

# A Case Report of a Pregnant Woman with Cystoid Transformation of a Pheochromocytoma Complicated by Bleeding, Initially Diagnosed as “Preeclampsia”

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## Case Report

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# Abstract

Pheochromocytoma is one of the rare causes of gestational hypertension. This case report reviews and analyzes a case of atypical cystic pheochromocytoma with hemorrhage that presented at the onset of labor at 36 + 6 weeks of gestation. It describes the diagnostic and therapeutic process and discusses methods for hormone level testing and imaging examinations in pregnancy-associated pheochromocytoma, as well as the clinical manifestations and treatment strategies for pheochromocytoma under different pathophysiological conditions. This case highlights a clinical challenge: due to the unique nature of pregnancy, pheochromocytoma during pregnancy lacks specific symptoms. Consequently, there are challenges in the timely diagnosis of pheochromocytoma during pregnancy and in the treatment of pheochromocytoma crises.

## Introduction

Pheochromocytoma is a tumor originating in the adrenal medulla, sympathetic ganglia, or other sites containing chromaffin tissue. It can secrete large amounts of catecholamines either paroxysmally or continuously; it may present with no symptoms or signs, or it may suddenly lead to cardiopulmonary failure, intracerebral hemorrhage, shock, and other complications<sup>[1]</sup>. Pheochromocytoma during pregnancy is extremely rare, with an incidence rate of only 0.002%, but it is associated with high maternal and fetal mortality rates<sup>[2]</sup>. In currently reported cases of pheochromocytoma during pregnancy, early diagnosis is often delayed or missed. The primary reasons are that gestational hypertension, eclampsia, and preeclampsia are more common than pheochromocytoma and are easily overlooked, or that the lack of specific symptoms leads to confusion with hypertensive disorders of pregnancy. The mortality rate for pregnant women and fetuses who do not receive timely diagnosis and treatment reaches 50%; conversely, when diagnosed early and treated aggressively and appropriately, maternal and fetal mortality rates drop to 5% and 15%, respectively<sup>[3]</sup>. Therefore, the timely identification of pheochromocytoma among cases of gestational hypertension is of critical importance.

This paper reports a case of postpartum diagnosis of cystic degeneration of pheochromocytoma complicated with hemorrhage during pregnancy and discusses the complexity of its diagnosis and treatment.

## Case Report

Patient, female, 32 years old, with one previous pregnancy and no previous deliveries. She has a regular menstrual cycle. Her last menstrual period was April 12, 2025, and her estimated due date is January 19, 2026. She did not undergo regular prenatal checkups during her pregnancy. Blood pressure readings during prenatal visits fluctuated within the normal range. She experienced no blurred vision, palpitations, or shortness of breath during pregnancy, nor did she have dizziness, headaches, abdominal discomfort, or edema in both lower limbs. At 13:00 at 36 + 6 weeks of gestation, the patient developed intermittent dizziness and headache without any apparent cause. This was followed by nausea and vomiting of a small amount of gastric contents; the symptoms of dizziness and headache subsided slightly after vomiting. She subsequently sought medical attention at a local hospital, where she was administered oral vitamin B6 tablets and an intramuscular injection of "metoclopramide," but these treatments were ineffective, leading to her transfer to our hospital. On admission, her temperature was 36.5°C, pulse 132 beats/min, respiratory rate 24 breaths/min, and blood pressure 132/92 mmHg. Laboratory findings showed urine protein 3+ and creatinine 90.80 µmol/L. Obstetric examination revealed an abdominal circumference of 95 cm and a fundal height of 30 cm; no uterine contractions were present, and she was not in labor. Fetal heart rate monitoring: baseline 180 beats per minute, with persistent prolonged decelerations lasting 6 minutes, indicating acute fetal distress. A preliminary diagnosis of severe preeclampsia was made. Given the critical nature of the situation, a cesarean section via a transverse lower uterine segment incision was performed immediately.

During preoperative preparation, the patient suddenly experienced convulsions and lost consciousness. Auscultation revealed no fetal heart tones, so she was rushed to the operating room for a lower segment cesarean section. A transverse incision was made in the lower segment of the uterus; the amniotic fluid was clear, with a volume of approximately 400 mL. A live female infant was delivered in the cephalic presentation, weighing 2,710 g, with a 1-minute Apgar score of 2. After endotracheal intubation, the infant was transferred to the neonatal ward (5-minute Apgar score of 6, 10-minute Apgar score of 8).

Intraoperatively, complete separation of the placenta and membranes was observed, but uterine contractions were weak. After administering 100 µg of carboprost intravenously and 250 µg of carboprost trisilolate solution via fundal injection, uterine contractions improved. Intraoperative blood loss was approximately 300 mL. Exploration of the uterus and both adnexa during the procedure revealed no abnormalities. During surgery, the patient's blood pressure fluctuated between 80–200 mmHg/47–110 mmHg, heart rate between 100–120 beats per minute, and oxygen saturation at 100%. Upon completion of the procedure, the patient continued to experience seizures; the endotracheal tube was left in place, and the patient received sedation, analgesia, antispasmodic therapy, and mechanical ventilation. Postoperatively, her heart rate fluctuated between 130 and 170 beats per minute, her blood pressure fluctuated between 60–190/40–90 mmHg, and her temperature was 38.9°C. Postoperatively, nifedipine was administered via infusion to lower blood pressure, and esmolol was administered via infusion to control heart rate. When blood pressure dropped to 90/60 mmHg, crystalloid solution (30 mL/kg, 500 mL/h) was administered for symptomatic fluid replacement, and norepinephrine and metaraminol were infused intravenously to symptomatically raise blood pressure. A postoperative echocardiogram revealed normal cardiac systolic function and left ventricular wall thickening; an abdominal ultrasound revealed a hypoechoic mass (measuring 11 cm × 10 cm) visible between the left kidney and the spleen; a left adrenal hematoma or retroperitoneal hematoma could not be ruled out. Postoperative blood tests showed: Hb 150 g/L, RBC  $5.23 \times 10^{12}/L$ . Within 6 hours postoperatively, blood pressure fluctuated between 80–200/40–100 mmHg, and the patient was subsequently transferred to the Department of Critical Care Medicine.

The patient's postoperative temperature was 39°C, and all inflammatory markers were elevated to varying degrees; however, the alternating episodes of hypertension and shock did not meet the criteria for septic shock, so this diagnosis was preliminarily ruled out. Postoperative follow-up revealed markedly elevated cardiac enzymes, but the electrocardiogram (ECG) showed only mild ST-segment depression. Echocardiography indicated normal left ventricular dimensions with an LVEF of approximately 57%, essentially ruling out peripartum cardiomyopathy. There were no obvious signs of blood loss postoperatively, and laboratory results showed Hb at 150 g/L, which also preliminarily ruled out the possibility of hemorrhagic shock (see Table 1 for postoperative inflammatory markers, cardiac enzyme levels, etc.). Twelve hours postoperatively, the patient's heart rate fluctuated between 150 and 170 beats per minute, and blood pressure fluctuated between 50–90/30–60 mmHg. Symptomatic fluid resuscitation was administered, and norepinephrine and methoxamine were infused via an IV pump to raise blood pressure, while esmolol was infused via an IV pump to control ventricular rate. During actual monitoring, the patient's blood pressure fluctuations appeared to show no correlation with the infusion doses of vasoactive agents. Approximately 20 hours postoperatively, the patient's blood pressure dropped to 40–60/20–40 mmHg and remained at this level for more than 30 minutes; at this time, high-dose infusions of norepinephrine and metaraminol were still being administered. Upon further review of the medical records and in conjunction with postoperative ultrasound findings, it was considered highly likely that the patient had an adrenal hematoma and was experiencing an adrenal crisis. Consequently, 100 mg of hydrocortisone was administered via slow, continuous infusion. During this period, the patient's blood pressure appeared to stabilize, fluctuating between 80–120/50–60 mmHg. Twenty-four hours postoperatively (4 hours after blood pressure had stabilized relatively with hydrocortisone), blood pressure began to fluctuate widely once again, characterized primarily by sudden spikes and drops, with irregular alternation between hypertension and shock. Although the infusion rates of vasoactive agents were adjusted based on blood pressure trends and symptomatic fluid therapy was administered, neither measure produced a significant effect. Since these hemodynamic manifestations did not align with the typical presentation of an adrenal hematoma complicated by an adrenal crisis, hydrocortisone was discontinued. A subsequent abdominal CT scan revealed a massive mass (12.6 cm × 9.4 cm × 8.8 cm) in the left adrenal region with cystic changes and hemorrhage, highly suggestive of a pheochromocytoma (Fig. 1). At this point, the patient's blood pressure fluctuations were considered to be caused by a "left-sided pheochromocytoma with cystic changes and hemorrhage, presenting as a pheochromocytoma crisis," and catecholamine-related tests were completed. Treatment involved precise fluid resuscitation and regulation of vasoactive drug infusion rates under PICCO guidance to maintain relatively stable cardiac function, adequate intravascular volume, and relatively moderate vascular resistance; in addition, α-receptor blockers (intravenous infusion of uradil and nasogastric administration of terazosin) were administered in combination to control blood pressure symptomatically and prevent hypertensive crisis, heart failure, and severe cerebrovascular adverse events caused by excessively high blood pressure.

Table 1  
Inflammatory Markers, Myocardial Enzymes, Echocardiography, Electrocardiogram, etc.

| WBC                     | NEUT%  | PCT        | CRP      | IL-6     | BNP          | CK     | CK-MB   | HBDH   | LDH    |
|-------------------------|--------|------------|----------|----------|--------------|--------|---------|--------|--------|
| 15.0×10 <sup>9</sup> /L | 86.60% | 5.157ng/ml | 38.6mg/L | 109pg/ml | 1969.88pg/ml | 957U/L | 44.4U/L | 358U/L | 498U/L |

Laboratory results showed that the patient's serum levels of catecholamines—including dopamine, norepinephrine, and epinephrine—were significantly elevated (Table 2), further confirming the diagnosis of pheochromocytoma. On the fifth postoperative day, the patient's blood pressure gradually stabilized, fluctuating between 90–120/40–70 mmHg (with no symptoms of hypotensive shock). Norepinephrine was gradually tapered and discontinued, while the patient continued to take terazosin and received intravenous urapidil via an infusion pump to control blood pressure. On the sixth postoperative day, the patient was transferred to a general ward; on the fifteenth postoperative day, the patient was transferred to a higher-level hospital to undergo left adrenalectomy. Intraoperatively, a hard mass measuring approximately 12.6 cm × 9.4 cm × 8.8 cm was found within the left adrenal gland (Figs. 2 and 3). Histopathological examination confirmed the diagnosis of a pheochromocytoma. On the day of surgery, blood pressure remained stable at 100–110/60–70 mmHg. The patient was discharged on the 7th postoperative day and did not continue taking any medication after discharge. At the six-month follow-up, the patient's blood pressure fluctuated around 110/70 mmHg, and the patient reported no significant discomfort. Three months after discharge, follow-up tests showed that markers such as blood catecholamines were within normal limits (Table 2).

Table 2  
Detection of Pheochromocytoma Markers

| Test Items                         |                       | Pre-operative   | Post-operative (3 months) | Reference Values     |
|------------------------------------|-----------------------|-----------------|---------------------------|----------------------|
| Plasma methoxyadrenergic analogues | 3-Methoxytyramine     | 0.51nmol/L      | < 0.08                    | ≤ 0.1nmol/L          |
|                                    | Methoxyadrenaline     | 19.05nmol/L     | < 0.08                    | ≤ 0.42nmol/L         |
|                                    | Methoxynorepinephrine | >20.56nmol/L    | 0.75                      | ≤ 0.1nmol/L          |
| Plasma catecholamines              | Dopamine              | 1933.9pmol/L    | 92.2                      | ≤ 195.7pmol/L        |
|                                    | Adrenaline            | 16733pmol/L     | 76.7                      | ≤ 605.4pmol/L        |
|                                    | Norepinephrine        | 66480pmol/L     | 1708.2                    | ≤ 414.0–4435.5pmol/L |
| 24-hour urinary catecholamines     | Dopamine              | 1369.4nmol/24h  | -                         | 750–2088nmol/24h     |
|                                    | Adrenaline            | 6357.2nmol/24h  | -                         | 4.3–61.6nmol/24h     |
|                                    | Norepinephrine        | 16547.3nmol/24h | -                         | 60–352nmol/24h       |

## Discussion

Pheochromocytoma in pregnancy (PIP) is a rare and high-risk endocrine disorder associated with pregnancy, with an estimated incidence of 1 in every 15,000 to 45,000 pregnancies<sup>[4]</sup>. The clinical symptoms and signs of pheochromocytoma are diverse and nonspecific, resulting in a very high risk of missed or misdiagnosis. Furthermore, due to the unique physiological state of pregnancy and the stress associated with labor and delivery, the clinical presentation of pheochromocytoma in pregnancy is easily confused with conditions such as gestational hypertension and preeclampsia. The disease progresses rapidly, and the condition is life-threatening, potentially leading to severe adverse maternal and fetal outcomes such as maternal hypertensive crisis, cardiopulmonary failure, cerebrovascular accidents, fetal distress, and intrauterine fetal death, making clinical diagnosis and treatment extremely challenging<sup>[5]</sup>.

During prenatal care, the patient did not undergo regular checkups, and neither hypertension nor a pheochromocytoma was detected. She sought medical attention prior to admission due to “intermittent dizziness and headaches accompanied by nausea and vomiting.” Given fetal distress and the patient's symptoms—including seizures and altered consciousness—severe eclampsia

was suspected, and the condition was considered urgent. an emergency cesarean section was performed without completing the relevant diagnostic tests. During the procedure, the patient's blood pressure began to fluctuate widely. Postoperatively, ultrasound, CT scans, and plasma and urinary catecholamine levels confirmed a diagnosis of "pheochromocytoma." Following aggressive multidisciplinary resuscitation, a relatively favorable maternal and neonatal outcome was ultimately achieved. Based on clinical practice and relevant literature reports, the following issues related to the diagnosis and treatment are discussed below:

The clinical manifestations of pheochromocytoma during pregnancy are often atypical, and its incidence is far lower than that of other forms of chronic hypertension during pregnancy. Furthermore, compared with non-pregnant women, pregnant women have a lower incidence of specific symptoms such as paroxysmal sweating, palpitations, and hypertensive crises. A review of the literature shows that in 90% of pregnant women, symptoms of pheochromocytoma do not appear until just before labor, making the condition easy to overlook<sup>[6]</sup>. As for the fetus, although maternal catecholamines barely cross the placental barrier<sup>[7]</sup>, high levels of catecholamines can cause severe vasoconstriction in the uteroplacental vasculature, leading to placental ischemia, placental abruption, intrauterine growth restriction, and fetal hypoxia, ultimately resulting in increased fetal mortality<sup>[8]</sup>. The patient in this case did not present with paroxysmal dizziness and headaches until the prenatal period. She had mildly elevated blood pressure (diastolic pressure > 90 mmHg), and upon admission, her urine protein test was strongly positive (+++). Additionally, the patient experienced seizures and impaired consciousness prior to surgery. These findings fully met the diagnostic criteria for "severe preeclampsia." Her blood pressure spiked sharply during surgery and responded poorly to magnesium sulfate treatment, postoperative blood pressure fluctuated widely, which was also inconsistent with severe preeclampsia; therefore, further diagnostic tests—including ultrasound, CT, and catecholamine biochemical tests—were performed to confirm the diagnosis. Thus, timely diagnosis and treatment are crucial for improving maternal and fetal survival rates.

The diagnosis of this condition is typically established by detecting elevated levels of catecholamines and their metabolites in plasma and urine<sup>[9]</sup>. For fetal safety reasons, MRI is the preferred imaging modality for tumor localization during pregnancy, as it is superior to CT scans<sup>[10, 11]</sup>. While ultrasound may be effective in early pregnancy, its sensitivity is questionable in the second and third trimesters because the pregnant uterus can obscure tumor visualization. Due to its atypical and insidious nature, this case was missed preoperatively. A case report published by SAMIR HADDAD<sup>[12]</sup> and colleagues recommends that patients with intractable or fluctuating hypertension during pregnancy, as well as those presenting with the classic triad of headache, sweating, and palpitations, should undergo evaluation for pheochromocytoma. I recommend that for patients referred from other hospitals—particularly those with pregnancy-induced hypertension who have had irregular prenatal checkups during pregnancy—a comprehensive abdominal ultrasound should still be performed by an experienced sonographer during the prenatal or preoperative period. Although ultrasound has certain limitations in screening for pheochromocytoma during late pregnancy, it still offers advantages due to its immediacy, non-invasiveness, and speed; if refractory hypertension or significant blood pressure fluctuations occur postpartum or postoperatively, PIP should be the primary consideration; relevant imaging studies and catecholamine biochemical tests should be performed as soon as possible to confirm the diagnosis<sup>[13]</sup>.

Pregnancy-associated pheochromocytomas often result in central ischemic necrosis and cystic changes due to the tumor's rich blood supply, fragile vascular structure, and rapid growth; combined with multiple factors such as increased blood volume during pregnancy, endocrine hormonal imbalances, and sympathetic nervous system excitation—compounded by mechanical compression of the uterus in late pregnancy, uterine contractions, and elevated intra-abdominal pressure—these factors collectively lead to intratumoral vascular rupture and bleeding. Furthermore, sudden changes in intra-abdominal pressure during cesarean section, mechanical traction from surgical maneuvers, and severe hemodynamic fluctuations caused by anesthesia and surgical stress are major external factors that trigger rupture and hemorrhage in gestational pheochromocytomas. Additionally, since the tumor has already undergone pathological changes such as cystic degeneration and vascular atrophy during pregnancy, its structural stability is significantly reduced, making it highly susceptible to capsular rupture and intratumoral vascular rupture under the combined stress of surgical procedures. When a pheochromocytoma undergoes cystic degeneration accompanied by intratumoral hemorrhage, the large amounts of catecholamines stored within the tumor are rapidly released into the bloodstream, strongly stimulating  $\alpha$  and  $\beta$  receptors. This triggers severe systemic vasoconstriction and tachycardia, resulting in a sharp rise in blood pressure; Meanwhile, a temporary depletion of hormones due to the necrosis of tumor endocrine cells caused by hemorrhage, combined with factors such as reflex vasodilation triggered by the body's baroreceptors and insufficient

venous return to the heart, leads to a rapid drop in blood pressure. The ongoing progression of hemorrhage and cystic changes repeatedly stimulates the tumor tissue, causing catecholamine secretion to alternate between paroxysmal surges and depletion, ultimately resulting in characteristic hemodynamic changes marked by erratic, sudden rises and falls in blood pressure<sup>[14]</sup>.

This case fully corresponds to the pathophysiological changes described above. However, in actual clinical practice, it was observed that during the shock episode, adequate volume replacement and administration of vasoactive agents such as norepinephrine and metaraminol did not raise the patient's blood pressure. During the period of persistent hypotensive shock, ultrasound revealed adrenal hemorrhage. Given the suspicion of an adrenal crisis, a short-term, low-dose course of hydrocortisone was administered, after which the blood pressure rose and stabilized within the normal range. Following completion of relevant tests, the diagnosis was confirmed as cystic chromocytoma with hemorrhage. Hydrocortisone was discontinued, after which the patient's blood pressure again exhibited significant fluctuations. In clinical practice, neither fluid resuscitation nor the use of vasoactive agents stabilized the patient's blood pressure. The underlying pathophysiological mechanism may be that prolonged, high-dose catecholamine stimulation has led to a reduction in the number of adrenergic receptors and decreased catecholamine sensitivity, resulting in poor efficacy of conventional vasoactive agents such as norepinephrine and metaraminol. In this case, our team found that brief hydrocortisone therapy was effective during the phase of persistent, refractory shock—when the diagnosis was unclear and vasoactive agents were ineffective. The mechanism may be that exogenous hydrocortisone enhanced vascular sensitivity to catecholamines. After the diagnosis of “pheochromocytoma” was confirmed, the medication was discontinued due to concerns that it might trigger a catecholamine storm or exacerbate blood pressure fluctuations, which could further worsen the patient's condition. However, I remain open to the use of hydrocortisone during the refractory shock phase, although its dosage, duration of use, and associated risks require further investigation. Furthermore, I believe that minimizing postoperative stress as much as possible was also a key factor in the successful management of this case.

For pheochromocytoma, surgical resection of the tumor is the only curative treatment<sup>[15]</sup>. The optimal time for surgery in pregnant patients with pheochromocytoma is during the second trimester; surgery during the first trimester may lead to miscarriage, so medical management should be used to bridge the gap until the second trimester, at which point surgery can be performed. In the third trimester, medical management can be continued until the fetus is full-term, and surgery should be performed before 24 weeks of gestation. Adequate preoperative preparation with alpha-receptor blockers is required; blood pressure should ideally be maintained below 140/90 mmHg to ensure preoperative hemodynamic stability. The recommended duration of medication is 10–14 days. Beta-receptor blockers may be administered to control arrhythmias. Volumetric expansion during the preoperative preparation period can reduce the incidence of postoperative hypotension<sup>[16]</sup>. The preferred surgical approach is laparoscopic tumor resection. For patients diagnosed in late pregnancy, tumor resection should be performed during a cesarean section<sup>[17]</sup>, and vaginal delivery should be avoided whenever possible.

## Conclusion

For patients with pheochromocytoma that went undetected during pregnancy and present with atypical symptoms, pheochromocytoma should be included in the differential diagnosis if refractory hypertension or irregular, wide-ranging blood pressure fluctuations occur during late pregnancy or before delivery.

In cases of cystic pheochromocytoma complicated by hemorrhage, shock should still be corrected by replenishing effective circulating blood volume and administering adequate vasoactive agents. Whether medications such as hydrocortisone are necessary for refractory shock requires further investigation.

In addition, minimizing postoperative stress is one of the key aspects of treatment.

## Abbreviations

ECG  
electrocardiogram  
LVEF

Left Ventricular Ejection Fraction  
Hb  
Hemoglobin  
IV  
Intravenous injection  
CT  
Computed Tomography  
PICCO  
Pulse index Continuous Cardiac Output  
PIP  
Pheochromocytoma in pregnancy

## Declarations

### **Ethics approval and consent to participate**

This case report has been approved and consent to participate by the Ethics Committee of Datong Third People's Hospital.

### **Consent for publication**

This case report has obtained written informed consent from the patient and their family for publication.

## Competing interests

None declared.

## Funding

None.

## Author Contribution

Zhigang Zhao contributed towards collected clinical data, original draft and editing. Kaiyu Li designed the case report, reviewed the manuscript and approved the final version. Ziru Guo was responsible for searched literature, revised the text and organized figures. Xiulin Yan, Zhizhou Song, Jianling Zhu and Ye Chen completed clinical management and data verification. All authors have read and approved the final manuscript.

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None.

## Data Availability

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

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## Figures

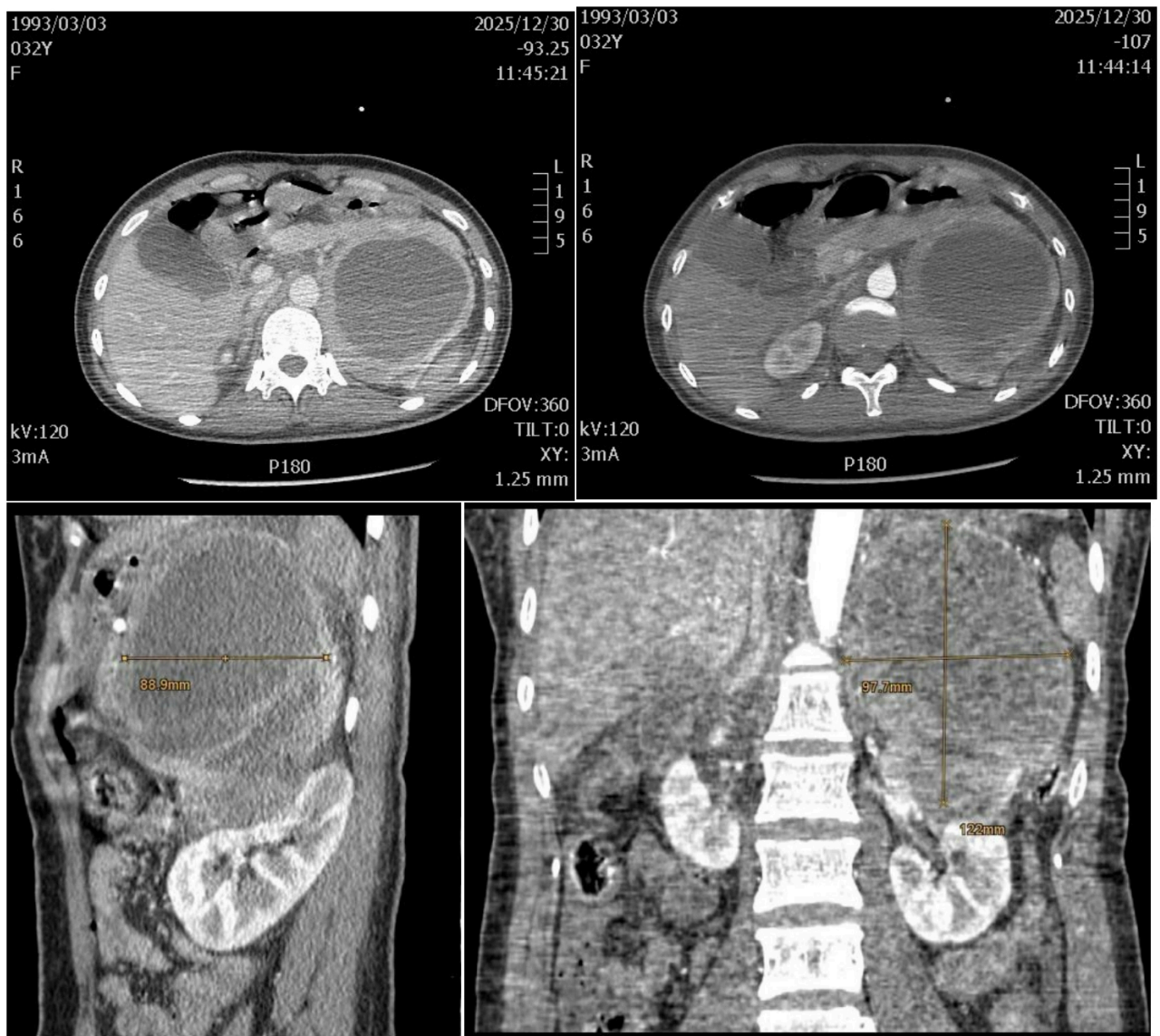


Figure 1

CT scan shows a large mass with cystic degeneration and hemorrhage in the left adrenal region

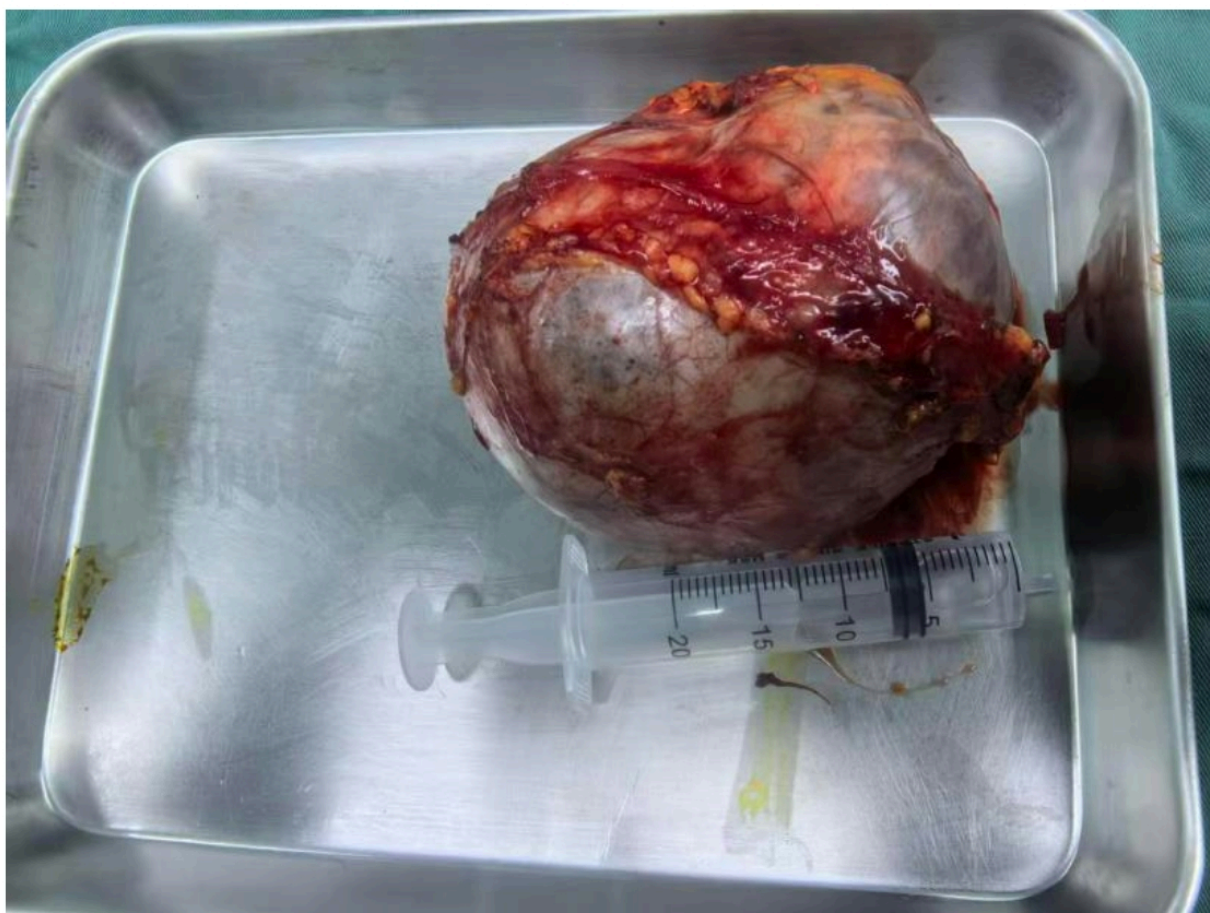


**Figure 2**

**shows the cross-section of the adrenalectomy specimen**

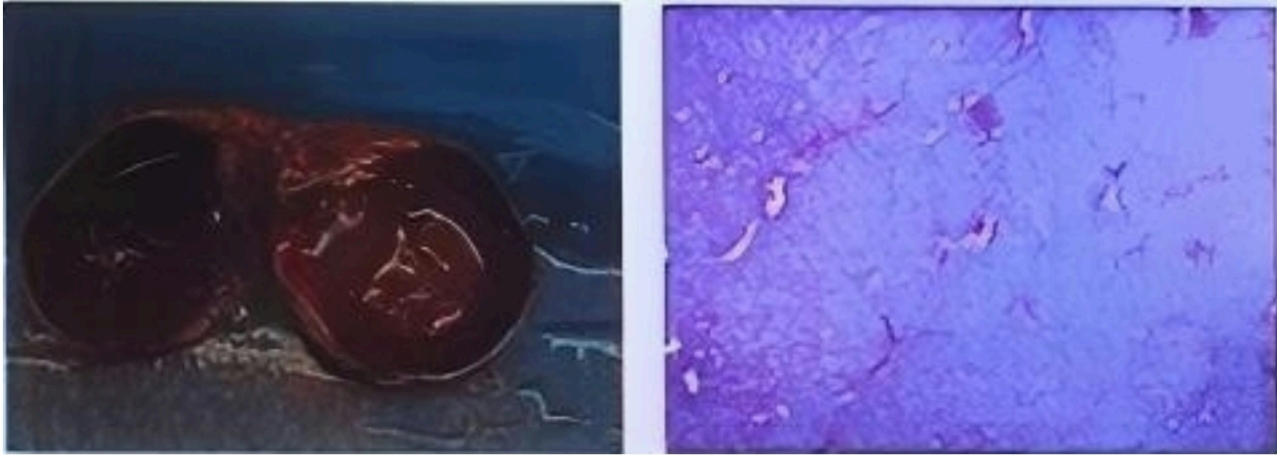


**Figure 3**

**histopathological examination:**(Left faint gland and mass) combined with morphology and immunoimmunization results, in line with adrenal pheochromocell tumor, with active intensity growth and the growth trend in the focal chest map. It is recommended to punish the review.

**IHC:1 (1-6 solid zone):** CgA (+). Syp(+),Inhibin-a(-). Ckpan(-). Ki-67(2M). D-catenin (post -), OPI1B1 (-), OYP11B2 (-), SF-1 (-), Molan-A (-), GATA 3 (post point+).