterior uterine wall which signifies an invasive components and endometrial collection, B/L enlarged multi-lobulated and multi-cystic ovaries, Rt. 96cc and Lt. 90 cc lesion likely ovarian hyperstimulation and mild hepato-splenomegaly (Figure:2 A,B). On the basis of all findings and clinical assessment she was kept under high risk GTN and EMA- CO regimen chemotherapy was started.

On 6th day of 2nd admission repeat USG showed bulky uterus with RPOC measuring 27×26 mm within endometrial cavity and B/L enlarged multi-septated ovaries, right measuring 76×72×76 mm & left measures 69×71×72 mm.

On 24th day of 2nd admission repeat USG showed bulky uterus with RPOC measuring 20×18 mm within endometrial cavity and B/L ovaries were unremarkable.

There were complete regression and normalization of β-HCG after completion of therapy.

DISCUSSION

This case underscores the rare but critical complication of spontaneous ovarian hyperstimulation syndrome (OHSS) following a complete hydatidiform mole (CHM). Traditionally, OHSS is an iatrogenic condition associated with ovulation induction in assisted reproductive technologies. However, rare instances, such as in this case, illustrate that it may develop spontaneously in the setting of gestational trophoblastic disease due to excessive β-HCG secretion.

The patient presented with classic signs of molar pregnancy, vaginal bleeding, an enlarged uterus, and extremely elevated β-HCG levels. Initial imaging revealed characteristic features consistent with an H-mole. Management with suction and evacuation was performed, which aligns with the standard approach for CHM. Notably, a significant surge in β-HCG was observed post-suction and evacuation, reaching 668,339 mIU/mL, which is suggestive of persistent trophoblastic disease.

Subsequent clinical deterioration and imaging revealed bilateral ovarian enlargement with multi-loculated, multi-septated cysts, invasive uterine lesion and pulmonary nodules, hall mark of both OHSS and high-risk gestational trophoblastic neoplasia (GTN). The pivotal role of serial imaging (USG and CECT) in tracking the disease progression and identifying complications such as metastasis and ovarian changes is well demonstrated in this case.

Moreover, this case draws attention to the timing and duration of risk for spontaneous OHSS after molar evacuation. While most reported cases develop within the first one to two weeks, our patient presented more than a month later, suggesting that heightened vigilance must extend beyond the early post-evacuation period. Such delayed onset may reflect ongoing trophoblastic activity, fluctuating hormone levels, or delayed ovarian responsiveness to β-HCG, and highlights the importance of regular β-HCG surveillance and imaging even when initial follow-up results appear reassuring. Early involvement of a multidisciplinary team including gynecologic oncology, radiology, and critical care is crucial to promptly recognize progression, tailor chemotherapy regimens, and prevent life-threatening sequelae such as thromboembolism or respiratory compromise.

Table 1: Summary of reported cases of OHSS accompanied by chorionic diseases.

References	Age (years)	Gestation (weeks)	Gravid and Para status	Pathology	Outcome of pregnancy	HCG before treatment for chorionic disease	HCG at occurrence of OHSS	Occurrence time after treatment	Maximum size of enlarged ovary	Massive ascites	Pleural effusion	Main treatment for OHSS	Duration of hospitalization	Duration of recovery to normal-sized ovary
Hooper et al[8]	25	7	G0P0	Mole (type, not described)	Spontaneous abortion	Not described	Not described	7 days	Right:17.5 cm Left:12.5cm	+	Not described	Paracentesis, right salpingo-oophorectomy, left wedge resection	Not described	6 weeks
Moneta et al[8]	25	16	G2P0	Mole (type, not described)	Legal induced abortion	280,000 IU/24h	About 5000 IU/24h	8 days	Right: about 10 cm Left: about 10cm	+	+	Paracentesis, infusion, albumin, blood transfusion	1 month	Not described
Ludwig et al[8]	Not described	14	G3P1	Partial hydatidiform mole	Legal induced abortion	1350 IU/L	7320 IU/L	14 days	Right:17x10x15cm Left:16x10x14cm	+	+	Infusion, heparin	6 days	Not described
Arora et al[8]	23	12	G3P2	Partial hydatidiform mole	Legal induced abortion	400,000 IU/L	65,554 IU/L	3 days	Right:11×11.2cm Left:13.6×12.8cm	+	+	Paracentesis, infusion	11 days	Not described
Strafford et al[8]	19	12	G1P0	Complete hydatidiform mole	Legal induced abortion	811,506IU/L	23,681IU/L	6 days	Right:12x8x7cm Left:9x8x11cm	+	+	Infusion, heparin, pleural drainage	20 days	3 months
Rachad et al[8]	34	12	G4P3	Invasive mole	Ovarian drilling followed by chemotherapy	2,000,000IU/L	2,000,000 IU/L	0	Right:16cm Left:12cm	+	+	Infusion, albumin, heparin, chemotherapy, Paracentesis, hysterectomy	2 months	2 months
Zhou et al[8]	38	16	G5P4	Complete hydatidiform mole	Legal induced abortion	860,000IU/L	45,674IU/L	7 days	Right:12.1 cm	+	+	Paracentesis, thoracocentesis, albumin	2 weeks	3 months

Continued.

References	Age (years)	Gestation (weeks)	Gravid and Para status	Pathology	Outcome of pregnancy	HCG before treatment for chorionic disease	HCG at occurrence of OHSS	Occurrence time after treatment	Maximum size of enlarged ovary	Massive ascites	Pleural effusion	Main treatment for OHSS	Duration of hospitalization	Duration of recovery to normal-sized ovary
									Left:11.7cm					
Diness et al[8]	29	15	Not described	Partial hydatidiform mole	Legal induced abortion	151,000IU/L	5220IU/L	3 days	Right:14cm Left:14cm	+	–	Infusion	9 days	4 months
Suzuki et al[8]	31	10	G not described P2	Partial hydatidiform mole	Legal induced abortion	390,000IU/L	9830IU/L	8 days	30 cm (not described about right or left)	+	+	Not described	9 days	1 month
Wu et al[8]	29	10	G1P0	Partial hydatidiform mole followed by invasive mole	Legal induced abortion followed by chemotherapy	225,000IU/L	67,800IU/L	19 days	Right:17.2×15.9×17.8 cm Left:11.4×18.1×17.7cm	+	+	Infusion, albumin, heparin, chemotherapy	A few days	7 months
Davoudian et al[8]	20	16	G1P0	Placental Mesenchymal dysplasia	Spontaneous Abortion	Not described	24,890IU/L	Not described	Right:10cm Left:9cm	–	–	–	0	A few weeks
Tsubokura et al[8]	16	12	G1P0	Complete hydatidiform mole	Legal induced abortion	980,823IU/L	44,815 IU/L	4 days	Right: 10cm x12cm Left:25cm x11cm	–	–	–	0	8 months
Cohen et al[11]	19	15.2	G2P1A0	Partial hydatidiform mole	Legal induced abortion	1.3million mIU/mL	1.3million mIU/ML	0	Right: 13.5x11.1x 8.8cm Left:13.5x 13x10.8cm	+	+	Methotrexate, Thoracocentesis, Paracentesis, IV fluids, albumin, diuretics and pain control.	Not described	Not described
Lemrabott et al[13]	27	10	G3P2	Complete hydatidiform mole	Legal induced abortion	300,000 IU/L	300,000IU/L	0	Not described	Grading not done	+	Not described	17days	1 month

Continued.

References	Age (years)	Gestation (weeks)	Gravid and Para status	Pathology	Outcome of pregnancy	HCG before treatment for chorionic disease	HCG at occurrence of OHSS	Occurrence time after treatment	Maximum size of enlarged ovary	Massive ascites	Pleural effusion	Main treatment for OHSS	Duration of hospitalization	Duration of recovery to normal-sized ovary
Mishra et al[12]	25	17	G3P2	Complete hydatidiform mole	Legal induced abortion	10,00,000 mIU/mL	10,00,000 mIU/mL	0	Right: 7.4x4.2x5.1cm Left: 6.4x3.2x5.9cm	–	–	Supportive treatment	6days	Not described
Begum et al[10]	30	14	G3P2	Complete hydatidiform mole	Legal induced abortion	625,000 IU/L	Not described	8 days	Right: 9.0x8.4x5.5cm Left:13.0x8.4x9.7cm	–	–	Supportive treatment	Not specified	42 days
Guimaraes et al[4]	38	Not described	G2Pnot described	Complete hydatidiform mole	Legal induced abortion	1,000,000 mIU/mL	48,769 mIU/mL	10 days	Right: 1329 cm3 Left:500 cm3	–	–	IV fluids, DVT prophylaxis, pain management, Anti-emetics, MTX and folinic acid.	35 days	Only left ovary normalized in 6 months.
Our patient	17	12	G1P0	Complete hydatidiform mole	Legal induced abortion followed by chemotherapy	273600 mIU/mL	273600 mIU/mL	35 days	Right: 10.2cm x5.4cmx9.1cm Left:11.2cmx 5.6cmx9.1cm	–	+	Supportive treatment Chemotherapy	9 days	5months

Spontaneous form of OHSS generally develops between 8 and 14 weeks of amenorrhea, differing from iatrogenic OHSS, which usually starts between 3 and 5 weeks of amenorrhea. The recent identification of mutations in the follicle stimulating hormone (FSH) receptor gene, which display an increased sensitivity to β-HCG and are responsible for the development of spontaneous OHSS. In iatrogenic OHSS, the follicular recruitment and enlargement occur during the administration of exogenous FHS. In the spontaneous form however, the follicular recruitment and growth occur later through the promiscuous stimulation, by pregnancy-derived β-HCG, of a mutated FSH receptor that is abnormally sensitive to β-HCG or a wild type FHS receptor in the presence of abnormally high level of β-HCG. Thus, the symptomatology of spontaneous cases of OHSS usually develops at 8 weeks' amenorrhea and culminates at the end of the first trimester of pregnancy.[3] In hyperstimulated ovaries, there is increased production of pro-inflammatory mediators such as VEGF. Increased vascular permeability leads to accumulation of fluid in the third space, manifested by dehydration, hypovolaemia, oliguria, haemoconcentration, reduced osmolality, electrolyte imbalance (including hyponatremia and hyperkalaemia), ascites, and pleural or pericardial effusion.[3-10] OHSS is classified as mild, moderate, or severe depending on the clinical symptoms and blood test findings. Treatments are done on the basis of severity ranging from observation (Mild) to symptomatic treatment (Moderate to Severe).[8] We have summarized previously reported cases to best of our knowledge (Table: 1) along with our case.

Importantly, this case reinforces the clinical lesson that post-evacuation monitoring is essential, particularly in patients with high β-HCG levels and H-moles, which are known to have a higher risk of developing into OHSS. The detection of lung metastases and invasive uterine changes on imaging prompted timely initiation of EMA-CO chemotherapy, which resulted in normalization of β-HCG levels and clinical improvement, confirming its effectiveness in high-risk GTN.

Contraception for at least 6–12 months after normalization of β-HCG remains the standard recommendation to reduce the risk of a new pregnancy. Psychological support is equally important, as young patients may experience anxiety and depression regarding malignancy and fertility

CONCLUSION

This case exemplifies the rare but clinically significant association between hydatidiform mole and spontaneous ovarian hyperstimulation syndrome. It emphasizes the importance of medical imaging in considering OHSS in the diagnosis when molar pregnancy is complicated by ovarian enlargement, especially with persistently elevated β-HCG levels post-evacuation. Prompt recognition, aggressive monitoring, and timely initiation of chemotherapy are critical for preventing serious complications such as metastatic spread leading to morbidity and mortality.

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REFERENCES

1. Solo VE, Tamphasana A, Laishram G, Singh LR, Praneshwari Devi R, Nungsangtemjem. A clinical study of hydatidiform mole. IOSR J Dent Med Sci. 2019;18(7):15-9.
2. Alhalabi K, Lampl BS, Behr G. Ovarian hyperstimulation syndrome as a complication of molar pregnancy. Cleve Clin J Med. 2016; 83(7):504-6.
3. Rachad M, Chaara H, Fdili FZ, Bouguern H, Melhouf A. Ovarian hyperstimulation syndrome in a spontaneous pregnancy with invasive mole: report of a case. Pan Afr Med J. 2011;9:23.
4. Guimarães JB, Bruno A, Silveira MMP da, Rodrigues ARN, Brito MB. Exceptional case of spontaneous ovarian hyperstimulation syndrome in complete molar pregnancy: a case study. Braz J Case Rep. 2024; 4(4):43–9.
5. Riccio S, Galanti F, Scudo M, Di Troia L, Ferrillo MG, Manzara F, et al. Recurrent hydatidiform moles: a clinical challenge, A case report and an update on management and therapeutical strategies. Case Rep Obstet Gynecol. 2023;2023:3752274.
6. Hafezi M, Chekini Z, Zamanian M. Which one is more prominent in recurrent hydatidiform mole, ovum or sperm?. Int J Fertil Steril. 2020;14(2):154–8.
7. Sousa I, Sampaio A, Sampaio J, Mendonça R, Rocha T, Ponte C, et al. Partial hydatidiform mole with spontaneous ovarian hyperstimulation syndrome. Gynecol Reproduct Endocrinol. 2020;4(2):1-5.
8. Tsubokura H, Ikoma Y, Yokoe T, Yoshimura T, Yasuda K. Ovarian hyperstimulation syndrome following surgical removal of a complete hydatidiform mole: a case report. J Med Case Rep. 2019;13(1):292.
9. Tanaka Y, Furuya K, Sumi M, Yamashita S, Chang Y, Shikado K, et al. Multidisciplinary perioperative management in dilatation and evacuation for a giant hydatidiform mole: A case report. Case Rep Women's Health. 2023;40:e00556.
10. Begum DA, Fatema F, Yesmin F. Ovarian Hyperstimulation Syndrome Post-dilation and

Evacuation of a Hydatidiform Mole: A Case Report. EMJ Repro Health. 2023.
11. Cohen E, Lanzer JL, Mittal P. Comparison of the Clinical Presentation of Ovarian Hyperstimulation Syndrome in a Partial Molar Pregnancy Case Versus a Fertility Treatment Case. Cureus. 2019;11(5): e4718.
12. Mishra A, Gaire S, Mahat P, Neupane J, Chaubey MK, Bipana KC. Ovarian hyperstimulation syndrome complicating spontaneous molar pregnancy: a case report. J General Pract Emerg Med Nepal. 2022;9(14):82–5.
13. Lemrabott E, Mahmoud M, Saoud M, Mamouni N, Errarhay S, Banani A, et al. Ovarian hyperstimulation syndromeas a complication of molar pregnancy: case report. Service de Gynecologie Obstetrique I CHU Hassan II, Fes.Marco Universite Sidi Mohamed Ben Abdellah Fes. IJAR. 2020;8(11):836–9.

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