70TH ESCVS CONGRESS & 7TH IMAD MEETING

20 I 23 JUNE 2022

Liege I Théâtre de Liège I Belgium

www.escvs2022.com

70th ESCVS
International congress of the European Society of Cardiovascular and Endovascular Surgery

7th IMAD meeting

TAXINOMISIS

MRI plaque analysis

Marija Jovanovic, University of Belgrade, Serbia
Georgios Karagiannis, University of Athens, Greece

The authors have no conflicts of interest to declare

MRI plaque analysis

- the new risk model to discriminate high versus low risk cases for cerebrovascular complications from carotid artery disease
- asymptomatic and symptomatic moderate to severe extracranial carotid artery stenosis
- MRI of carotid tree from aortic arch up to the circle of Willis (ACC, ACI, ACE)
- baseline and FU after 12 and 36 months

Goal of MRI

- stenosis degree
- intimal thickening, lipid or necrotic core, hemorrhage, fibrous cap, calcification, fibrosis
- FU - carotid plaque progression
- plaques morphology classified according to the American Heart Association criteria

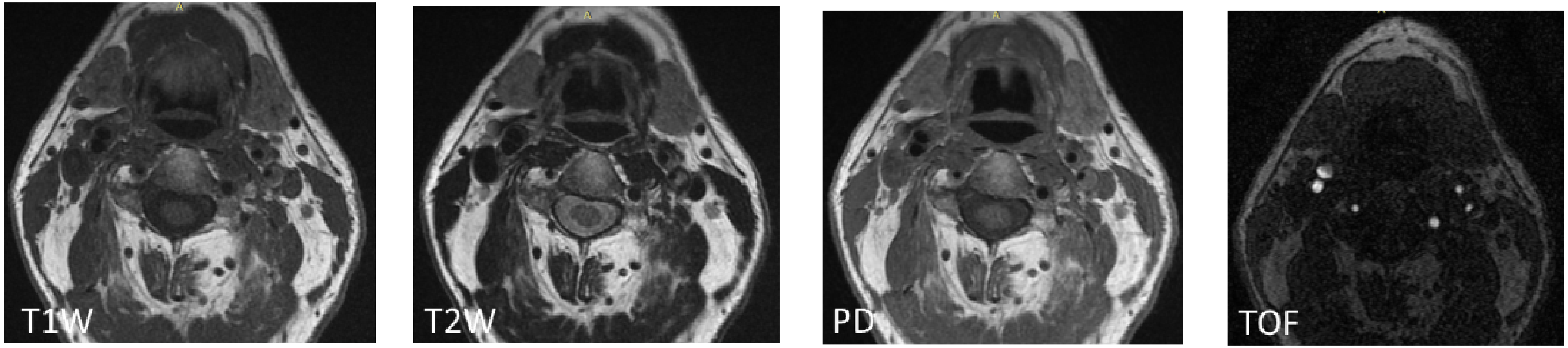
TABLE 1. Conventional and Modified AHA Classification of Atherosclerotic Plaque

Conventional AHA Classification	Modified AHA Classification for MRI
Type I: initial lesion with foam cells Type II: fatty streak with multiple foam cell layers	Type I–II: near-normal wall thickness, no calcification
Type III: preatheroma with extracellular lipid pools	Type III: diffuse intimal thickening or small eccentric plaque with no calcification
Type IV: atheroma with a confluent extracellular lipid core Type V: fibroatheroma	Type IV–V: plaque with a lipid or necrotic core surrounded by fibrous tissue with possible calcification
Type VI: complex plaque with possible surface defect, hemorrhage, or thrombus	Type VI: complex plaque with possible surface defect, hemorrhage, or thrombus
Type VII: calcified plaque	Type VII: calcified plaque
Type VIII: fibrotic plaque without lipid core	Type VIII: fibrotic plaque without lipid core and with possible small calcifications

Cai et al. Classification of Human Carotid Atherosclerotic Lesions With In Vivo Multicontrast Magnetic Resonance Imaging. Circulation 2002; 106:1368-73

MRI protocol

- T1W, T2W, PD, TOF, Phase-contrast images
- T1W BB with FS before and after applying the contrast media



MRI criteria for annotation of plaque components

Classification of Human Carotid Atherosclerotic Lesions With In Vivo Multicontrast Magnetic Resonance Imaging

Jian-Ming Cai, MD, PhD; Thomas S. Hatsukami, MD; Marina S. Ferguson, BS; Randy Small, BS; Nayak L. Polissar, PhD; Chun Yuan, PhD

Background—Rec
atherosclerotic
this study was t
atherosclerotic
Methods and Resu
were imaged wi
different contras
was 0.25×0.25
images and hist
obtained by MR
of 0.74 (0.67 to
as follows: type
lesions, 82% an
Conclusions—In v
lesions in the hu
atherosclerotic

Quantitative Evaluation of Carotid Plaque Composition by In Vivo MRI

T. Saam, M.S. Ferguson, V.L. Yarnykh, N. Takaya, D. Xu, N.L. Polissar, T.S. Hatsukami, C. Yuan

Magnetic Resonance in Medicine 55:659–668 (2006

Objective—Th
Methods and
time-of-fligh
correspondi
lipid-rich/ne
the total wa
reproducibil
LR/NC (23.
64%; P=0.
P<0.001).
ranging fro
Conclusions—
clinical tria
regression.

Automated In Vivo Segmentation of Carotid Plaque MRI with Morphology-Enhanced Probability Maps

Fei Liu,[1]* Dongxiang Xu,[1] Marina S. Ferguson,[1] Baocheng Chu,[1] Tobias Saam,[1] Norihide Takaya,[1] Thomas S. Hatsukami,[2] Chun Yuan,[1] and William S. Kerwin[1]

MRI is a promising noninvasive technique for characterizing atherosclerotic plaque composition in vivo, with an end-goal of assessing plaque vulnerability. Because of limitations arising from acquisition time, achievable resolution, contrast-to-noise ratio, patient motion, and the effects of blood flow, automatically identifying plaque composition remains a challenging task in vivo. In this article, a segmentation method using maximum a posteriori probability Bayesian theory is presented that divides axial, multi-contrast-weighted images into regions of necrotic core, calcification, loose matrix, and fibrous tissue. Key advantages of the method are that it utilizes morphologic information, such as local wall thickness, and coupled active contours to limit the impact from noise and artifacts associated with in vivo imaging. In experiments involving 142 sets of multi-contrast images from 26 subjects undergoing carotid endarterectomy, segmented areas of each of these tissues per slice agreed with

review, automated segmentation would reduce the considerable amount of training required to read these images and the corresponding inter-rater variability. Additionally, a viable, automated segmentation procedure would permit various combinations of contrast weightings and image characteristics to be objectively analyzed for accuracy in plaque characterization. Such studies have been conducted using automated segmentation of ex vivo endarterectomy specimens (6,7), but these results are difficult to translate to in vivo imaging, given the different constraints regarding acquisition time, resolution, contrast-to-noise ratio, and effects of blood flow. Recent efforts to demonstrate in vivo segmentation of some plaque components (8,9) are promising, but they have not yet been histologically validated.

Published March 22, 2012 as 10.3174/ajnr.A3028

ORIGINAL RESEARCH

Automated Versus Manual In Vivo Segmentation of Carotid Plaque MRI

R. van 't Klooster
O. Naggara
R. Marsico
J.H.C. Reiber
J.-F. Meder
R.J. van der Geest
E. Touzé
C. Oppenheim

The official journal of the Japan Atherosclerosis Society and the Asian Pacific Society of Atherosclerosis and Vascular Diseases

Original Article

J Atheroscler Thromb, 2018; 25: 1222-1230. http://doi.org/10.5551/jat.43679

Association of Age and Size of Carotid Artery Intraplaque Hemorrhage and Minor Fibrous Cap Disruption: A High Resolution Magnetic Resonance Imaging Study

Yuanyuan Cui[1], Huiyu Qiao[2], Lu Ma[1], Mingming Lu[1], Jiafei Yang[3], Guoen Yao[4], Jianming Cai[1,3] and Xihai Zhao[2]

Jianming Cai and Xihai Zhao are joint senior authors.

[1]Department of Radiology, PLA General Hospital, Beijing, China
[2]Center for Biomedical Imaging Research, Department of Biomedical Engineering, Tsinghua University School of Medicine, Beijing, China
[3]Department of Radiology, First Affiliated Hospital of PLA General Hospital, Beijing, China
[4]Department of Neurology, First Affiliated Hospital of PLA General Hospital, Beijing, China

Aim: To investigate the association between the volumes of different aging intraplaque hemorrhage (IPH) and minor fibrous cap disruption (MFCD) in carotid arteries.

Methods: Patients with cerebrovascular symptoms and carotid atherosclerotic plaques determined by ultrasound were recruited and underwent multi-contrast magnetic resonance (MR) vessel wall imaging for carotid arteries. Carotid plaques with IPH on MR imaging were included in the analysis. The age (fresh or recent) and the volume of IPH for each plaque were evaluated.

Results: In total, 41 carotid plaques in 37 patients (mean age 70.2±11.0 years old; 32 males) were eligible for statistical analysis. The absolute volume of fresh IPH in plaques with MFCD was significantly larger than that in plaques without MFCD (109.83±75.49 mm³ vs. 30.54±20.62 mm³, P= 0.002). Logistic regression showed that the absolute volume of fresh IPH was significantly associated with MFCD before (odds ratio [OR], 1.735; 95% confidence interval [CI], 1.127–2.670; P=0.012) and after adjusting for confounding factors (OR, 1.823; 95% CI, 1.076–3.090; P=0.026). There was no significant association between recent IPH volume and MFCD (P>0.05).

Conclusion: The volume of fresh IPH is independently associated with MFCD in carotid plaques, suggesting that integrity of fibrous cap may change with different age and size of IPH.

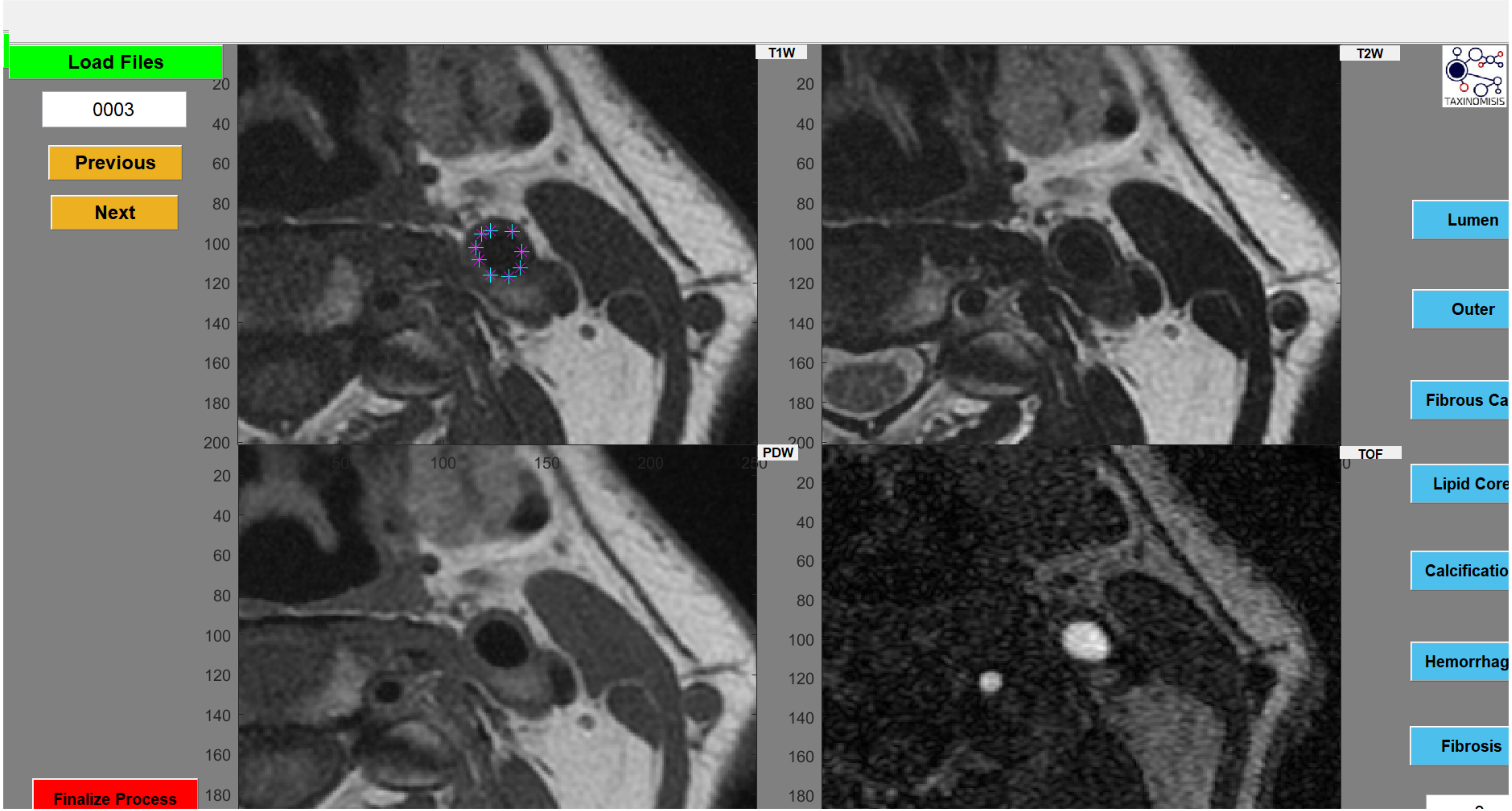
MRI criteria for manual annotation of plaque components

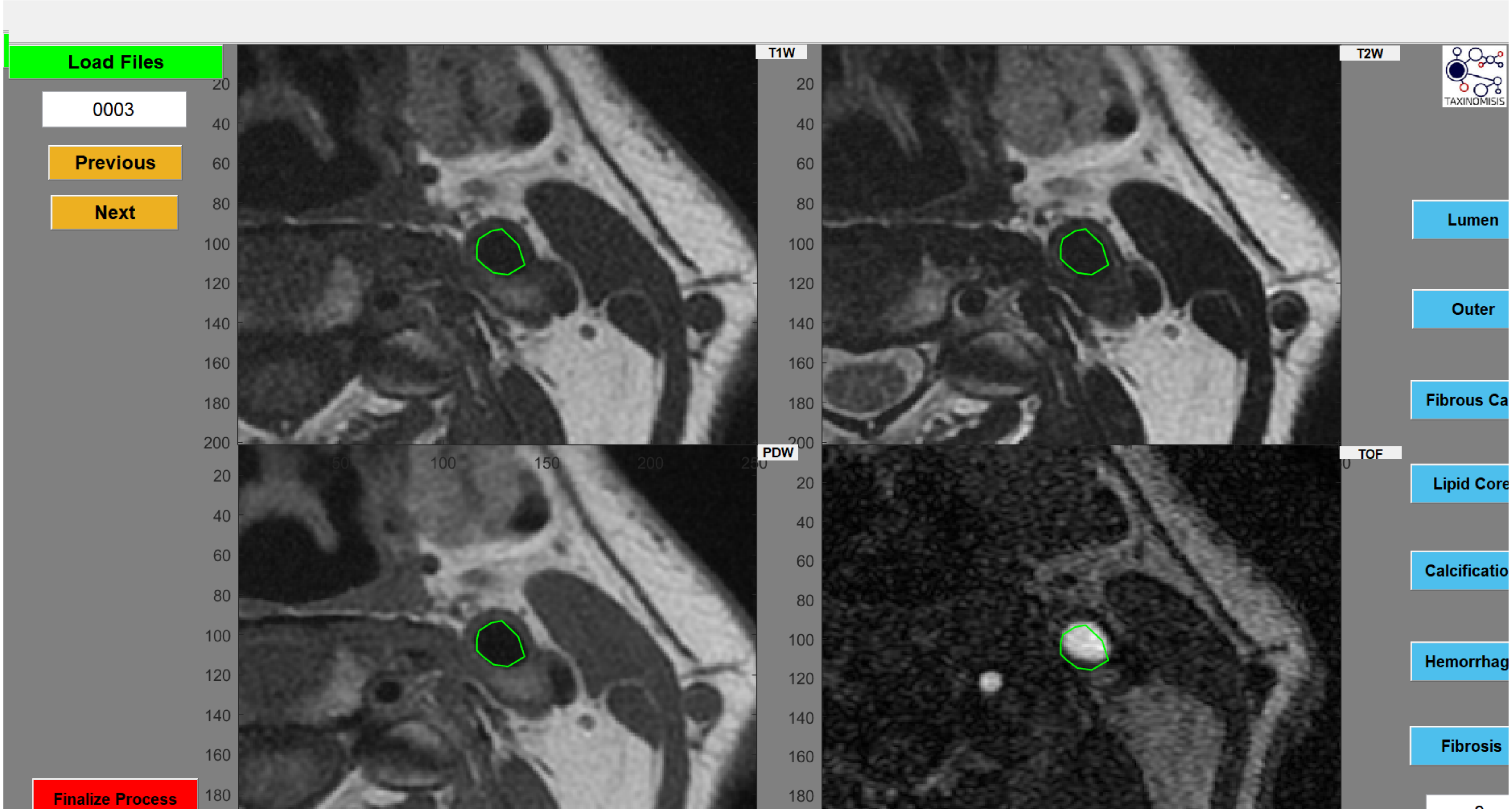
	TOF	T1W	T2W	PDW	Morphological cues	Most added-valued image	Image where annotation should be performed
Lipid core (LC)	0	0 / +	- / 0	0 / +	Diffuse or eccentric thickening of blood vessel wall, the crescent-shaped	T1W, T2W, TOF	T2W
Fibrous cap (FC)	-	0	0	0	Juxtaluminal lesion	T1W (adjacent IPH or LC will accentuate FC)	TOF
Recent hemorrhage (late subacute IPH)	+	+	+	+	Irregular, like LC, etc.	T1W, T2W, TOF	T1W
Calcification	-	-	-	-	Irregular, dot-like or tubular formations	All sequences	T1W
Fibrosis	0/+	0/+	0/+	-		All sequences	T1W

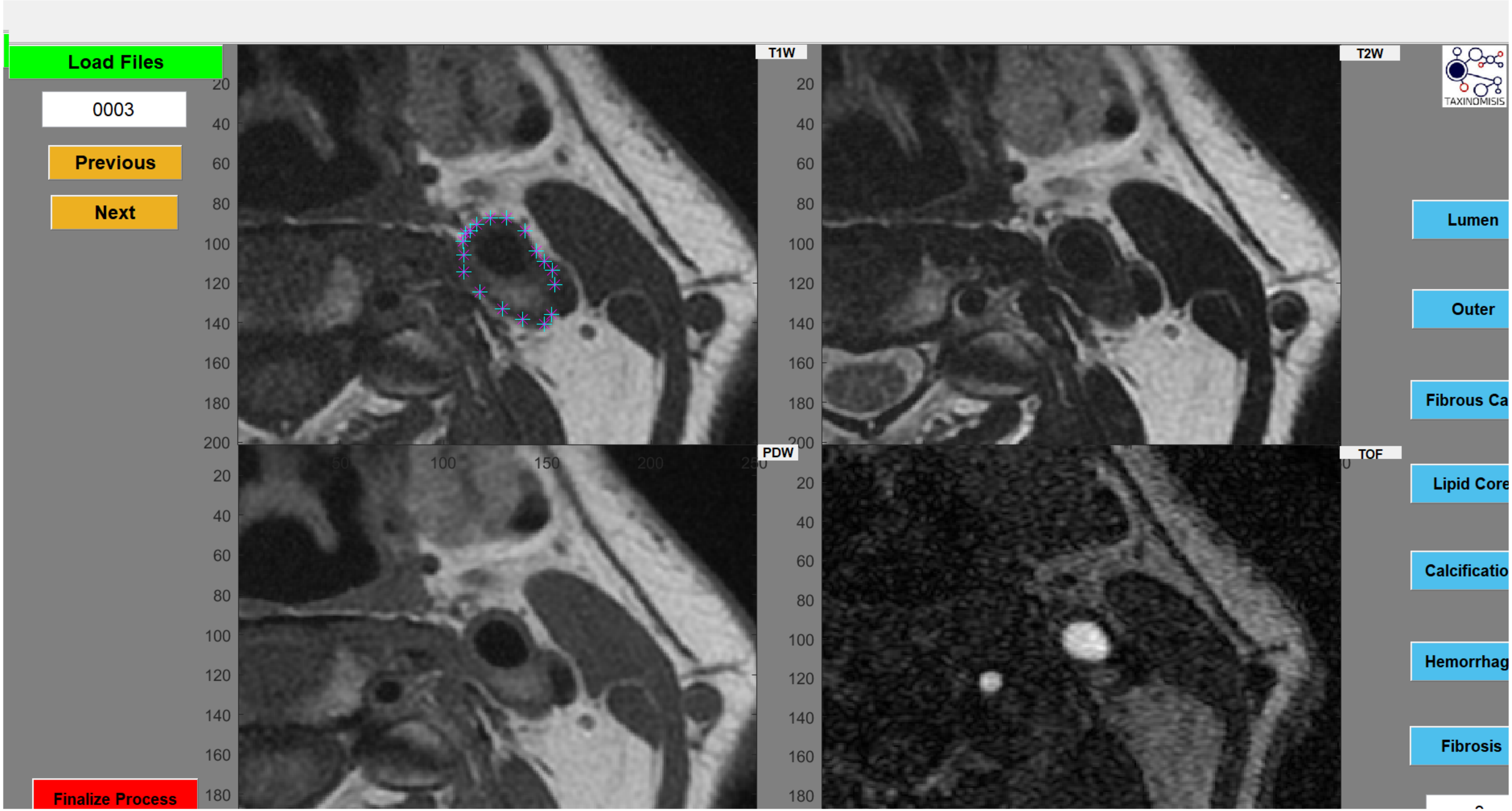
* 0 isointense, + hyperintense, - hypointense

** Signal intensities relative to the adjacent sternocleidomastoid muscle

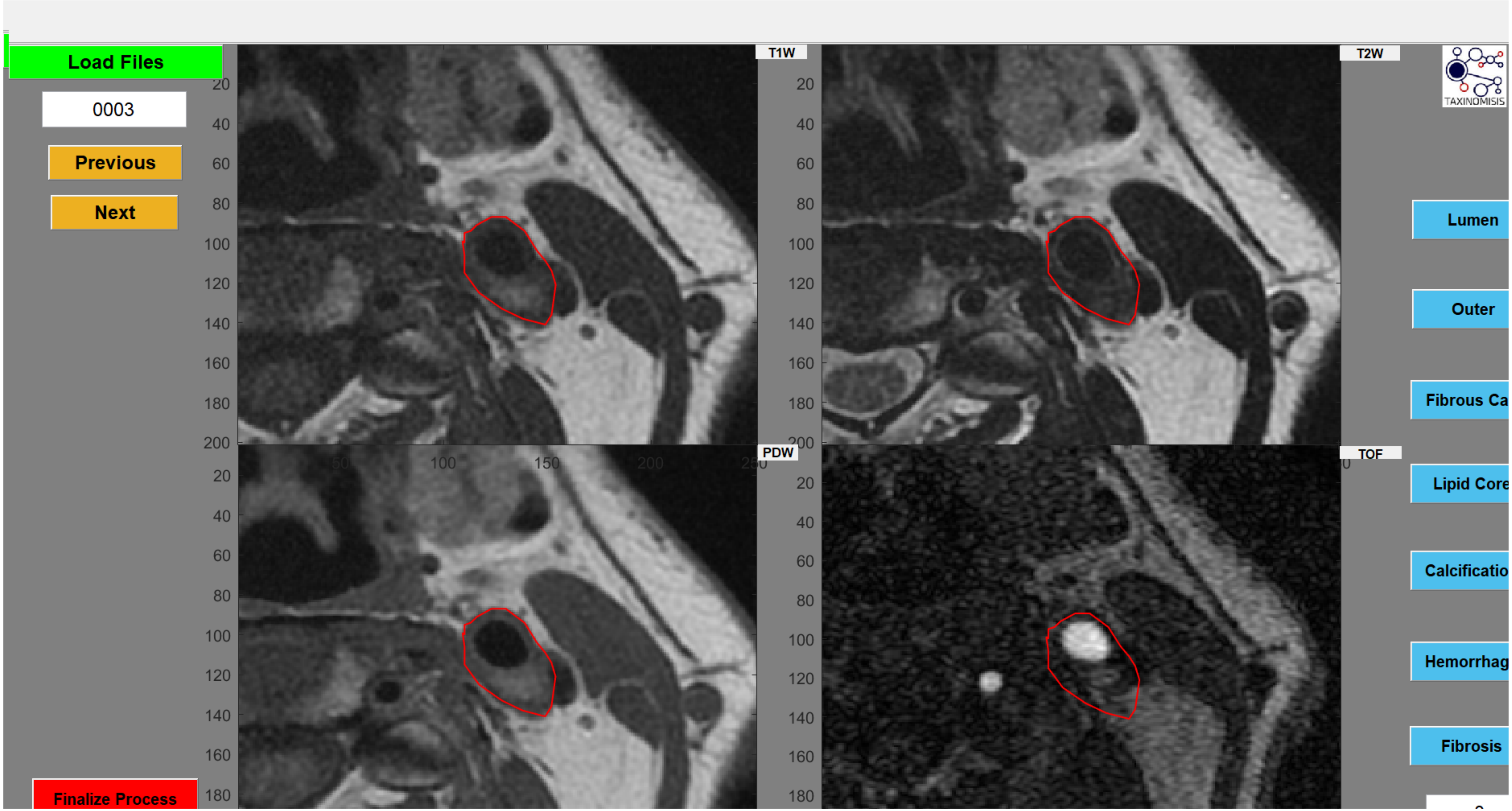
Case 1


Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis

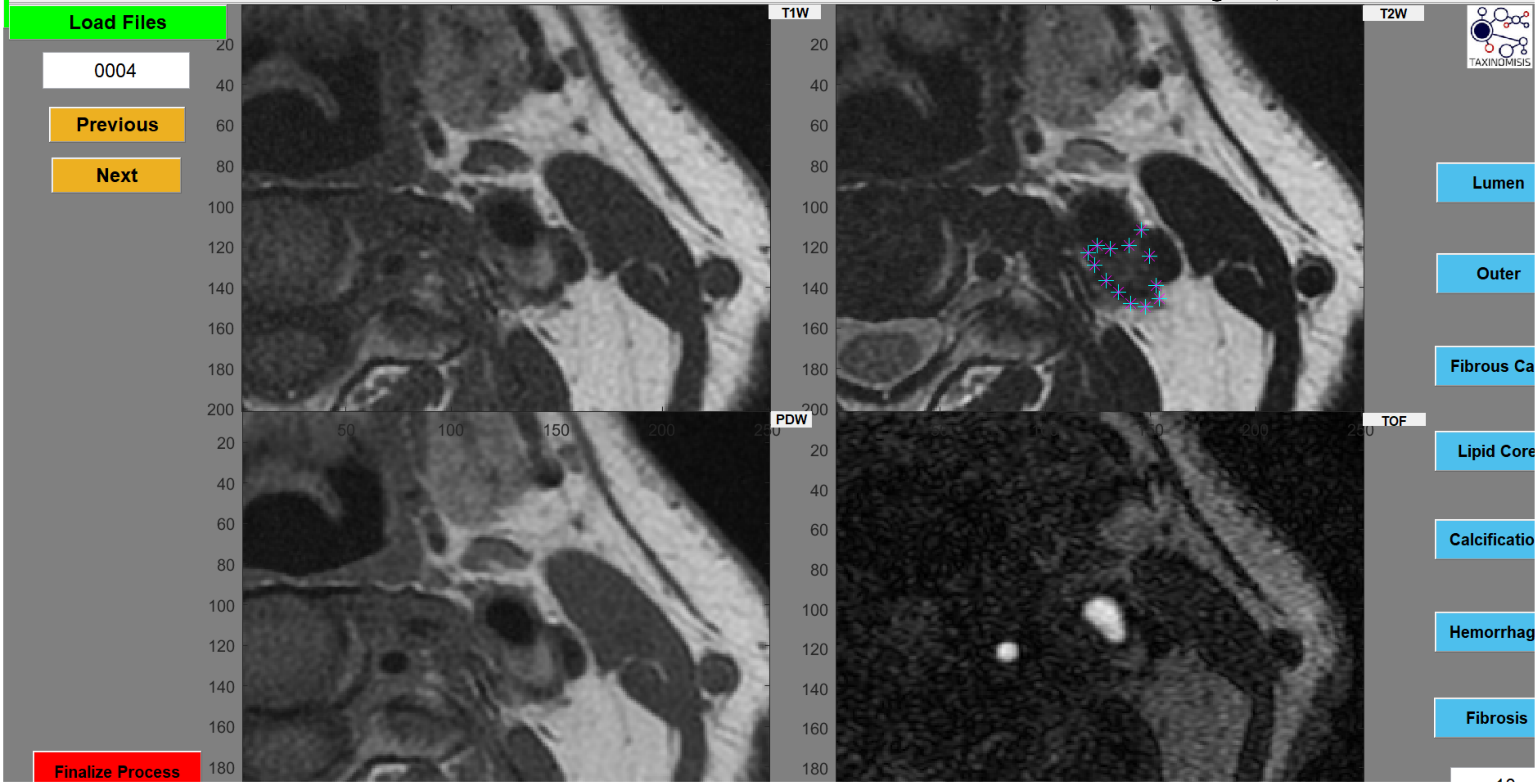

Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis

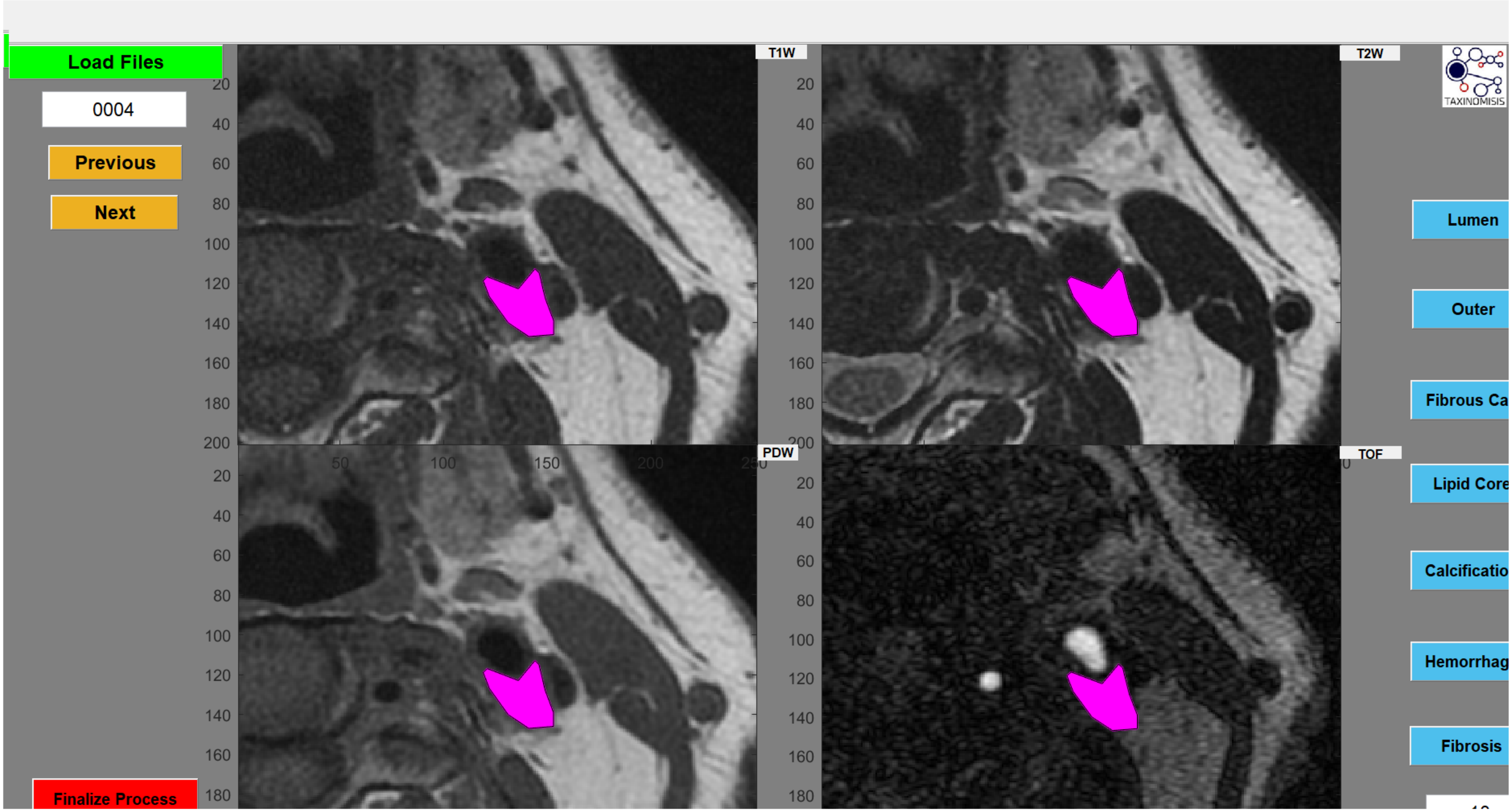


Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis

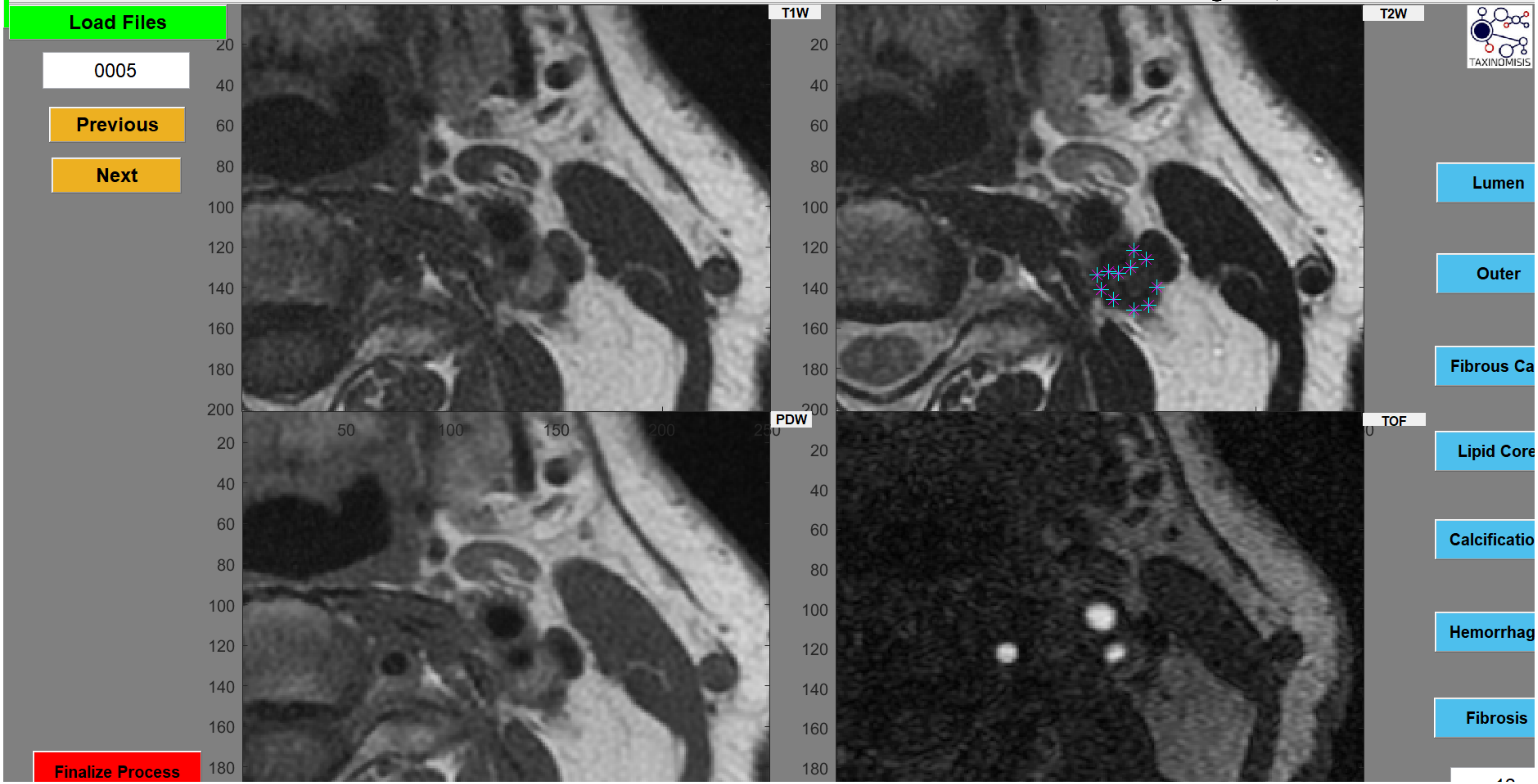

Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis

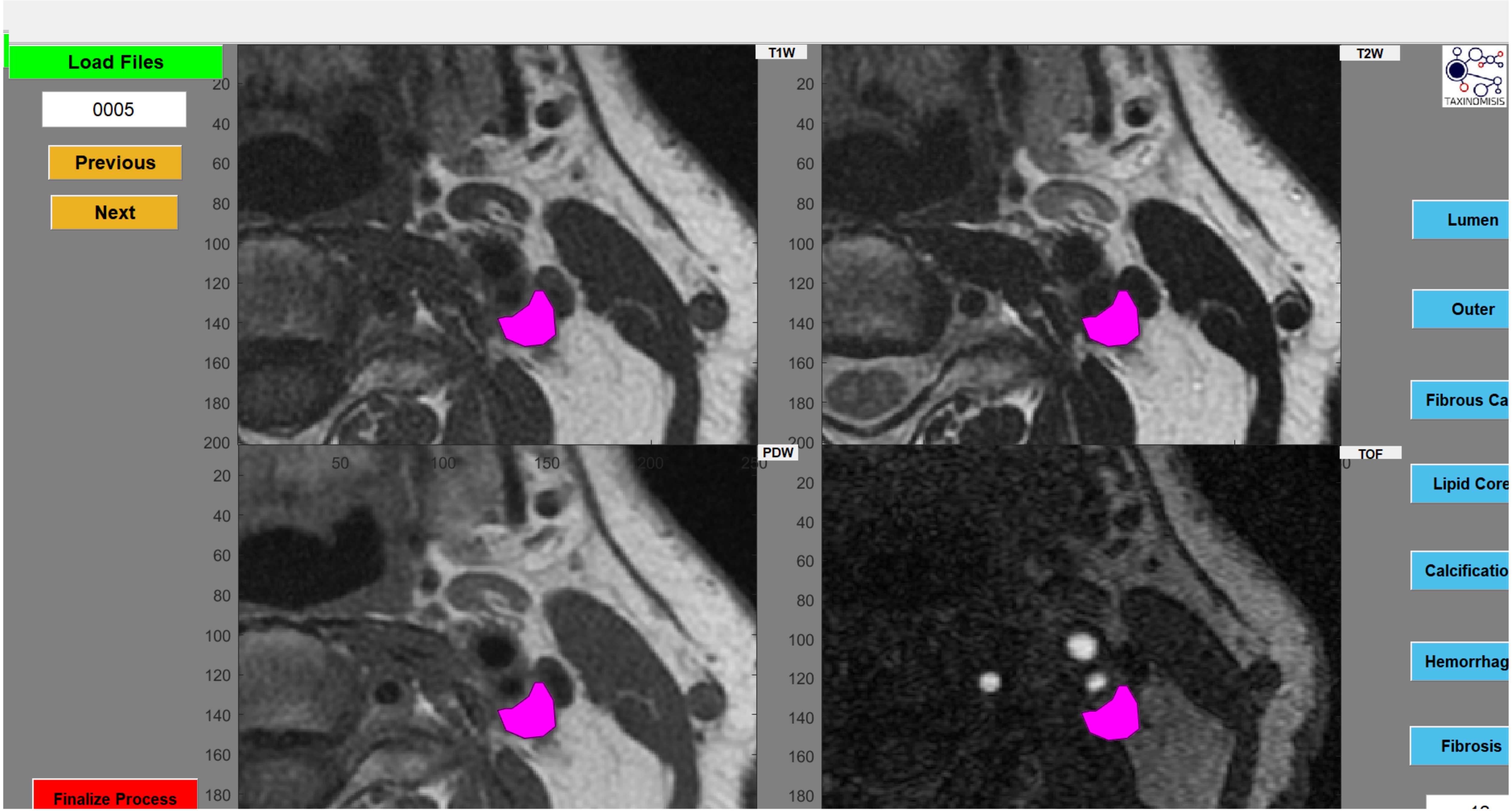
High T1, low T2 - LC



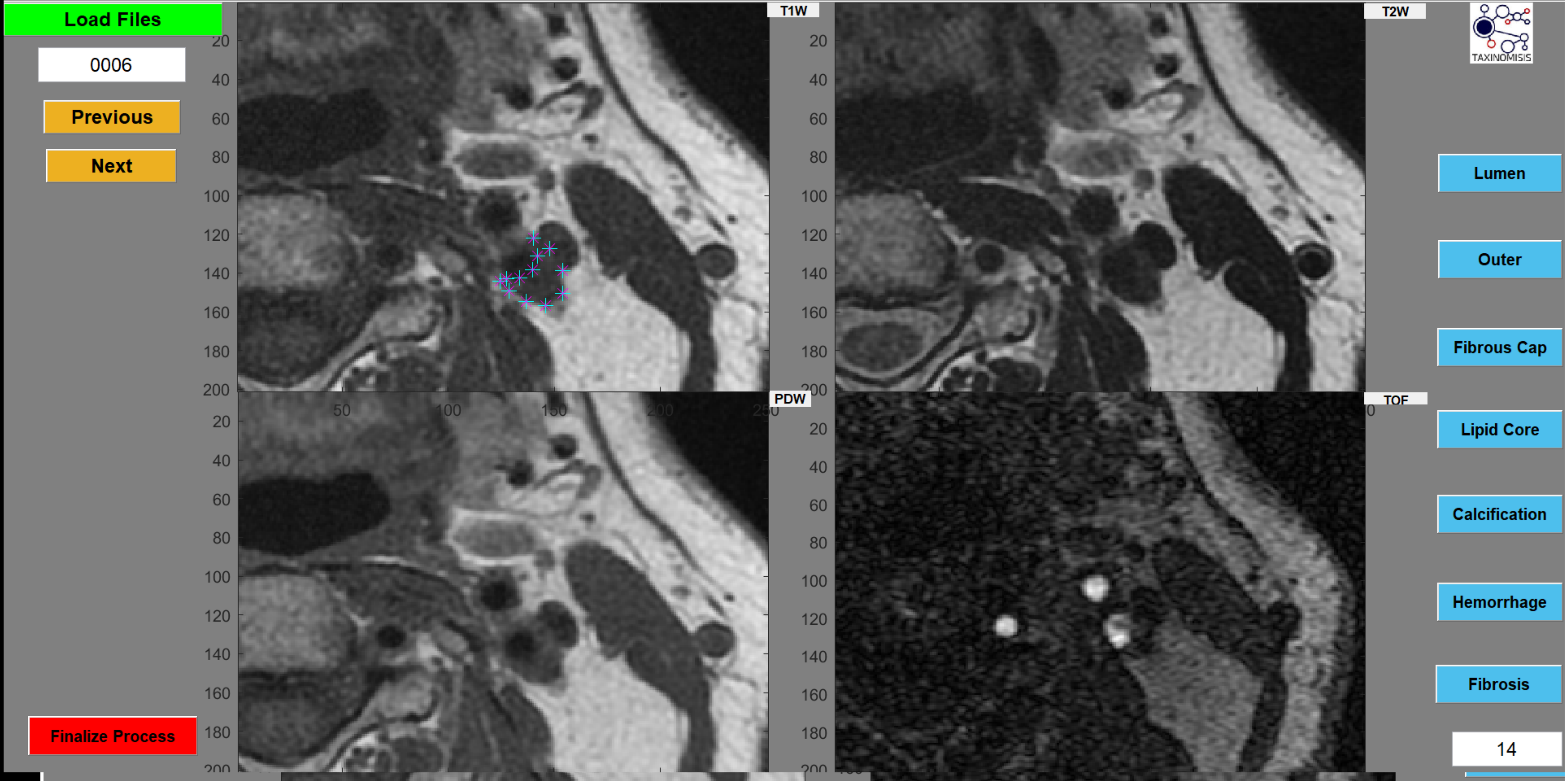

Load Files
0004
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis

High T1, low T2 - LC

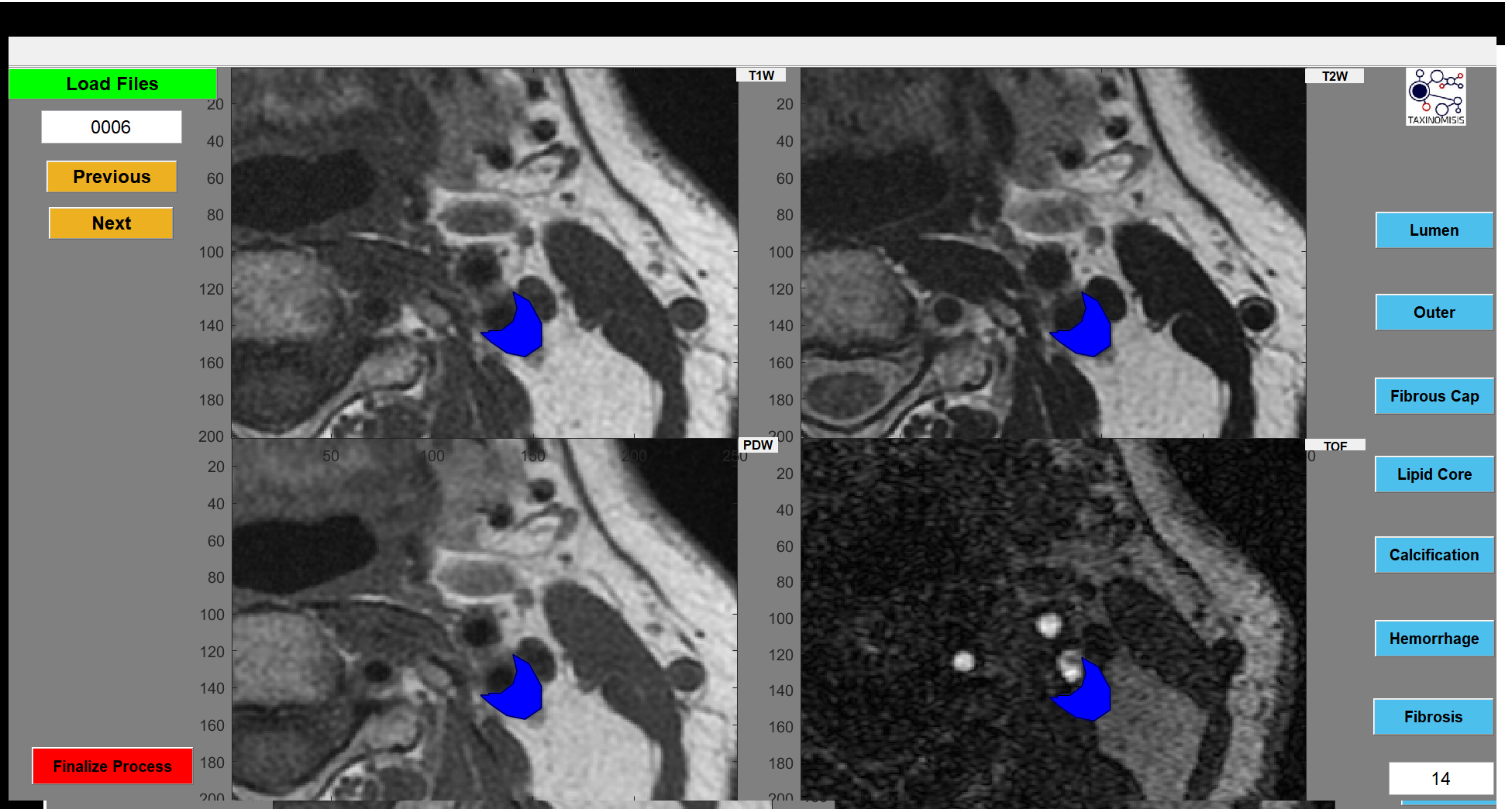



Load Files
0005
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Ca
Lipid Core
Calcificatio
Hemorrhag
Fibrosis
20
40
60
80
100
120
140
160
180
200
50
100
150
200

Low T1, T2 - Fib

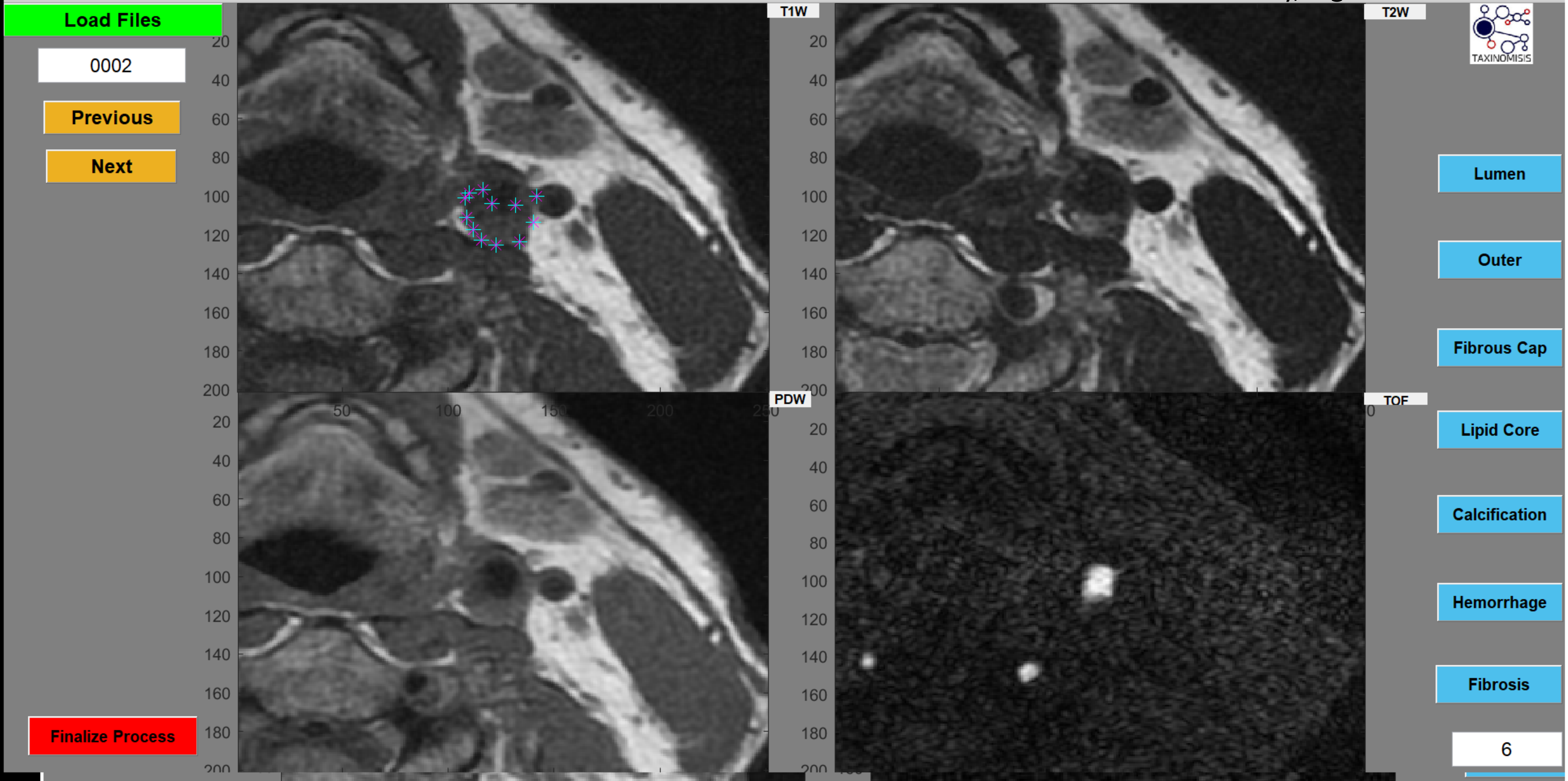


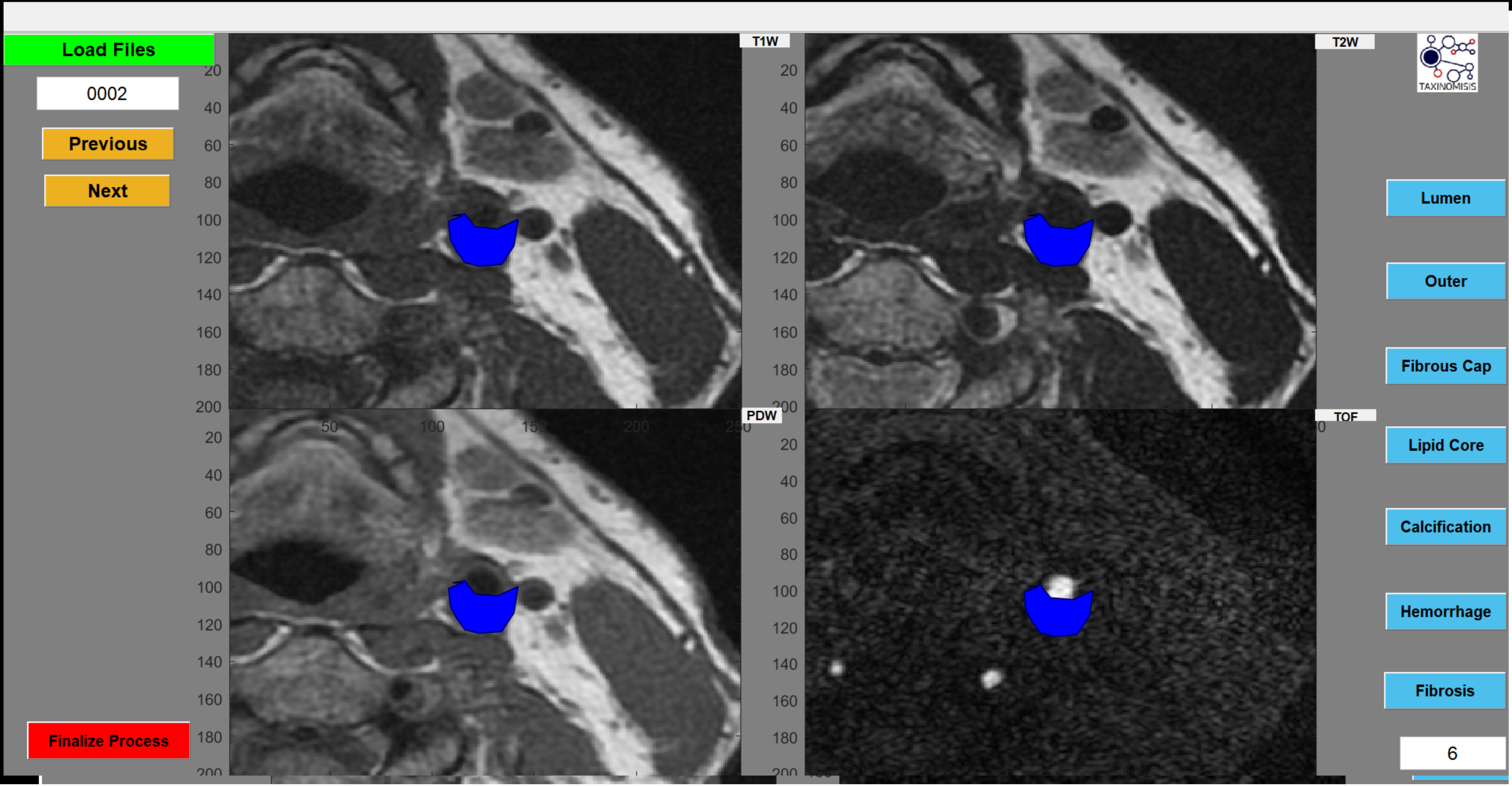
Load Files
0006
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
14


Load Files
0006
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
14

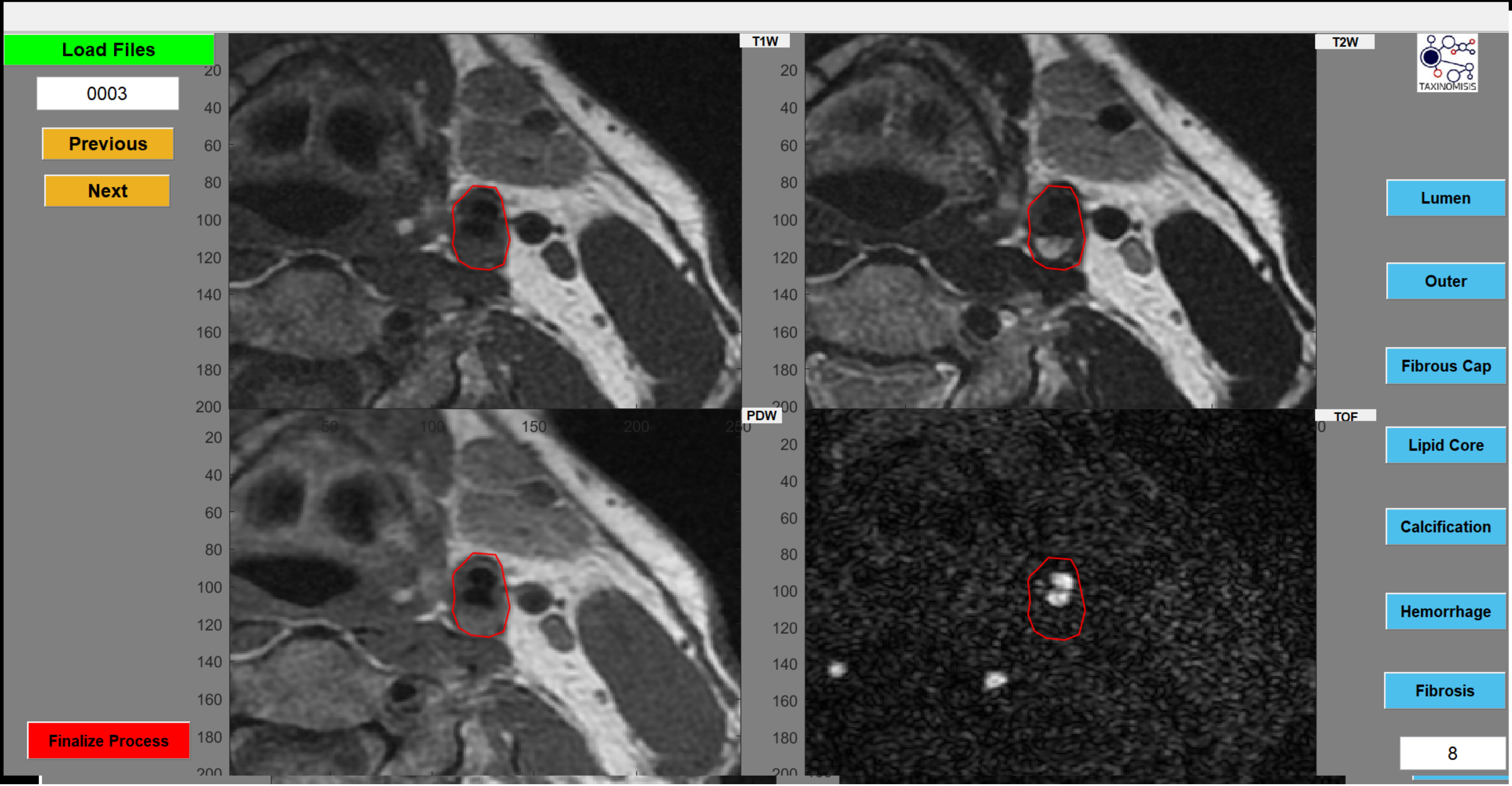
Case 2

Iso T1Wy, high T2W - Fib



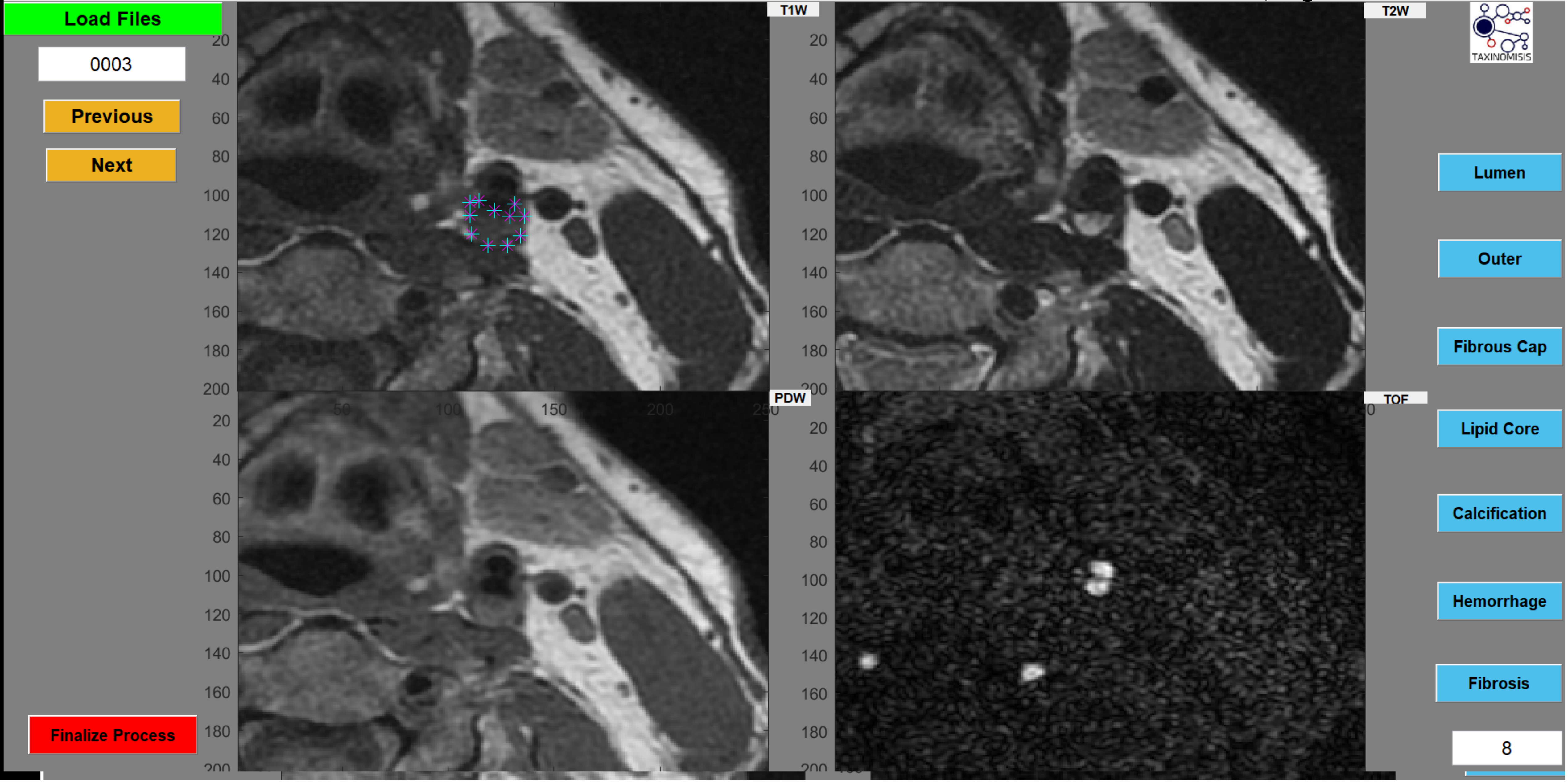


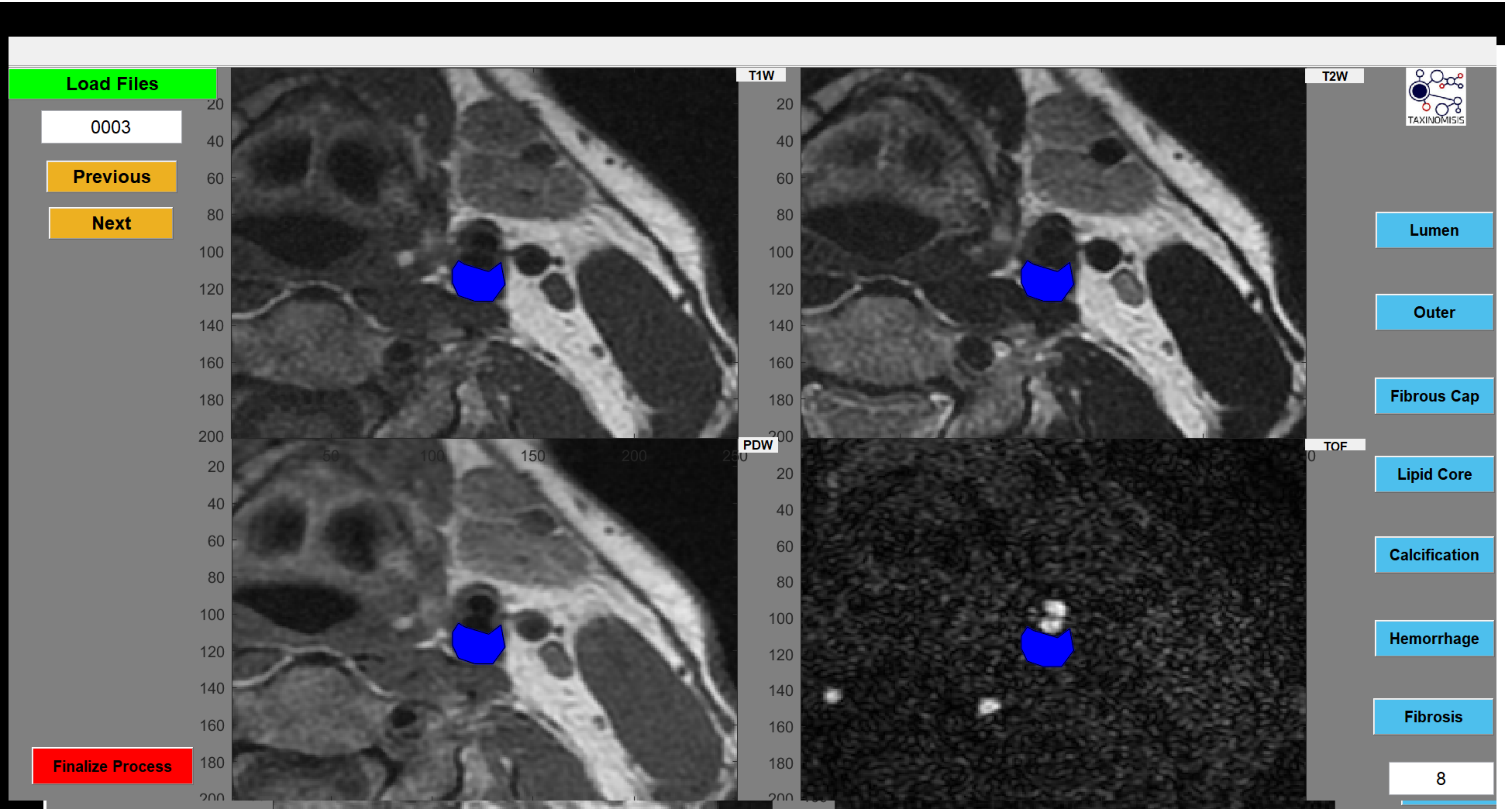
Load Files
0002
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
6



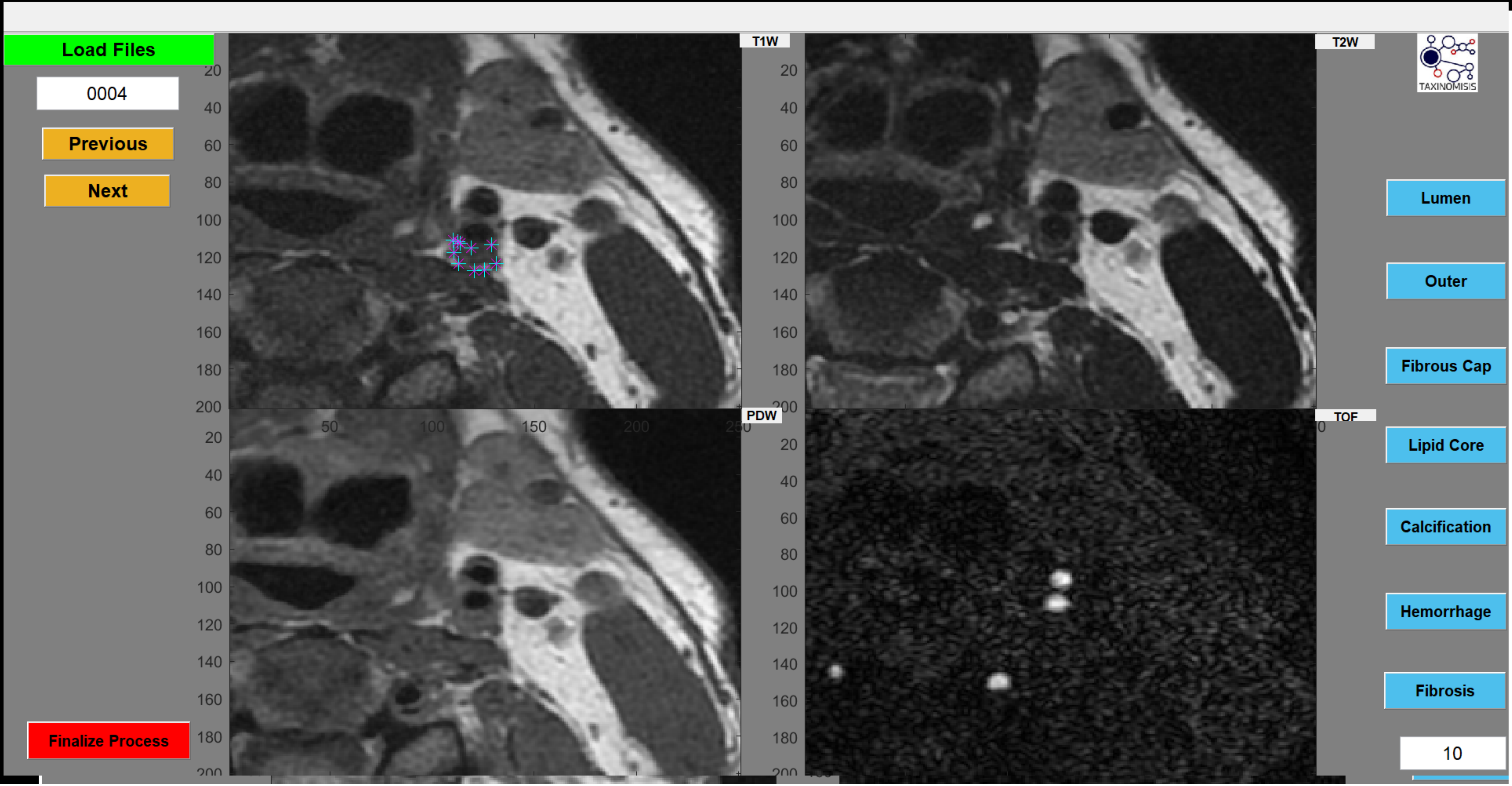
Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
8

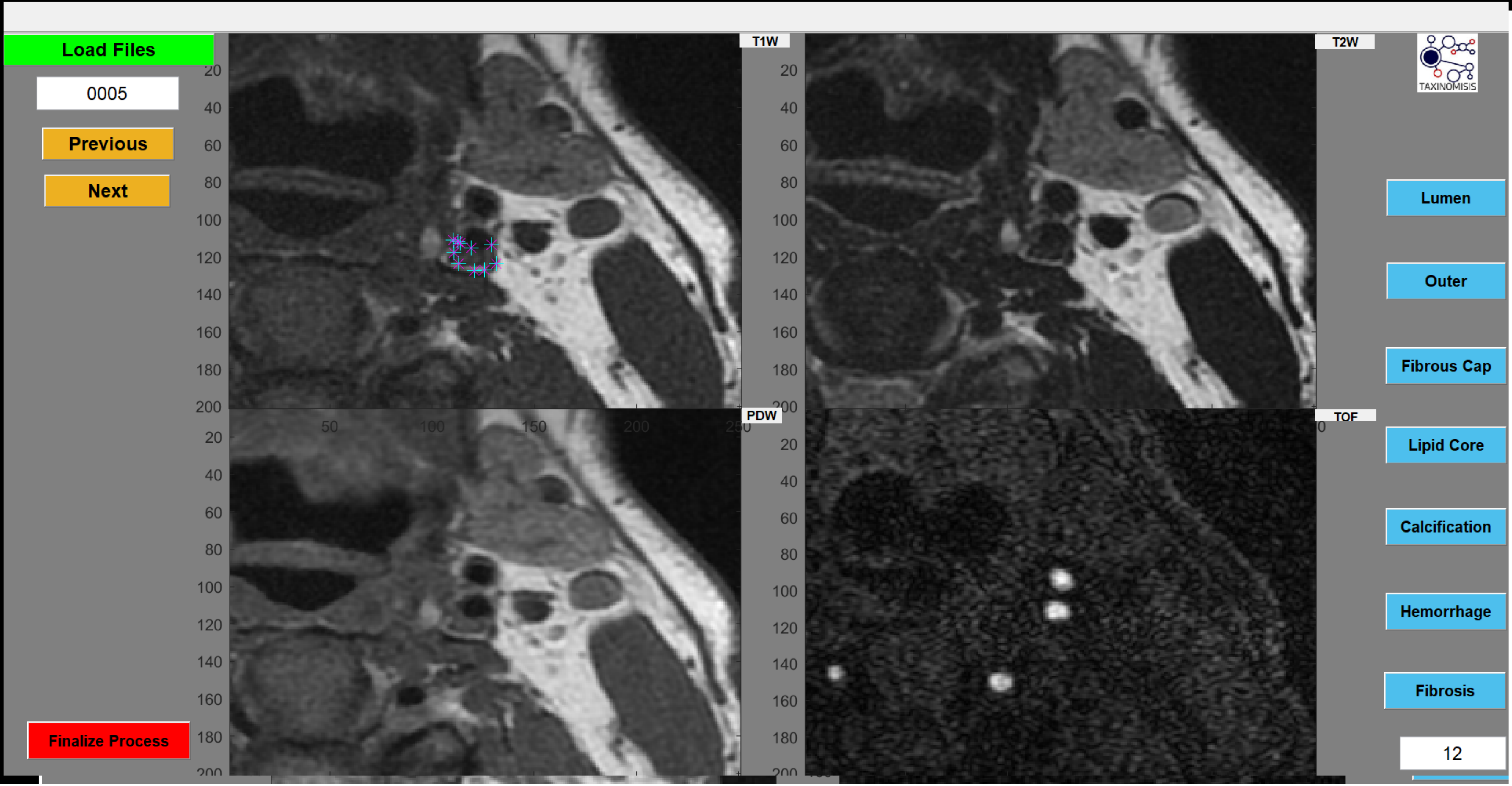
Iso T1W, high T2W - Fib





Load Files
0003
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
8


Load Files
0004
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
10


Load Files
0005
Previous
Next
Finalize Process
T1W
T2W
PDW
TOF
TAXINOMISIS
Lumen
Outer
Fibrous Cap
Lipid Core
Calcification
Hemorrhage
Fibrosis
12

Thank you